TÜRK KARDİYOLOJİ DERNEĞİ

32. ULUSLARARASI KATILIMLI TÜRK KARDİYOLOJİ KONGRESİ

OLGU SUNUMLARI *CASE PRESENTATIONS*

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20 - 23 EKİM 2016

REGNUM CARYA KONGRE MERKEZİ **ANTALYA**

Bilimsel Sekretarya

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32nd TURKISH CARDIOLOGY CONGRESS
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TURKISH SOCIETY OF CARDIOLOGY
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32nd TURKISH CARDIOLOGY CONGRESS
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Değerli Meslektaşımız,

Türk Kardiyoloji Derneği yıl boyunca çeşitli eğitim programları ve etkinlikler düzenlemenin yanı sıra her zaman olduğu gibi Ekim 2016'da da Uluslararası Katılımlı Türk Kardiyoloji Kongresi'ni gerçekleştirmeyi planlamaktadır. Hem katılımcı sayısı hem yüksek kalitedeki bilimsel içeriği ile ulusal ve uluslararası düzeyde önde gelen bilimsel toplantılardan biri olan kongremizin zengin bilimsel programı, Kardiyologlar ve Kardiyoloji Asistanlarının yanı sıra İç Hastalıkları, Kalp Damar Cerrahisi, Çocuk Kardiyolojisi ve Kalp Damar Cerrahisi, Nöroloji, Nöroşirürji, Endokrinoloji, Nefroloji, Gastroenteroloji, Aile Hekimliği gibi çok çeşitli alanlardan tüm katılımcılarımıza hitap edecek geniş bir yelpazede doyurucu bir içerikle hazırlanmaktadır.

32. Kongremiz de, uzun süredir büyük bir başarıyla kongremize ev sahipliği yapan Antalya'nın yeni ve güzel bir tesisinde, Regnum Carya Otel Kongre Merkezi'nde yapılacaktır.

Kongremizi Kardiyoloji alanında bölgenin lider toplantısı yapma hedefimiz devam etmektedir. Bu yıl gerçekleştireceğimiz Kongremize Avrupa Kardiyoloji Derneği üye ülkelerinden meslektaşlarımızın yanı sıra başka kıtalardan meslektaşlarımız da katılacaklardır. Geçen yıl 3000'e yakın kişinin katıldığı kongremize katılımın bu yıl daha da artmasını bekliyoruz.

Kongremizde sizler için en iyi programı hazırlamaya gayret ettik. "Sempozyumlar", "Karşıt Görüş" ve "Nasıl Yapalım" oturumları ile kalp ve damar hastalıklarıyla ilgili son bilgilerimizi güncelleyip tartışacağız. Her oturumumuzda Türkiye'den ve dünyadan konularında büyük ağırlığı olan değerli konuşmacılar ve tartışmacılar yer alıyor. ESC, ACC, Türk Dünyası Kardiyoloji Birliği, EHRA ve EAPCI ile ortak oturumlarımızın dikkatle izleneceğine inanıyoruz.

Uluslararası boyutu bu yıl daha da güçlenen kongremiz yine hem TTB hem EBAC tarafından kredilendirilecektir. Bu yıl da, geçen yıl olduğu gibi, hakemlerimiz tarafından değerlendirilip Sözlü ve Poster Sunuma kabul edilecek bildiriler SCI-E indeksinde yer alan dergimiz The Anatolian Journal of Cardiology 'nin özel ekinde yayınlanacak; hakemlerimizden en yüksek puanı alacak En İyi Sözlü Bildiri ve her gün asılacak posterler arasından uluslararası jürimizce belirlenecek Günün En İyi Posteri Ödüllerimiz bu Kongremizde de verilecektir.

Kongremizde sizleri de aramızda görmekten memnuniyet duyacağız. Türkiye'den ve dünyanın birçok ülkesinden gönderdikleri, özenle seçilmiş konularda, titizlikle hazırlanmış bildirileriyle ve klinik uygulamalar sırasında karşılaştıkları dikkat çekici olgularla Kongremizi güçlendiren tüm katılımcı yazar ve meslektaşlarımıza; her yıl olduğu gibi bu yıl da yüksek sayıda bildiri ve olguyu titizlikle değerlendirmeye gönüllü katkıda bulunacak uluslararası ve Türk hakemlerimize şükran borçluyuz.

20-23 Ekim 2016'da 32. Ulusal Kardiyoloji Kongremizde buluşmak, bilgilerimizi paylaşmak dileği ve saygılarımızla.

Prof. Dr. Mahmut Sahin, FESC
TKD Başkanı

Prof. Dr. Mustafa Kemal Erol
TKD Gelecek Başkanı

TURKISH SOCIETY OF CARDIOLOGY
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32nd TURKISH CARDIOLOGY CONGRESS
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Dear Colleagues,

In addition to its various training events and activities, Turkish Society of Cardiology has planned to hold this year's National Cardiology Congress in October at a high scientific level as always to meet the expectations on its 53rd anniversary. The congress, as a leading scientific event both at the national and international level with a remarkable number of participants and high quality scientific content, is being designed to appeal to all participants in a satisfactory way and broad range of its scientific program and diversified flavors of social events.

The 32nd Congress will again take place in Antalya, a venue having successfully served our congresses in the past, but this time at the Regnum Carya Hotel and Congress Center.

We sustain our ambition to make our congress as the leading congress of cardiology in our region. In addition to our colleagues from the European Society of Cardiology, other colleagues from different continents will also attend this year's congress. We expect increased attendance to our congress, in which we hosted almost 3000 people last year.

We've been working hard to prepare the best program for you. During our "Symposia", "Debates" and "How To Do" sessions, we will be updating our latest knowledge on cardiovascular diseases. Every session will take place with participation of esteemed speakers and discussants from Turkey and from around the globe. We strongly believe that our joint sessions with the ESC, ACC, Turkic World Cardiology Association, EHRA and EAPCI will be closely followed by the attendees.

The congress having strengthened more than ever with its international flavor will again be credited by the Turkish Medical Association and EBAC. As it was the case last year, the papers accepted for Oral and Poster Presentations after being evaluated by referees will be published in the Special Edition of the Anatolian Journal of Cardiology cited in the SCI-E index.

We will be more than happy to have you with us during our congress. Authors and colleagues participating with meticulously prepared papers and clinical cases on selected topics will be strengthening our congress. We owe gratitude to our referees, who have carefully reviewed high number of papers and cases with enthusiasm.

Hoping to see you on 20-23 October 2016 for the occasion of our 32nd National Cardiology Congress.

Prof. Mahmut Sahin, M.D., FESC
President of TSC

Prof. Mustafa Kemal Erol, M.D.
President Elect of TSC

32nd TURKISH CARDIOLOGY CONGRESS
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İÇİNDEKİLER
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OLGU SUNUMLARI
CASE REPORTS

OS-01

Selective atrial vagal denervation only via conventional electrophysiological recordings to treat significant sinus pauses

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Aims: Cardio neuroablation (CNA) is a lesser-known technique for management of patients with excessive vagal activation on the basis of radiofrequency catheter ablation (RFCA) of the areas related to the autonomic ganglia around the heart. To detect these ganglionic sites, various tecniques as like high frequency stimulation and spectral mapping were used in the previous literature. We presented a case with symptomatic sinus pauses undergoing succseful CNA procedure only via conventional electrophysiological recordings.

Methods and Results: We report the case of a patient with frequent symptomatic daytime sinus pauses of 3-11 s without atrioventricular (AV) block. Echocardiography, treadmill test, thyroid function test levels, and polysomnogram were normal. On Holter recordings, minimum hart rate 18/bpm and mean heart rate was 38/bpm. After atropin sulfate infusion, more than 50% increase in heart rate were observed. So, it was decided that CNA could be useful. Both atria were mapped by using Ensite NavX system and atrial electrical potentials demonstrating high amplitude fractioned electrograms (>0.7mV and ≥4 deflections) was used to localize endocardial vagal innervation in both atria, responsible for the sinus node and AV node modulation, and RF pulses were applied in those sites only (Figure 1-3). After finishing the procedure, significant changes were observed in the heart rate (34-90 b.p.m.), atrial-His interval (115-74 ms), Wenckebach cycle length (820-570 ms), and sinus node recovery time (1700-760 ms). There was no >3 s sinus pauses at the end of 1 month follow-up Holter recordings (At 1, 2 3 and 4. weeks, respectively).

Conclusion: Ablation of the endocardial vagal innervation sites seems to be safe and efficient in reducing the frequency and the length of the sinus pauses. It may be possible by identifying certain components of the atrial electrogram, resulting in a conservative approach.

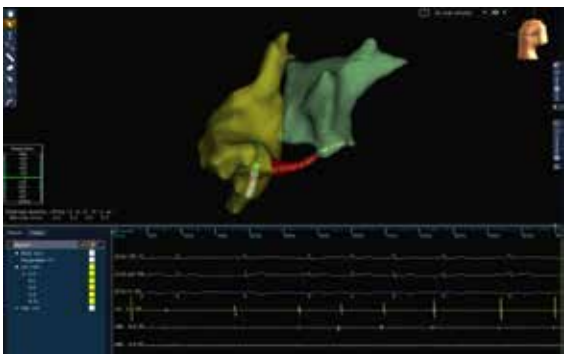

Figure 1. Electroanatomical mapping view of both atria Figure 1 legend Please see the intracardiac electrogram recordings on below. Preprocedural basal cycle lenght is nearly 1500 ms.

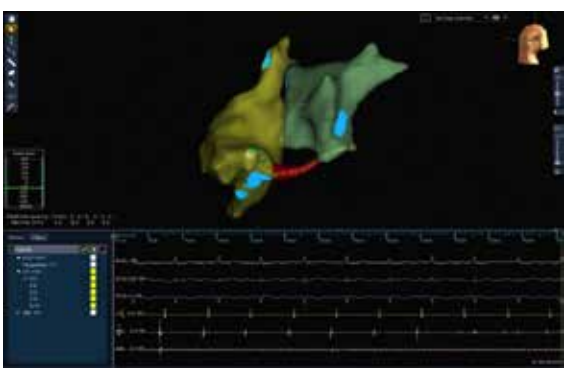

Figure 2. Electroanatomical mapping view of both atria Figure 2 legend Please see the intracardiac electrogram recordings on below. Postprocedural basal cycle lenght is nearly 650 ms.

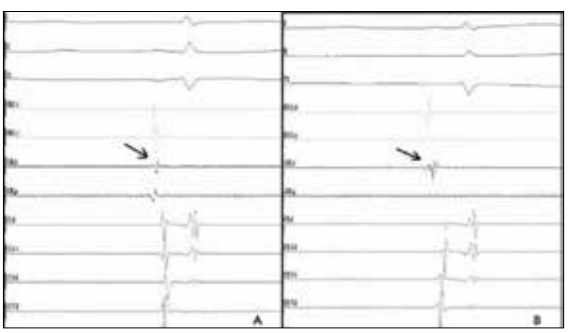

Figure 3. Intracardiac electrograms of compact **(A)** and fibrillary **(B)** atrial myocardium. Please see biphasic atrial electrogram **(A)** and high amplitude fractioned atrial electrogram **(B)** in compact and fibrillary atrial myocardium, respectively.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OS-02

Boğazda ağrı ve yutkunma sonrası gelişen çoklu senkop atakları: Nadir karşılaşılan bir durum, vagoglossofaringeal nevralji sendromu

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Bu olguda boğazda derin ve bıçak saplanır tarzda paroksismal ağrı sonrası uzun duraklamaları olan ardından senkop ve kasılmaları olan bir hasta sunuldu. Olgumuz karbamazepin tedavisi ve pacemaker implantasyonu ile sağlığına kavuştu. Olgumuzda vagoglossofaringeal nevralji sendromu tanımlanmaya ve tedavi seçenekleri tartışılmaya çalışıldı.

Olgu sunumu: Yetmiş yaşında erkek hasta son 2 aydır çiğneme ve yutkunma ile gelişen boğaz ağrısı, son 24 saattir ağrıya eşlik eden bayılma ve kasılma şikayetleri ile acil servisimize başvurdu. Takiplerde yutkunma ve sıvı gıda alımında 30 sn kadar uzayan duraklamalar izlenen hastada çoklu senkop atakları ve senkoba eşlik eden kasılmalar izlendi (Video 1) Yapılan radyolojik ve labaratuar değerlendirmelerinde (beyin, boyun, toraks bt ve mr, eeg...) patoloji saptanmadı. Hastaya ağrıları nedeni ile karbamazepin ve steroid tedavisi başlandı, geçici pacemaker implante edildi. Semptomları gerileyen hastaya pacemaker kapatıldığında bradikardi ve senkop ataklarının devam etmesi nedeni ile kalıcı pacemaker implante edildi.

Tartışma: Senkop atakları ile glossofaringeal nevraljinin başlangıcı arasında genellikle değişken bir zaman süreci yer alır. Ağrı ataklarına bradikardi, hipotansiyon ve senkop eşlik edebilir. Glossofaringeal nevralji ve senkop arasındaki ilişkiye ait en geçerli teoriye göre posterior farinks duvarından kaynaklanan duysal implulslar medulla oblangatada bulunan traktus solitorius ve onun nükleuslarına ulaşarak kollateral lifler aracılığıyla vagusun dorsal nükleusuyla ilişki kurar. Dorsal nükleus kalp, bronş ve abdomene giden parasempatik lifleri sağlayan temel otonomik nükleustur. Olguların çoğu idiyopatik olarak görülür. Sekonder nedenler içinde serebellopontin köşe tümörleri, inrakranial vasküler kompresyonlar, larigeal ve nazofarigeal karsinomlar sayılabilir. Tedavide amaç ağrıyı kontrol altına almak ve bardikardi, senkobu önlemektir. Bu amaçla medikal, anestetik ve cerrahi prosedürler kullanılmaktadır. farmakolojik olarak karbamazebin, gabapentin kullanılmaktadır. medikal tedaviye dirençli durumlarda cerrahi prodesürler devreye girmektedir. Senkop ve bradikardi ataklarını önlemede pacemaker kullanımının sonuçları güz güldürücüdür. Olgumuz ve literatürde yer bulan diğer olgular incelendiğinde vagoglossofaringeal nevralji sendromu tanımlaması yapmak yerinde olacaktır.

OS-03

Sol ventrikül summit bölgesinden kaynaklanan ventriküler erken vurulara (VEV) ikincil takikardiyomiyopati gelişen hastaya radyofrekans kateter ablasyon (RF ablasyon) uygulanması

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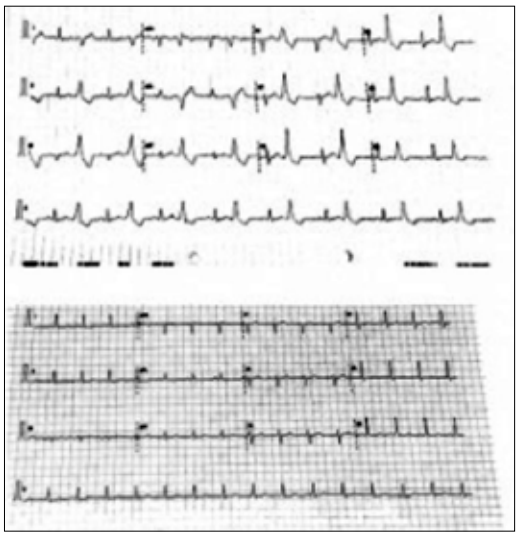
Giriş: VEV ler masum gözükmekle birlikte takikardiyomiyopatiye yol açabilirler. VEV ler medikal ve RF ablasyon yöntemi ile tedavi edilirler. RF ablasyon yöntemi medikal tedavi ile kıyaslandığında daha başarılı bir tedavi olarak karşımıza çıkmaktadır. VEV lar en sık RVOT kaynaklı olmasına rağmen, %15 oranında epikardiyal kaynaklıdır. LV epikardiyal kaynaklı VEV lerin en sık köken aldığı bölge LV summit bölgesidir. LV summit bölgesi şekilde RF ablasyonda kullanılan anatomik giriş yerleri GCV ve AIV dir (Şekil 1).

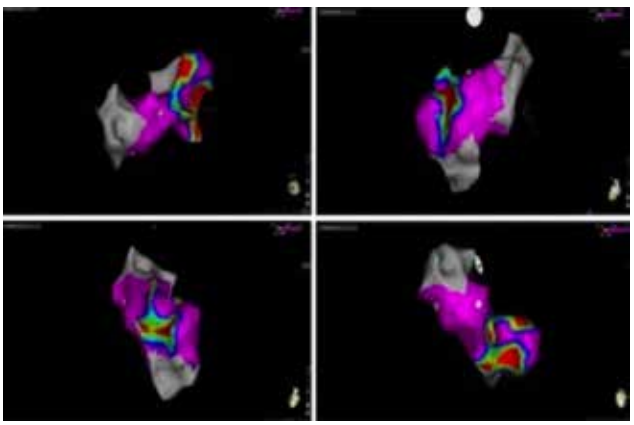
Olgu Sunumu: 46 yaşında kadın hastada HT, Tip 2 DM mevcuttu. Son 6 aydır çarpıntı ve nefes darlığı şikayeti bulunmaktaydı. EKG de bigemine VEV ((RBBB), inferior aks) izlendi. (Şekil 2a). Yirmi dört saat Holter EKG monitörizasyonunda 61987 adet VEV (günlük sıklık %42, 240 adet couplet, 26 adet nsvt). EKO da 1. derece mitral yetmezlik (MY) izlendi.EF: %40 olarak değerlendirildi. Hastanın sık VEV lerinin olması, EF: %40 saptanması üzerine VEV e bağlı gelişen TKMP düşünüldü, hastaya RF ablasyon yapılması planlandı. Hasta kateter laboratuvarına alındı. 2 adet sağ femoral venden 8F sheat, 1 adet sağ juguler venden 8F sheat yerleştirildi. CARTO ile LVOT ve koroner küspisler aktivasyon yöntemleri ile haritalandı. Endokardiyal haritalamada LV summit bölgesinde en erken sinyaller alındı (Şekil 3), endokardiyal olarak alınan kayıtlarda en erken aktivasyon -8-10 msn kadar izlendi ve VEV kaynağının CS olabileceği düşünüldü. Sağ juguler venden 8F Marıner RF ablasyon kateteri ile CS distaline ulaşılmaya çalışıldı fakat ulaşılamadı, teleskopik CS yerleşim kateteri ile CS kanüle edildi ve bu yolla CS venografisi yapıldı (Şekil 5d, e). Bu kateter içinden RF ablasyon kateteri ile AIV proksimaline ulaşıldı. -22 msn lik erkenlik saptandı.(Şekil 4). Pace maping te 12/12 konfirmasyon sağlandı. KAG ile LAD ve CX komşuluğu değerlendirildi. CX'e 5 mm uzaklıkta uygun ablasyon alanı tespit edildi (Şekil 5b, c, f, g). 20W, 50 derece güç ile 60 sn RF ablasyon uygulandı. Ablasyon sonrası VEV ler kayboldu. İşlem sonrası EKG sinde VEV izlenmedi (Şekil 2b). Hasta kalp yetmezliği tedavisi ile taburcu edildi. 2. ay kontrolde hasta asemptomatikti ve yapılan EKO da EF:%65, 24 saatlik holterde toplam 61 adet VEV izlendi.

Tartışma: RF ablasyon TKMP gelişmiş hastalarda LV fonksiyonlarının geri döndürülmesi bakımından etkin bir tedavi yöntemidir. LV summit bölgesinden kaynaklanan VEV ler AIV ve GCV kullanılarak RF ablasyon yöntemi ile tedavi edilebilir. LV summit bölgesinden kaynaklanan VEV lere RF ablasyon uygulanırken sol koroner sistemin zarar görmesi, yavaş kan akımı nedeni ile impedansın hızla yükselerek RF ablasyonun yeterli süre yapılamaması gibi problemler karşımıza çıkabilir. Bu zorlukları ortadan kaldırabilmek için öncelikle CS nin iyi görüntülenip AIV ve GCV nin anatomik lokalizasyonları tam net olarak belirlenmeli ve RF ablasyon sırasında eş zamanlı olarak KAG yapılarak ablasyon kateterinin koroner sistem ile yakınlığı göz önünde bulundurulmalıdır.

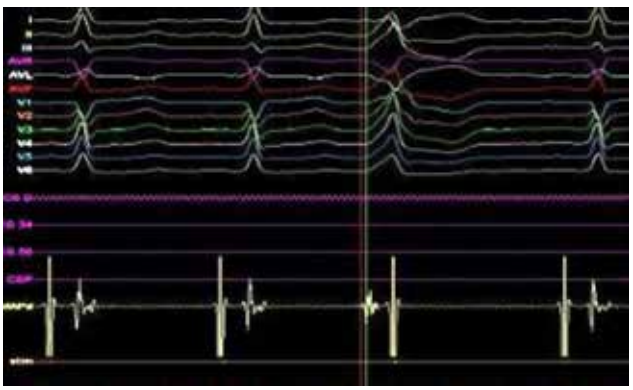
Şekil 1. GCV ve AIV anatomik lokalizasyon.



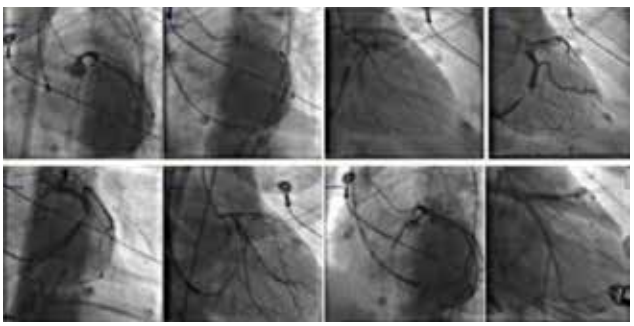
Şekil 2. EKG görüntüleri.



Şekil 3. CARTO görüntüleri.



Şekil 4. EGM görüntüleri.



Şekil 5. KAG görüntüleri.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OS-04

Nadir bir elektriksel fırtına nedeni; Conn sendromu

Mustafa Yenerçağ[1], Serkan Yüksel[2], Ömer Kertmen[2], Metin Çoksevim[2], Mahmut Şahin[2], Ömer Gedikli[2]

[1]*Samsun Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, Samsun*
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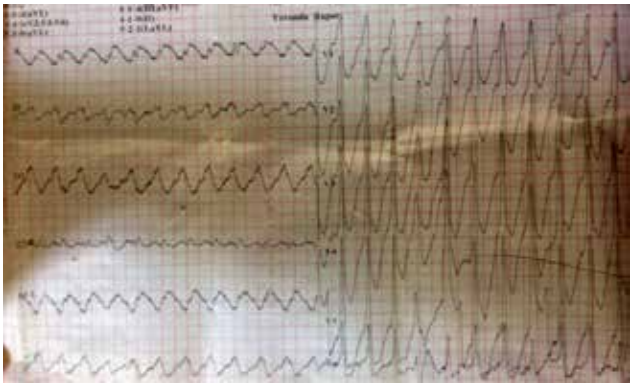
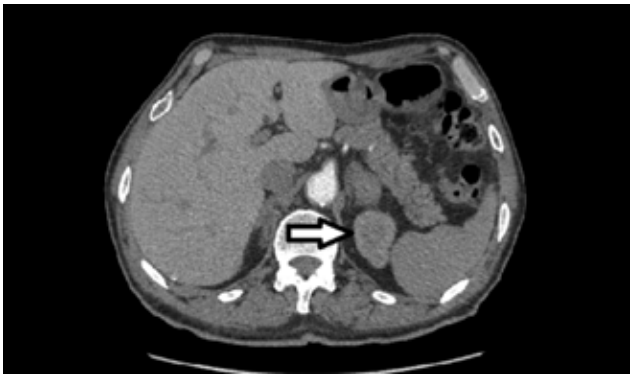
Giriş: Kalp yetersizliği hastalarında mortalitenin başlıca nedeni ventriküler aritmilerdir. Kalp yetersizliği hastalarında kullanılan diüretiklerinde etkisiyle elektrolit imbalansları sık görülmekte ve bu durum malign artimileri tetiklemektedir. Conn sendromu nadir görülen primer hiperaldosteronizmdir. Ciddi hipertansiyon, hipernatremi, derin hipokalemi ve metabolik alkalozla ilişkilidir. Bu olguda Conn sendromuna bağlı gelişen elektrolit imbalansının neden olduğu elektriksel fırtınanın, sürrenal adrenalektomi sonrası sonlanması anlatılacaktır.

Olgu Sunumu: İskemik olmayan dilate kardiyomiyopati nedeniyle CRT-ICD (Cardiac Resynchronization Therapy - İmplantable Cardioverter Defibrilator) pacemaker bulunan 64 yaşında erkek hasta acil servise nefes darlığı, çarpıntı ve ICD şoklaması şikayeti ile başvurdu. Tansiyonu 180/110 mmHg ölçülen, ortopneik ve takipneik olan hastanın bilateral akciğer orta zonlara kadar ralleri mevcuttu. Acil servisteki elektrokardiyografisinde geniş QRS' li taşikardi izlendi (Şekil 1). Pacemaker ayarlarında problem olmayan hastanın EGM (Elektrogram) kayıtlarında ventriküler taşikardiyi ve ventriküler fibrilasyonu sonladırmak için çok kez uygun ICD şoklamaları izlendi. Hastanın acil başvurusunda elektrolit değerleri Na: 138 mEq/L, K:2.7 mEq/L, Cl: 94 mEq/L, Ca:9.5 mg/dl, Mg: 2.1 mg/dl ölçüldü. Koroner yoğun bakımda potasyum replamanı yapılan ve antiaritmik tedavi verilen hastanın tekrarlayan ventriküler taşikardisi ve ICD şoklamaları oldu. Diüretik tedavisi kesilen, 100 mg spirinolaktone ve 10 mg ramipril oral tedavisi alan hastaya günlük 80 mEq potasyum replasmanına rağmen potasyum değerleri düşük (K <3.5 mEq/L) değerlerde ölçüldü. Ventriküler aritmilere hipopotaseminin neden olduğu düşünüldü. Endokrinolojik açıdan değerlendirilen hastaya primer hiperaldosteronizm açısından yapılan bilgisayarlı tomografide sol sürrenal 35x30 mm kitle izlendi (Şekil 2). Aldosteron üreten adenom düşünülen hastanın serum renin ve aldosteron değerleri aldığı tedavi nedeniyle tanısal olmadığından hastaya cerrahi adrenalektomi planlandı. Endokrinolojik cerrahlar tarafından 35X30 mm' lik sol sürrenal kitle başarılı şekilde eksize edildi. Patoloji sonucu adrenokortikal adenom ile uyumlu geldi. Hastanın kontrol potasyum değeri 4.1 mEq/L ölçüldü. Potasyum replasmanı ihtiyacı olmayan ve ventriküler malign aritmiler izlenmeyen hasta taburcu edildi. Son üç ay içindeki kontrollerinde ICD şoklaması ve hipopotasemi izlenmedi.

Tartışma: Elektrolit imbalansı (öncelikle de hipokalemi ve hipomagnezemi) malign aritmileri tetiklemektedir. Tedaviye dirençli elektriksel fırtınalara elektrolit imbalansının da neden olabileceği düşünülmelidir. Bu nedenle kalp yetersizliği hastalarında elektrolit imbalansı düzeltilmelidir. Dirençli hipokalemiye bağlı elektiriksel fırtınalarda hiperaldosteronizm akılda tutulmalı Conn sendromu açısından endokrinolojiye danışılmalıdır. Çünkü Conn sendromu nadirde olsa geri döndürülebilir bir ventriküler aritmi nedenidir.



Şekil 1. Geniş QRS'li taşikardi.



Şekil 2. Bilgisayarlı tomografide sol sürrenal kitle.

OS-05

Ablation of ventriculer premature contraction originating from left ventriculer summit

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36-year-old woman without coronary artery disease was admitted shortness of breath and palpitation. Medical theraphy instituted (karvedilol, 2x12.5 mg) since last 1 year but patient still symptomatic. A 12-lead electrocardiogram revealed bigeminy premature ventricular contractions (VPCs) with a left bundle branch block morphology right inferior axis and precordial R/S transition zone V1 (Figure 1). A transthoracic echocardiogram showed dilated LV dilatation (left ventricular internal dimension, diastole (LVIDd (2D) 6.6 cm) and severe LV systolic dysfunction (LVEF 34%). Doppler study revealed grade 1 LV diastolic dysfunction. LV posterior (PWd (2D) 0.74 cm) and interventricular wall thickness (IVSd (2D) 0.89 cm) were normal. Electrophysiologic (EP) study and catheter ablation was scheduled for eventual frequent VPCs induced tachycardiomyopathy and considering drug refractory VPCs. After providing the patient with detailed description of the EP study and the catheter ablation, written informed consent was obtained. We decided to start mapping from LV due to early transition. Activation and pace mapping in the LVOT was performed using ablation catheter introduced retrogradely via the right femoral artery. A three-dimensional shell of the aortic root was created using CARTO system (CARTO XP™ , Biosense Webster, Diamond Bar, CA, USA). The early ventricular electrograms (-32 ms) were obtained in the left coronary cusp (LCC). A 2-mm spacing decapolar catheter (CARTO 3, Biosense Webster Inc, Diamond Bar, CA) for radiofrequency ablation (power-control mode at 30 W with an irrigation flow rate of 20 mL/min) was performed at this site but VPCs still was observed. Second mapping was made from the left ventricular endocardium, just below the LCC. Activation mapping of PVCs in the left ventricle revealed that the site of earliest activation (-27 ms) and radiofrequency ablation was performed at this site suppressed the PVCs for a while. Then the PVC came out again. We thougth that arryhytmia focus would be epicardial because of failure of endocardial ablation approach and increased intrinsicoid deflection time. We planned mapping firstly from coronary sinus. Early ventricular activation recorded in ablation catheter at the junction of great cardiac vein (GCV) and anterior interventricular vein (AIV) preceded the QRS onset by 18 ms (Figure 2). Selective coronary angiography was performed prior to ablation due to its close proximity with the great cardiac vein of the left anterior descending (LAD) and circumflex (CX) coronary arteries in LV summit area (Figure 3). Ablation at this site achieved total suppresion of PVCs (Figure 4).

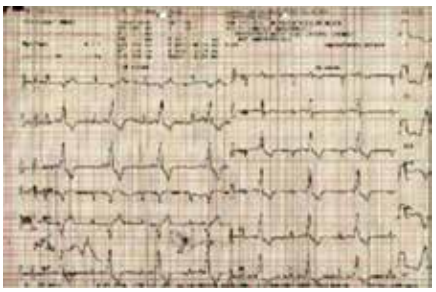

Figure 1. A 12-lead electrocardiogram revealed bigeminy premature ventricular contractions (VPCs) with left bundle branch block morphology right inferior axis and precordial R/S transition zone V1.

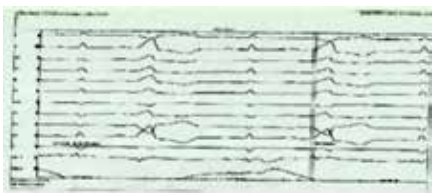

Figure 2. The ventricular electrocardiogram showing the earliest local activation at the coronary sinus by 18 ms.

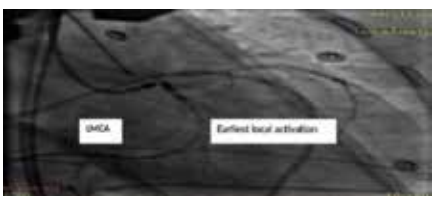

Figure 3. The successful ablation site and the left main coronary artery, respectively.

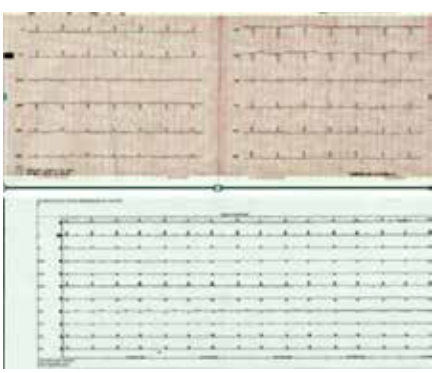

Figure 4. ECG findings and mapping catheter recordings at successful ablation site at the LV-summit.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OS-06

Report of a family with Wolff-Parkinson-White syndrome and craniofrontonasal syndrome

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Introduction: Wolff-Parkinson-White (WPW) syndrome is a common cause of supraventricular tachycardia with prevalence in Western countries of 1.5 to 3.1 per 1000 persons, may cause sudden death. To date, mutations in only one gene, PRKAG2, which encodes the 5'-AMP-activated protein kinase subunit γ-2, have been identified as causative for WPW. We identified a family with craniofrontonasal syndrome in which the WPW syndrome was shown in members of the family. Craniofrontonasal syndrome (CFNS) is a rare genetic entity with X-linked dominant inheritance, and is due to mutations in the Ephrin-B1 gene.

Case Report: A 14 years old girl was referred to the cardiology clinic because of paroxysmal palpitation. Her mother and father are cousins and she is inbred. Craniofrontonasal syndrome is present in our case, her father and her sister. She had a history of surgery due to frontonasal dysplasia. She has heterozygous missense mutation (c.451G>A; Gly151Ser) in exon 3 of Ephrin-B1 gene. Her 12-lead ECG showed short PR interval and Delta waves, and was considered as type-A WPW syndrome (Figure 1). Her father was also had successful catheter ablation for left lateral accessory pathway four years ago and has hemizygous missense mutation (c.451G>A; Gly151Ser) in exon 3 of Ephrin-B1 gene.

Discussion: CFNS is a rare genetic entity, characterized by coronal craniosynostosis, frontal bossing, severe hypertelorism, craniofacial asymmetry, downslant palpebral fissure, broad nasal root, bifid nasal tip, grooved fingernails, curly wiry hair, and abnormalities of the thoracic skeleton. Intelligence is usually unaffected. CFNS exhibits unexpected manifestations between males and females as the latter are more affected. Cellular or metabolic interference due to X inactivation explains greater severity in heterozygous females than in hemizygous males. There are very few cases such as describing association of CFNS with heart defects such as atrial septal defect. To the best of our knowledge, this family is the first CFNS family presenting with WPW syndrome. Genetic analyses are needed in order to explain this association between CFNS and WPW syndromes. Clinicians must be aware in patients with CFNS syndrome in terms of the presence of pre-excitation.

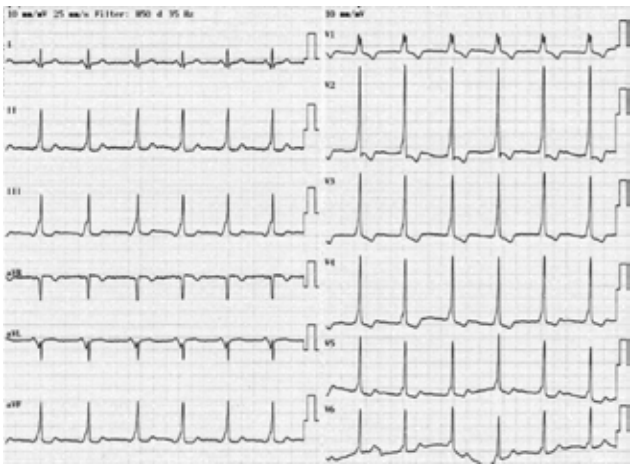

Figure 1. The 12-lead ECG of the patient showing Wolff-Parkinson-White pattern.

OS-07

Polymorphic ventricular tachycardia due to variant angina diagnosed on holter monitoring and confirmed with cold pressor test

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A 52 year old man presented at out patient clinic complaining of chest pain for three months. He emphasized recurrent pain occuring every night and increased intensity in early morning. Burning type of pain lasted 5 minutes and releaved by itself without any medication. He underwent coronary angiography which revealed normal coronaries three weeks ago in another hospital. The night pain continued despite spasmolytics prescribed at discharge. ECG was normal. Physical examination was unremarkable. Since it was a night pain, 24 hour Holter minotoring was ordered.It displayed gradual ST segment elevation resulting in huge ST segment elevation (Figre 1a). Recurrent episodes of non sustained polymorphic ventricular tachycardia which spontaneously terminated were observed (Figure 1b). The patient was taken into angiography laboratory. Coronary angiogram was normal. Since ergonovine was unavailable, we decided to perform cold pressor test. Right Judkins catheter left in coronary in order to visualize possible vasosapsm. Patient's left hand immersed in a ice water conatiner, in a half minute time huge ST segment elevation occured and rythm turned into polymorphic ventricular tachycardia (Video 1). Since it would be fatal, 100 mcg nitroglycerin was injected into catheter and the hand was taken out of container. ST segment elevation resolved in seconds.Extended release nifedipin 30 mg twice daily was prescribed. Control holter on 3rd day ruled out any vasospastic episode. Patient was followed up with regular Holter monitoring uneventfully for 1 year. Variant angina diagnosis could be challenging due to

requirement of additional drugs like ergonovine at time of diagnostic angiography. Due to unavalibility of drugs, operators sometimes skip the procedure which may result misdiagnosis. However 24 hours holter monitoring and cold pressor tests which are easily applicable still help clinician to diagnose variant angina.

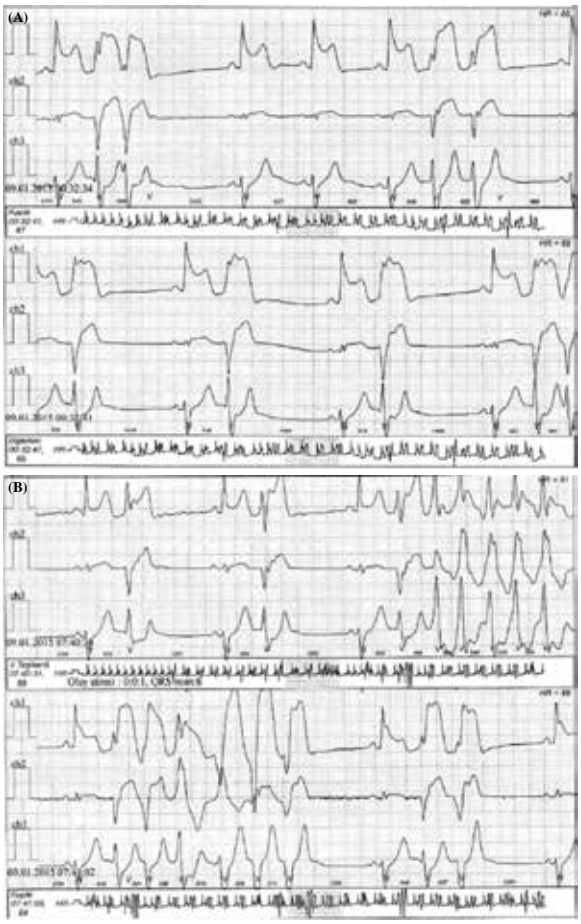

Figure 1. (A) Holter monitoring displayed huge ST elevation at midnight. **(B)** Polymorphic ventricular tachycardia occured in the morning and spontaneously terminated.

OS-08

First valve-in-valve bailout implantation of second generation Symetis ACURATE TF in a patient with mitral valve prosthesis

Mehmet Bilge, Sina Ali, Yakup Alsancak, Mustafa Duran, Serkan Sivri

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Introduction: Since the first case of TAVI with a balloon-expandable Edwards SAPIEN valve (Edwards Lifesciences, Irvina, CA) in 2002 (1), several other transcatheter valves have been introduced into clinical practice. One of second generation TAVI devices is the ACURATE neo™ Aortic Bioprosthesis. Herein, we present a patient with a history of mechanical mitral valve replacement who suffered embolization of ACURATE neo™ to the Sinus of valsalva which was successfully managed with a second ACURATE neo™ Bioprosthesis.

Case Report: A 66-year old man who previously underwent a coronary artery bypass graft surgery and mechanical mitral valve replacement presented with worsening dyspnea due to severe aortic stenosis. Transthoracic echocardiography revealed mildly depressed left ventricular systolic function and a calcified and degenerated tricuspid aortic valve with a mean gradient of 64 mmHg and a calculated aortic valve area of 0.7 cm^2 Mild aortic regurgitation was present. The distance between the mitral prosthesis and aortic annulus was 4.8 mm with computed tomography. Then balloon valvuloplasty was performed with a 24 mm aortic balloon cathater. Later a medium sized ACURATE neo™ valve was positioned within the aortic annulus with great care using fluoroscopic guidance with the aim not to interfere with the mitral prosthesis (Figure 1). Soon after deployment, fluoroscopy showed the bioprosthesis had embolized into the sinus of valsalva (Figure 2). A decision was made to implant a second medium sized ACURATE neo™ within the first valve. The second bioprosthesis was positioned within the annulus slightly lower than the first implant and sucessfully placed within the first ACURATE neo™ using the same stepped technique. The final aortography showed a mild paravalvular leak and no coronary obstruction (Figure 3).

Discussion: Finally, when using the ACURATE neo™ TAVI system for a valve-in-valve procedure, operators should pay attention that the upper crown of the embolized valve is positioned below the left main coronary artery ostia or the lower crown is positioned above the left coronary ostia. To the best of our knowledge, this is the first reported case of bail-out implantation of second generation ACURATE neo™ in a patient with mitral valve prosthesis.

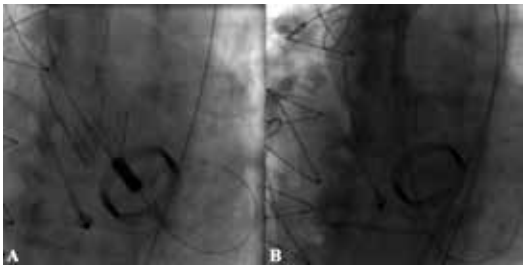

Figure 1. (A) The early position of ACURATE bioprosthesis after implantation. **(B)** Embolization of the ACURATE bioprosthesis into the sinus of valsalva.

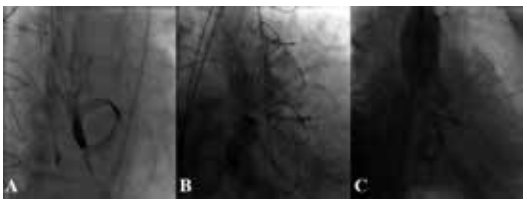

Figure 2. (A) The second ACURATE bioprosthesis position and association with the first valve and mitral prosthesis **(B)** Two Symetis ACURATE valve and their positions **(C)**.

OS-09

Successful percutaneous implantation of Symetis ACURATE neo transcatheter aortic bioprosthesis in a patient with a previous mechanical Mitral valve

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Atatürk Training and Research Hospital, Ankara

A 73-year old woman who had previous mechanical mitral valve replacement (MVR) presented with worsening symptoms of dyspnea. Echocardiographic evaluation demonstrated a severe aortic stenosis with calcific trileaflet aortic valve (aortic valve area was 0.9 cm2, mean gradient was 42 mmHg with 45% ejection fraction). The STS score of the patient was 8.9%, mean logistic EuroSCORE was 23.8% and she was found to be in high risk group according to SURTAVI risk model. After a multi-disciplinary discussion by the Heart Team, the patient was taken for transcatheter aortic valve replacement (TAVI) using the Symetis ACURATE TF™ (SATF) via transfemoral approach. During procedure a medium size valve was positioned for deployment following balloon valvuloplasty. Afterwards unsheathing of upper crown and opening of stabilization arches, full deployment of valve was performed through unsheating of lower crown (Figure 1, Video 1-3). After implantation, the final aortography showed no paravalvular leak (Figure 2). The number of high-risk patients with pre-existing MVR undergoing TAVI is still rising and most of the procedures are still performed with the first generation devices. It is well known that the presence of a mechanical mitral valve within the aorto-mitral curtain not only makes proper positioning challenging but complicate the procedure. Although the SATF has been designed as a novel prosthesis for TAVI, there is stil paucity of data in literature for patients with history of MVR who underwent TAVI. In our patient the SATF valve showed promising results in terms of safety and feasibility.

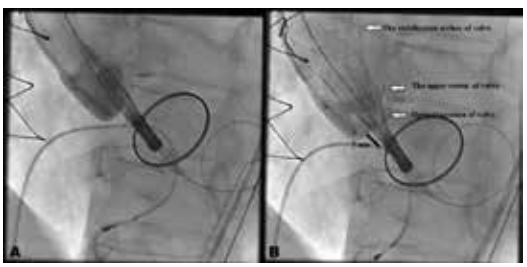

Figure 1. (A, B) Fluoroscopy is showing Symetis ACURATE neo bioprosthesis valve after unsheathing of upper crown and opening of stabilization arches and relationship between mitral prosthesis and aortic annulus

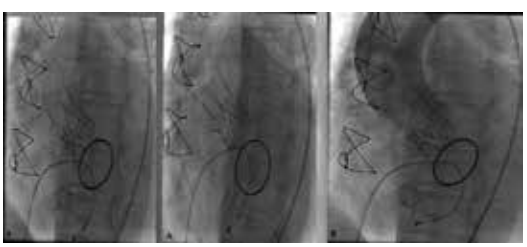

Figure 1. (A) The Symetis ACURATE neo after full deployment and association with mitral prosthesis **(B)** Arcus aortography is demonstrated no aortic regurgitation after the implantation of valve.

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalığı

OS-10

Transcatheter valve-in-valve implantation into a degenerated mitral bioprosthesis using a lotus aortic valve prosthesis

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A 67 year old man who had undergone a surgical bioprosthesis implantation in the mitral position (29-mm Hancock II, Medtronic Inc), and also in aortic position (25-mm Hancock II, Medtronic Inc) simultaneously in 2007, was admitted with progressive dyspnea. Transesophageal echocardiography revealed a degenerated mitral bioprosthesis with significant stenosis,and a normally functioning aortic bioprosthesis. Diagnostic coronary angiography revealed a chronic total occlusion in RCA. Calculated EuroScore, and STS score of mortality were 24% and 9% respectively. He was decided to undergo a transcatheter mitral valve-in-valve procedure using a transcatheter Lotus Aortic valve (Boston Scientific Inc). The procedure was performed in a hybrid operating room under general anesthesia. A left mini-thoracotomy was performed in the 5th intercostal space to gain access to the left ventricular apex. A 22 F sheath was placed and a guidewire was intended to be advanced through the degenerated mitral bioprosthesis under fluoroscopic guidance in the retrograde direction; however, it was not possible because of severe degeneraton in the prosthesis. Then a septostomy was performed using a Transseptal Guiding Lamp 90° Swartz, and a Terumo wire was introduced antegradely through the mitral bioprosthesis with transesophageal guidance, which was catched by a Snare Cath 7F catheter introduced transapically. After snaring, the Terumo wire was replaced by the preshaped Safari guidewire with a small curve through a Multipurpose catheter. After securing the correct position with a good gradient, confirmation of the absence of paravalvular leakage and the absence of LVOT obstruction, the transcatheter 27-mm Lotus valve was implanted successfully without rapid pacing. We targeted to align the transcatheter valve, such that the adaptive seal of the Lotus valve which is one of the main advantages of this valve, faces the ring of the previous bioprosthesis. Another important point was to avoid excessive protrusion of the implanted valve into the LVOT and cause subaortic obstruction. The procedure was successfuly ended after TEE confirmation of the absence of paravalvular leakage, mitral stenosis and LVOT obstruction. He was extubated in the operating room and was discharged on the 7th day. One of the most striking aspects of this case was the presence of another bioprosthesis in the aortic position, in addition to the mitral position, which complicates the procedure technically. Besides, the angulation between the LVOT and the mitral ring was unfavourable. In this manner, the Lotus valve is advantageous in terms of letting complete repositioning before the final release, if the initial implantation site is not considered to be successful during the operation. In addition, by selecting Lotus valve, we were able to avoid rapid pacing which could be poorly tolerated by a patient with limited hemodynamic stability.

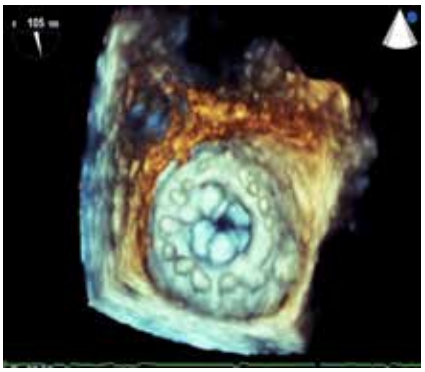

Figure 1. Transesophageal echocardiography demonstrating the significantly degenerated 29-mm Hancock II biological mitral valve. The ring is well identified.

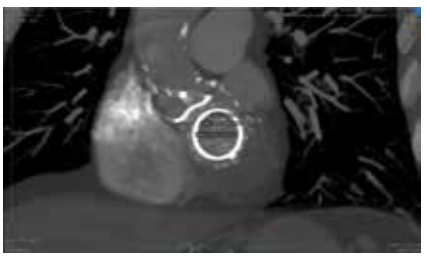

Figure 2. CT image demonstrating the distance between the aortic and mitral rings. Minimum distance was 0.4 cm and the maximum distance is 0.7 cm.

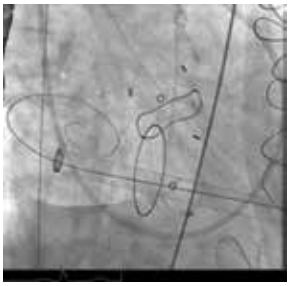

Figure 3. Transapical sheath and the guidewire crossing through the degenerated mitral prosthesis.

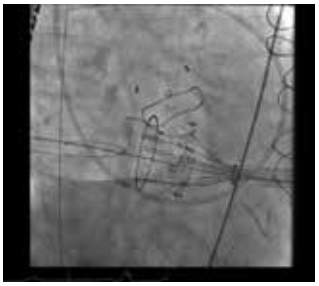

Figure 4. Opening of the Lotus valve in the degenerated mitral bioprosthesis. The waist (arrow) shows a correct size and anchoring.

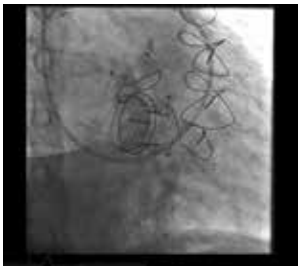

Figure 5. Lotus valve after final deployment in the degenerated mitral bioprosthesis.

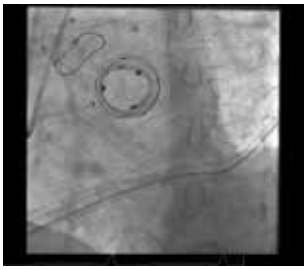

Figure 6. Lotus valve after final deployment in the degenerated mitral bioprosthesis with proper expansion.

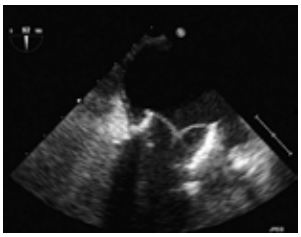

Figure 7. Transesophageal echocardiography after the implantation of the 27-mm Lotus Valve demonstrating that the prosthesis seals the ring completely.

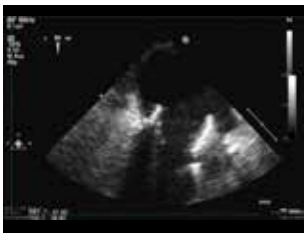

Figure 8. Transesophageal echocardiography after the implantation of the 27-mm Lotus Valve demonstrating the full and unrestricted opening of the prosthesis without any LVOT restriction.

OS-11

Valve-in-valve implantation due to malposition of transcatheter aortic valve applied by coronary guide wire protection in presence of lower-lying coronary ostium

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Introduction: Life-threatening complications such as coronary artery occlusion is still considered a potential risk for transcatheter aortic valve implantation (TAVI). Many studies reported that the impairment of the coronary blood flow was mainly due to presence of a low coronary ostium height.

Case Report: A 82-year-old female patient with history of cerebrovascular event and renal impairment with a creatinine level of 2.1 mg/dl was admitted to our hospital for worsening dyspnea caused by severe calcific aortic stenosis. Based on her age and high risk (logistic EuroSCORE: 23.2%; STS-PROM: 5.4%), the patient was referred to TAVI by heart team. The distances from left main coronary artery (LMCA) ostium to the aortic root and sinus of valsalva (SOV) diameter were measured as 6.1 mm, 18.6 mm respectively by multidetector computed tomography (Fig 1.1). The procedure was performed with implantation of a 23 mm Edwards Sapien XT valve. A bare-metal stent was advanced via guidewire into the left ascendan artery before deployment to protection coronary arteries from coronary obstruction. Prosthesis was implanted during rapid ventricular pacing on the appropriate position (Fig 1.2). However, malposition of the prosthesis into sinus valsalva was seen aortography after first deployment. LMCA ostium was assessed many different angles on aortography, no occlusion of LMCA was observed (Fig 1.3). Severe paravalvular leakage occurred. A bare-metal stent was withdrawn into the catheter successfully. The second THV, which is the same size with the first one, was implanted successfully to a deeper into the left ventricular outflow tract passing through first prosthesis. Normal coronary flow was seen by aortography (Fig 1.4-5). The early postoperative course was uneventful.

Discussion: Presence of a low coronary orifice is an important limitation of TAVI. In our case, malposition of the prosthesis was seen aortography after first deployment. LMCA was assessed on aortography, no occlusion of LMCA was observed. The possible reason of continuing coronary flow after the first prosthesis might be coronary ostium lateral to the stent frame or there might be flow laterally from the open sinuses (Fig 1.6). In patients with at high risk for coronary obstruction, we would suggest to implement an additional security measures during the TAVI procedure such as coronary protection with placing a guidewire to coronary artery. Sapien 3 and CoreValve could better prevent the occurrence of this severe complication. The use of a prosthesis which can be repositioned or retrieved in case of coronary obstruction following valve implantation should probably be preferred in high risk cases.

Conclusion: Coronary obstruction following TAVI is a rare but potentially fatal complication. Low lying coronary ostium and small SOV may be anatomical risk factors, emphasizing the importance of a proper pre-procedural evaluation in order to avoid this complication.

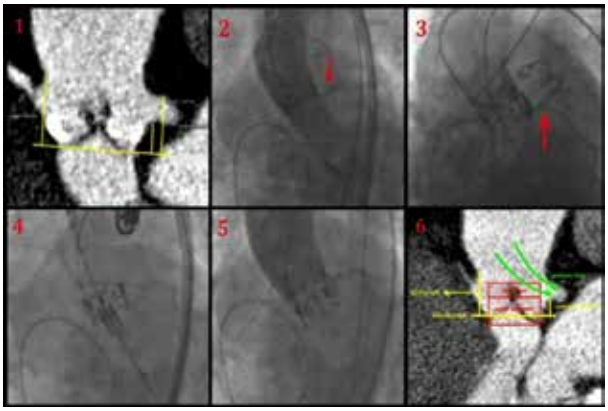

Figure 1. Pre-procedural evaluation by multidetector computed tomography (1); Stepwise implantation of the valve-in-valve procedure (2-6).

OS-12

A rare complication of transfemoral TAVI: Ventricular septal defect and successful treatment by closure device

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Introduction: Ventricular septal defect (VSD) is a rare complication of TAVI via the transfemoral approach. Herein we present a patient who had a VSD after transfemoral TAVI without known etiological factors and was also treated by closure device opened at ascending aorta.

Case Report: The patient was 85-year-old man presented with dyspnea in NYHA functional class III.Transthoracic echocardiography (TTE) showed an intensive calcified aortic valve with a mean gradient of 75 mmHg. Lojistic EuroSCORE was 26.13%. During TAVI procedure a 29-mm Edwards Sapien XT prosthesis was successfully implanted after aortic predilatation with 25 mm balloon. At first month, control TTE showed prominent left-to-right systolic shunt at the membranous interventricular septum under the prosthetic valve (Figure 1a). The defect was measured 7 mm at transesophageal echocardiography. Qp/Qs ratio was 2.1 at catheterization and left-to-right shunting was observed at ventriculography (Figure 1b). We decided to occlude the defect percutenously using an 8 mm Amplatzer muscular VSD occluder. Under local anaesthesia a 7-Fr TorqVue delivery sheath and dilatator were advanced over the guide wire. With respect to our experience we decided to open directly the left ventricular disc of the occluder in the ascending aorta and pull the disc through the prosthetic valve. Left ventricle angiography showed the size of the occluder was appropriate for the defect (Figure 1c). After procedure severe tricuspid regurgitation due to flail of lateral tricuspid leaflet was noticed which was a new finding. Probably, the delivery system passed through between tricuspid lateral leaflet chordae. So, catheter ruptured the lateral leaflet chordae.In spite of severe tricuspid regurgitation, he did not have any symptoms. Thus we decided to follow-up the patient with medical therapy.

Discussion: VSD is an unusual complication after transfemoral TAVI.In the literature there are about 8-9 percutaneous closure of the VSD after TAVI was reported for only one patient treated with the CoreValve ReValving system. In most of these cases, post-balloon dilatation or oversized prosthesis have been held responsible. The other possible causes are using oversize balloon for predilatation and/or overexpanded valve and high calcium score on the valve. Probable cause of our complication was dense calcification of aortic valve(Figure 1d).

Conclusion: Post-TAVI VSD is not only related to post-balloon dilatation and/or oversized valve implantation for Edwars Sapien XT.Intensive calcified valve can be enough to rupture interventricular septum as in our case. VSD can aggravate hemodynamic instability dramatically. Therefore, routine and careful TTE examination after TAVI is very important. Percutaneous treatment of the VSD after TAVI with Edwards Sapien XT prosthesis can be successfully treated percutaneously. Opening of the occluder device in the ascending aorta can be performed without any damage to the bioprosthetic aortic valve.

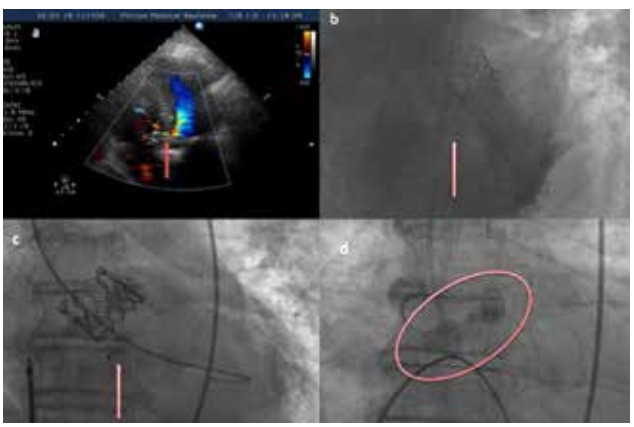

Figure 1. (A) Color doppler depict VSD shunt. **(B)** Intense contrast flow from left-to-right ventricle. **(C)** VSD closure device. **(D)** Dens calsific aortic valve. Left-to-right shunt,muscular VSD occluder and dens calsific aortic valve

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalığı

OS-13

Left main coronary artery intervention and transcatheter aortic valve implantation (TAVI) in cardiogenic shock

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Introduction: Left main coronary artery stenosis and severe aortic stenosis have been considered to be the most life threatening conditions for years. Urgent recognition and intervention is very important for survival.Few treatment options were available. Recent advances in interventional cardiology gives promising results for catheter based therapy(TAVI and complex coronary interventions).TAVI after Left main coronary artery intervention can be lifesaving solution in a high risk patient. Our case report illustrates TAVI after left main coronary artery intervention in a patient with cardiogenic shock.

Case Report: 92 year old male admitted to emergency department with shortness of breath (NYHA3) and stabbing chest pain radiating to neck, left shoulder. Past medical history included hypertension. Midsystolic ejection murmur was heard from all borders on physical examination. Bilateral rales were heard at the upper portion of lungs. Electrocardiography showed normal sinus rhytm, left axis deviation and left anterior fascicular bundle branch block and negative T waves on precordial leads. Transthoracic echocardiography revealed left ventricular hypertrophy, ejection fraction: 45%, anterior hypokinesia, sclerosis of aorta and severe aortic stenosis (Aortic valve area:0,9 and 42/64 mmhg gradients). He was classified as high risk for surgery by the heart team. (Euroscore: 23%, STS:16%) The treatment strategy was TAVI after coronary angiography. 80% distal stenosis of LMCA, 90% osteal stenosis of LAD, 90% osteal stenosis of CX were found on coronary angiography (Figure 1-2). While he was in coronary care unit hemodynamic parameters detoriated and he was intubated due to respiratory arrest. He was retaken to catheter laboratory. 3.0x30 DES was implanted from LMCA-LAD, 3.5x24 DES was implanted from LMCA-CX using Culotte technique. Post dilatation of LMCA was achieved with 5.0x10 noncompliant baloon. (Figure 3, 4). After LMCA intervention Transesophageal echocardiography revealed severe aortic stenosis with restricted movement aortic leaflets. Aortic root, left ventricular outflow tract were evaluated. 26 mm Edwards-Sapiens XT was implanted via femoral route without detailed evaluation with CT-angiography (Figure 4, 5). The patient recovered completely.

Discussion: TAVI is an alternative for sugery for severe symptomatic aortic stenosis in high risk patients. There are still gray zones like low risk, intermediate risk patients and emergency situations. TAVI after LMCA intervention can be a lifesaving solution in cardiogenic shock without detailed evaluation with ct- angiography. But TEE should be performed with three dimensional measurment of aortic root, left ventricular outflow tract. Sizing of the valve is very challenging.
Conclusion: TAVI with LMCA intervention can be a lifesaving solution in cardiogenic shock.

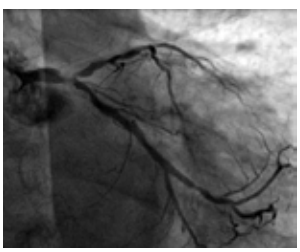

Figure 1. LMCA stenosis. Coronary angiography showed 80% distal LMCA stenosis, 90% osteal LAD stenosis, 80% osteal CX stenosis.

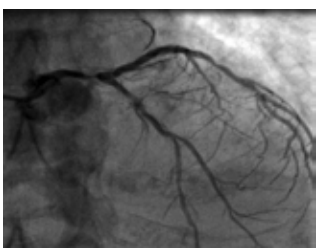

Figure 2. LMCA stenosis 2. Coronary angiography showed 80% distal LMCA stenosis, 90% osteal LAD stenosis, 80% osteal CX stenosis.

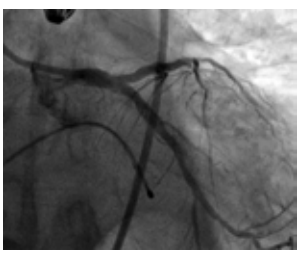

Figure 3. LMCA stenting. Lmca-Lad and Lmca-Cx were stented using Culotte technique.

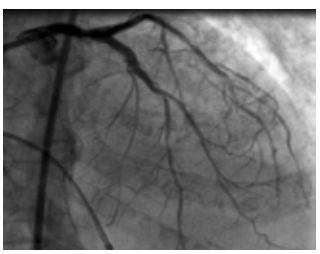

Figure 4. LMCA stenting 2. Lmca-Lad and Lmca-Cx were stented using Culotte technique.

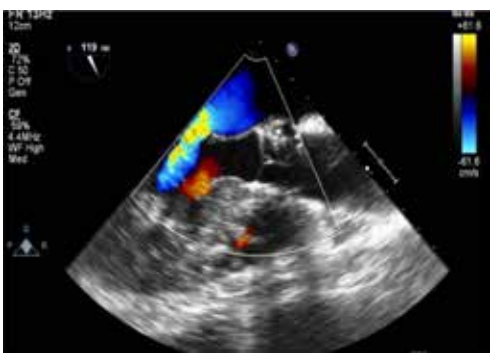

Figure 5. TAVI echocardiography. Control echocariography after TAVI.

OS-14

Aort darlığı ve abdominal aort diseksiyonu bulunan hastada diseksiyonun aortik greft stent ile endovasküler tamiri sonrası başarılı transfemoral aort kapak implantasyonu

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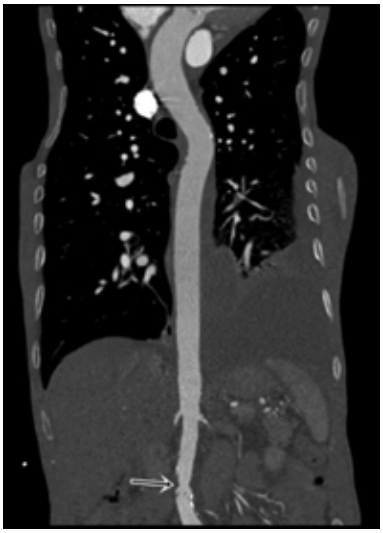
Giriş: Transkatater aort kapak implantasyonu (TAVİ) için seçilen hastalarda abdominal aort anevrizması (AAA)/diseksiyonu gibi ek hastalıklar eşlik edebilmektedir.Kritik aort darlığı (AD) varlığında abdominal aort cerrahisi son derece yüksek risklidir.AAA/diseksiyonu rüptür riski, TAVİ'nin perioperatif döneminde kan basıncında artış nedeniyle önemli ölçüde artabilir. Bu yazıda abdominal aortada diseksiyon ve aort lümeninde daralma bulunan;endovasküler aort tamiri (EVAR) sonrası TAVİ yapılmış olgu sunuldu.

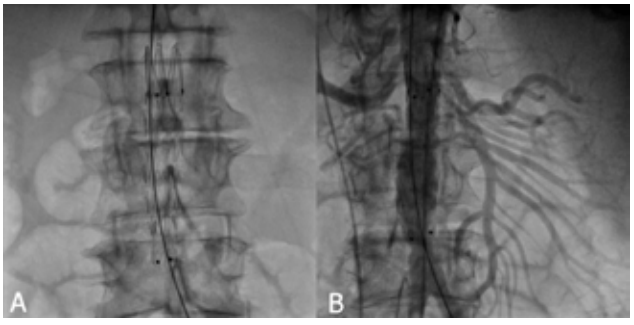
Olgu Sunumu: Yetmiş altı yaşında erkek hasta uzun süredir olan karın ağrısı, 3 aydır olan ve 2 gündür artan nefes darlığı ile başvurdu. Fizik muayenede aort odağında 3/6 sistolik üfürüm, her iki akciğerde krepitan ral, 2+ pretibial ödem saptandı. Hasta kalp yetmezliği tanısıyla servise yatırıldı ve tedavi başlandı. HT, DM ve kronik böbrek yetmezliği de olan hastanın ekokardiyografisinde ejeksiyon fraksiyonu (EF) %45, ciddi AD(kapak alanı 0.55 cm^2, peak/mean gradient: 74/43 mmHg), 1. derece aort ve mitral yetmezliği saptandı. Stabilizasyon sonrası aort kapak değiştirilmesi için cerrahi ile konsülte edilen hastada operasyonun yüksek riskli olduğu belirtilmesi üzerine TAVİ planlandı. TAVİ öncesi çekilen bilgisayarlı tomografik (CT) anjiografilerde koroner arterlerin plaklı olduğu, abdominal aortada diseksiyon flebi ve gerçek lümende darlık bulunduğu saptandı (Şekil 1). TAVİ'de kullanılacak geniş sheathin disseke aortik segmentten geçişi sırasındaki yüksek rüptür riski nedeniyle aynı seansta EVAR+TAVİ uygulanması kararlaştırıldı. Genel anestezi altında 23x23x70 mm greft stent disseke aortik segmente yerleştirildi (Şekil 2a). Sonrasında çekilen aortografide endoleak izlenmedi (Şekil 2b). Sonra TAVİ işlemine geçildi, hızlı pacing altında aort kapağa balon dilatasyon yapıldı, 26 no balon expandable bioprotez kapak aortik pozisyonuna ilerletildi ve hızlı pacing desteği altında yerleştirildi (Şekil 3).Taburcu edilen hastanın 3. ay kontrol ekokardiyografisinde EF'nin %60'a yükseldiği, aort bioprotez kapak peak/mean gradientin 11/6 mmHg olduğu saptandı.

Tartışma: TAVİ planlanan hastalarda AD'na sıklıkla çeşitli ek hastalıklar eşlik etmektedir. AAA/abdominal aort diseksiyonu bulunan hastalarda işlem sırasında kapak ilerletilirken veya TAVİ sonrası artan sistolik ve ortalama kan basıncı nedeniyle aortik rüptür riski artmakta, bu nedenle bu tür hastalarda TAVİ öncesi abdominal aort tamiri yapılması önerilmektedir. Literatürde aynı seansta EVAR+TAVİ uygulanan tek vaka bildirilmiştir. Bu vakada fusiform AAA bulunan hastaya aynı seansta EVAR+TAVİ uygulanmıştır. Bizim vakamızda ise abdominal aort diseksiyonu ve disseke segment gerçek lümende daralma olan hastaya aynı seansta EVAR+TAVİ uygulanmıştır. Bu özelliği ile vakamız literatürdeki ilk vaka olma özelliğini taşımaktadır.

Sonuç: TAVİ planlanan hastalarda eşlik edebilecek AAA/aort diseksiyonu gibi patolojiler nedeniyle CT anjiografik değerlendirme yapılmalı ve AAA/aort diseksiyonu saptanan hastalarda aortik rüptür riskini azaltmak için TAVİ öncesi EVAR yapılmalıdır.

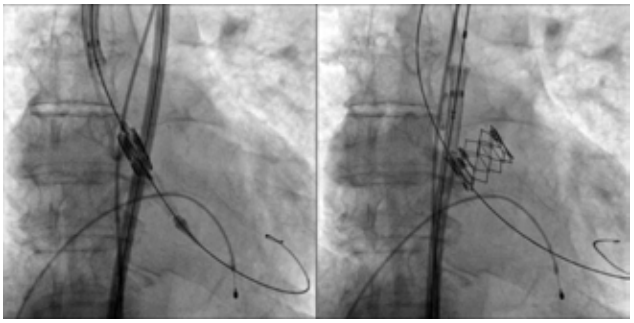


Şekil 1. Bilgisayarlı tomoğrafide abdominal aorta diseksiyonu ve gerçek lümende darlık.



Şekil 2. (A) Abdominal aortadaki disseke segmente aortik greft stent yerleştirilmesi. **(B)** Aortik greft stent yerleştirilmesi sonrası aortagrafi görüntüsü.

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalığı



Şekil 3. Transfemoral aortik bioprotez kapak yerleştirilmesi.

OS-15

Sinüs valsalva anevrizma rüptürünün perkütan kapatılması

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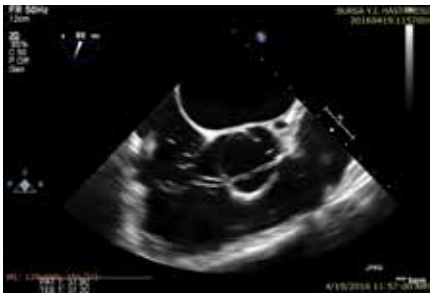
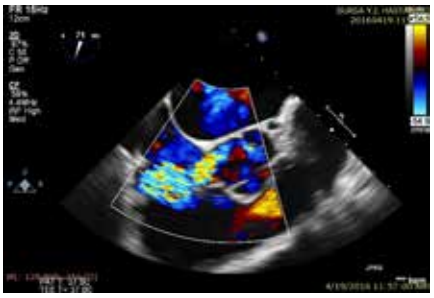
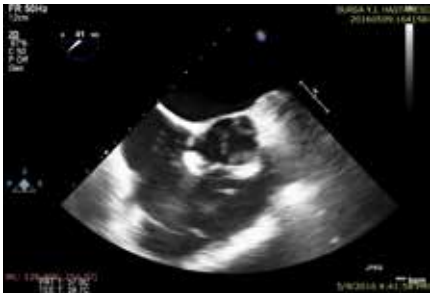
Otuz iki yaş erkek hasta yaklaşık bir aydır olan nefes darlığı, çarpıntı, çabuk yorulma şikayeti ile dış merkezden VSD ön tanısı ile tarafımıza refere edilen hastanın yapılan fizik muayenesinde aortik odaktan başlayıp sternum sağ tarafa yayılan 3/6 sistolo-diastolik devamlı üfürüm izlendi. Yapılan trans torasik ekosunda sinüs valsalvadan sağ atriuma renkli dopplerde geçiş izlendi. EF normal, ventriküler septum intakt izlendi. Hastanın yapılan trans özefagial ekosunda sinüs valsalva anevrizmatik ve aortik non coroner cüspisten sağ atriuma uzanan defekt ve renkli doppler ile geçiş izlendi (Şekil 1, 2), (Video 2). Hastaya yapılan koroner anjiorafide koroner arterler normal izlendi. Yapılan aortografide sinüs valsalvadan sağ atriuma uzanan geçiş izlendi. Yapılan sağ kalp kateterizasyonunda Qp/Qs 1,55 hesaplandı. Yapılan hastane konseyinde perkütan kapatma kararı alındı. Hasta ve yakınlarından gerekli onamlar alındıktan sonra arter ve ven yolu ile girilerek aortadan non koroner cüspisteki rüptüre anevrizmadan sağ atriuma geçildi. Snare ile wire inferior vena cavada yakalandı ve arteriyo venöz loop yapıldı. Loop üzerinden taşıma katater ile rüptüre anevrizmadan aortaya iletildi.Sonrasında 10x12 mm Memopart PDA kapama cihazı yerleştirildi rezidü kalmadı. (Şekil 3) (Video 1-3).



Şekil 1.



Şekil 2. NCC anevrizması.



Şekil 3. Perkütan kapama sonrası.

OS-16

Glue ile koroner fistül kapatma olgusu

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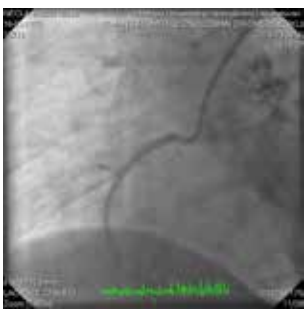
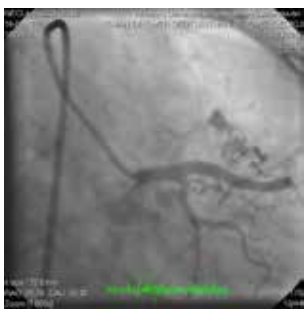
Giriş: Koroner arteriovenöz fistüller, koroner arterlerin konjenital anomalisidir. Çok büyük sıklıkla sağ koroner arter de rastlanır ve büyük bir kısmı kalbin sağ bölümüne açılır %56 RCA, %36 LAD, %5 RCA+LAD, nadiren CX %3 oranında ise tek koroner arter ve fistül bulunduğu anlaşılmıştır. Fistüller %39 oranında sağ ventrikül, %33 oranında sağ atrium (koroner sinüs, superior vena kava dahil), %20 oranında pulmoner arter, %6 oranında sol atrium ve %2 oranında sol ventrikül içine açılmaktadır. Hastaların çoğunda asemptomatik bir dönem vardır. Semptomlar fistülün genişliğine ve sol sağ şantın miktarına bağlıdır. Başka bir patolojinin birlikte oluşu da semptomları ve hastalığın gidişini etkiler. Efor dispnesi. Taşikardi, yorgunluk, anginal tipte ağrılar. Konjestif kalp yetersizliği, bakteryel endokardit, mİyokard enfarktüsü ve pulmoner hipertansiyon yaş ilerledikçe ortaya çıkacak komplikasyonlardır.

Olgu Sunumu: Kırk altı yaşında bayan hasta. İki yıldır Yokuş yukarı çıkmakla olan sol kola yayılan 2-3 dk süren, baskı tarzı göğüs ağrısı ve nefes darlığı ve Eforla ilşkisiz olan 2-3 dk süreli aralıklı gelip geçen asemptomatik çarpıntı şikayeti ile başvurdu. Şikayetleri, son 2 aydır artmıştı. Hikayesinde Annede kalp kapak hastalığı vardı. Ekg: Nsr hr: 68/dk pr: 160 msn Qrs: 90 ms Tansiyon: 125/65 Apikal 2/6 şiddetinde sistolik üfürüm(+) Biyokimyasal parametrelerde belirgin patoloji yoktu. Sağ sol selektif koroner anjiografide LAD ve RCA ile pulmoner arter arasında fistül izlendi hastaya ayrıntılı anatomik inceleme için koroner BT anjiogarfi yapıldı (Şekil 1). LAD 7f JL4 kateter eşliğinde flopy tel klavuzluğunda mikrokatater ile GLUE (cyanoacrylate) enjeksiyonu yapıldı. Sonrasında akımın kesildiği tespit edildi. Sağ taraflı fistül için balon şişirilerek güvenli bölge tespit edilip filder tel klavuzluğunda mikrokatater ile GLUE (cyanoacrylate) enjeksiyonu yapıldı. İşlem sonrasında hastada nefes darlığı ve göğüs ağrısı oldu kardiyak marker, EKG takibi ve ventilasyon perfüsyon sintigrafisinde patoloji izlenmedi.

Sonuç: Koroner fistülerde semptom olsun olmasın komplikasyonlarından dolayı kapatma önerilmektedir. Transkatater kapatma (Antegrad, Retrograd) ve Cerrahi kapatma kulanılan yöntemlerdir. Kapatma işleminde çeşitli cihazlar ve cihaz, GLUE birlikte kullanılmıştır ancak literatürde daha çok intrakranial patolojilerde tercih edilen GLUE ile tek başına fistül kapatma işlemi yapılmamıştır.Bizim vakamızda güvenli olarak kapatama işlemi yapıldı.



Şekil 1. GLUE ejeksiyonu sonrası fistül görüntüsü.



Şekil 2. GLUE enjeksiyonu sonrası fistül görüntüsü.

OS-17

Giant coronary fistula to left ventricle

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Coronary artery fistulas are rarely seen and they have been identified for the first time in 1865. More than half of them are originated from the right coronary artery. Only a few fistula drain into the left side of the heart. Fistulas can be congenital or acquired due to trauma or surgery. 62-year-old woman who underwent surgical closure of atrial septal defect 40 years ago presented with dyspnea, atypical chest pain and pretibial edema for almost one week. A grade 4/6 systolic murmur was heard at mitral area and apex. The symptoms and other physical examination findings thought to be consistent with heart failure. An electrocardiogram showed atrial fibrillation and there was no ischemic evidence. Transthoracic echocardiography revealed severe mitral and tricuspid regurgitation and left ventricular ejection fraction of 64%. Since the patient described chest pain, coronary angiography was performed before surgery. Left anterior descending artery was extremely aneurismatic and tortuous, and a fistula was found between left anterior descending artery and left ventricle. After signs and symptoms of heart failure improved, the patient was transferred to surgery. The majority of fistula are congenital and asymptomatic. Symptoms may also occur in relation to the size of fistula. Only a few of fistula is terminating in the left ventricle. If fistulas drain into the left ventricle, they may cause hemodynamic results which are similar to aortic insufficiency. Clinically, myocardial ischemia or high output cardiac failure has been identified due to fistula. In symptomatic patients, surgical or interventional therapies (coil placement) are available. This case illustrates a rare extremely tortuous and aneurysmal left anterior descending artery due to coronary fistula to the left ventricle.

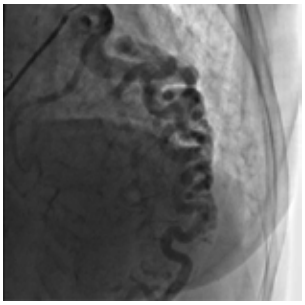

Figure 1. Extremely aneurismatic and tortuous left anterior descending coronary artery with fistula.

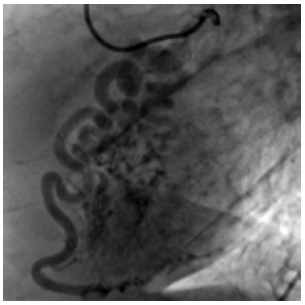

Figure 2. Extremely aneurismatic and tortuous left anterior descending coronary artery with fistula.

OS-18

Dev anevrizma ile komplike olmuş aorta-sağ atrial tünelin başarılı perkütan kapatılması

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Yirmi yedi yaşında bayan hasta atipik göğüs ağrısı şikayeti ile başvurdu. Bilinen sistemik hastalık öyküsü olmayan hastanın muayenesinde sağ üst sternal kenarda en iyi duyulan 2-3/6 sürekli üfürüm mevcut. Transtorasik ekokardiyografide sağ atriuma komşu anevrizma kesesi izlendi. Transözofageal eko ile anevrizma kesesi izlendi. Ancak atriumla ilişkisi net değerlendirilemedi. Bunun üzerine Multislice BT çekildi (Şekil 1). Sol sinüs valsalva'dan köken alan ve uzun bir tünel sonrası anevrizma ile sağ atriuma boşaldığı görüldü. Tanısal aortografi ile de tanı doğrulandı. Cerrahi veya perkütan kapama mümkün olan vakada hasta cerrahi istememesi üzerine perkütan kapama kararı alındı. İşlem esnasında tünelin keskin dönüş açısı nedeniyle taşıyıcı kateter ilerletilemedi. Farklı taşıyıcı kateterler (Cook, Amplatz) denenmesine rağmen ilerletilemeyince kısa taşıyıcı uzun dilatör kullanılarak ilerlendi. Anevrizma öncesi tünel çapı: 12 mm, %50'ye kadar yüksek çaplı cihaz kullanılabildiği için öncelikle 16 mm Amplatzer vascular plug II denendi. Bu cihaz anevrizma kesesi içine düştü. Bunun ardından daha büyük bir cihaz (22 mm Amplatzer Vascular Plug II) yerleştirildi (Video 1). Kontrol aortografide anevrizmaya kan akımının kesildiği izlendi. Komplikasyon izlenmedi, işlem sonlandırıldı.

OS-19

Giant right atrial appendage aneurysm: diagnosis with cardiac magnetic resonance imaging

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Right atrial appendage aneurysms are rare abnormalities of the heart. Patients can be asymptomatic or can present with atrial arrhythmias, thromboemboli or dyspnea. We describe a young male patient with a giant right atrial appendage aneurysm where cardiac magnetic resonance (CMR) imaging added incremental value to the transthoracic echocardiography (TTE). Specifically, cardiac magnetic resonance imaging allowed us to identify the aneurysm with exact localization and size. A 22 year-old young male patient admitted to our hospital with palpitation. There was no past medical history. Cardiorespiratory examination was normal except a systolic murmur at the lower left sternal border. Electrocardiogram showed sinus tachycardia with heart rate 106 and normal P-wave morphology and QRS axis. TTE revealed the presence of a large, thin-walled aneurysmal structure in continuity with the free wall of the right atrium which was compatible with a possible right atrial aneurysm (RAA) (Figure 1a, b; Movie 1, 2). For further evaluation, the patient was referred to CMR imaging. Cardiovascular magnetic resonance imaging (1,5 Tesla Aera; Siemens, Enlangen, Germany) was performed. Cine imaging using a balanced steady-state free precession sequence and dynamic perfusion images revealed a noncontractile, smooth-walled, right atrial appendage aneurysm (RAAA) extending anterior to the body of the right atrium (Figure 2, 3, 4; Movie 3, 4, 5). The aneurysm had a 48 mm broad-base and the size of the aneurysm was measured as 103x43 mm. There was mild indentation at right ventricule wall. No thrombus was detected in the aneurysm cavity. A 1 – day cardiac rhythm monitor revealed no evidence of arrhythmia. According to there was no proved arrhythmia and the patient was asymptomatic, we decided conservative management with anticoagulation for thromboemboli prophylaxis. During the close follow-ups patient was asyptomatic with no significant increase of RAAA size. Right atrial appendage aneurysms are rare abnormalities of the heart. Less than 20 cases of RAAAs have been reported till 2014. Most commonly patients present with palpitation and dyspnea, but some of them are asymptomatic. Potential complications of RAAA are atrial arrhythmias, thromboembolic phenomenon and rupture of RAAA. Surgery is recommended for symptomatic patients and whose RAAA size increases significantly on follow-ups. Anticoagulation is indicated for asymptomatic patients for thromboembolic prophylaxis and obtaining the sinus rhytm is recommended. In conclusion, CMR imaging appears to be a valuable supplement by more comprehensive and accurate delineation to TTE in evaluating the diagnosis and the prognosis. Thus, in conservatively managed patients, follow-up imaging with CMR imaging can be considered.

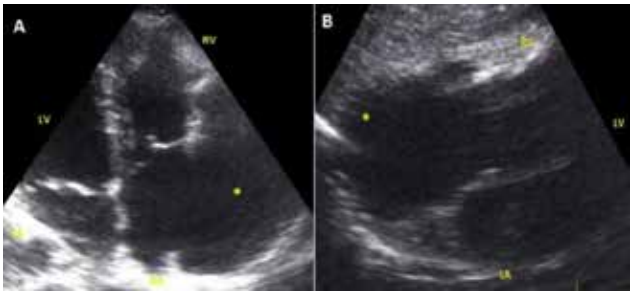

Figure 1. (A, B) Two- dimensional transthoracic echocardiography A. Four chamber view. Asterisk points possible right atrial aneursym. Left atrium (LA), left ventricule (LV), right atrium (RA), right ventricule (RV) are also seen. B. Subcostal view.

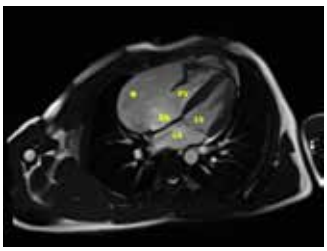

Figure 2. Four chamber balanced steady-state free precession sequence image demonstrates atrial appendage aneurysm (*) communicating with right atrium (RA), indenting right ventricule wall (RV). Left atrium (LA), left ventricule (LV) are also seen.

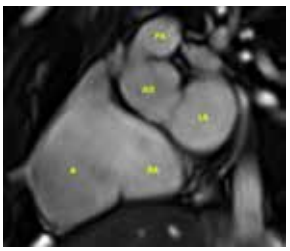

Figure 3. Short axis balanced steady-state free precession sequence image demonstrates atrial appendage aneurysm (*) communicating with right atrium (RA). Left atrium (LA), aorta (AO) and pulmonary artery (PA) are also seen.

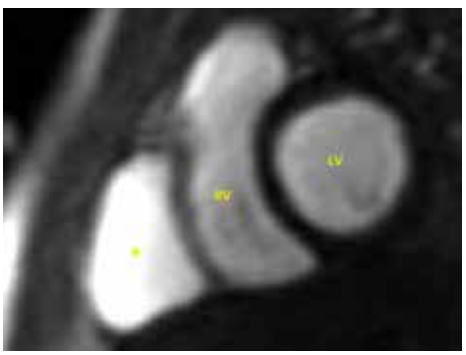

Figure 4. Dynamic perfusion short axis image. Right atrial appendage aneurysm (*), right ventricule (RV) and left ventricule (LV) are seen.

OS-20

Giant calcific pericardial cyst: Certainly unexpected during primary percutaneous coronary intervention

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A 50 years old male patient had admitted to our emergency department with chest pain and subsequently accepted to the catheterization laboratory with a diagnosis of acute anterior MI. While obtaining images for right coronary artery (RCA) a huge calcific mass bordering the RCA was noticed (Figure 1A). Following a percutaneous intervention to the left anterior descending artery, the patient was evaluated for the location and the nature of the calcific mass. His occupation, as well as past medical history was unremarkable, and had no previous symptoms compatible with cardiac or pulmonary compression. Two and three dimensional echocardiograms obtained from various windows, including subcostal 4-chamber view did not show any mass compatible with the image obtained from the angiogram, and it was decided that the location of the mass was outside the pericardium. A high-resolution thoracic CT scan, however, suggested that the calcific mass was cystic, located intrapericardially and had a bilobar morphology extending from superior vena cava (SVC) to the right atrium (RA) and right ventricle (RV) (Figure 1B and 1C). To further evaluate the mass, 2D and 3D transoesophageal echocardiograms was performed with a Phillips iE33 ultrasound platform (Phillips Healthcare, Andover, MA). 2D echocardiograms obtained from modified four-chamber and bicaval views showed two calcified masses compressing the right atrium RA and the SVC, with partial obstruction within caval flow (Figure 1D). Cropping of the 3D datasets allowed visualization of the bilobar mass to its full extent, beginning from the SVC and reaching to the RV (Figure 1E and 1F). Compression of the superior vena cava was also noted on 3D views. As laboratory investigations showed negative antibodies for Echinococcus spp., calcified pericardial cyst was considered as the most probable diagnosis. Surgical removal of the mass was considered for definitive diagnosis of the condition, but the patient refused further interventions. Unlike previous cases, where the diagnosis was sought due to right ventricular failure symptoms, present case shows an asymptomatic phase of the disease in which the diagnosis was suspected during an emergent coronary angiogram.

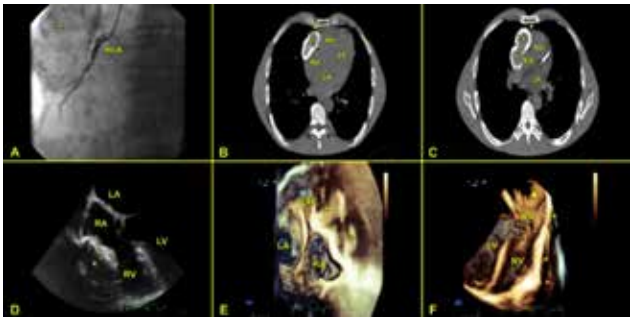

Figure 1. Images of calcific pericardial cyst obtained with different modalities. Panel **A** shows the calcific cyst (marked with asterisk) first noticed during the coronary angiography. Panels **B** and **C** shows the cyst as seen on CT scans. The pericardial lining covering the cyst is shown with arrows. The linear opacity seen at interventricular septum in Panel **C** is due to stent in left anterior descending artery. Two- and three-dimensional transesophageal echocardiographic images of the cyst are given in Panels **D-F.** Arrowhead in Panel **F** shows occlusion of the ostium of the superior vena cava during inspiration due to compression of the cyst. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle; SVC, superior vena cava.

OS-21

Percutaneous mitral annuloplasty in a patient with coronary sinus perforation; it is not an obstacle

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A 69-year-old male with nonischemic heart failure and severe funtional mitral regurgitation(FMR) was referred to our clinic. He had diabetes mellitus (insulin dependent for 10 years), hypertension for 2 years and he was severely symptomatic (NHYA class III). There was bilateral iliac stenting on his previous history. There was bilateral inspiratory rales on basis of the lungs and 3/6 systolic sufl on mitral area. Left bundle branch block (QRS duration: 205 msn) was seen on his electrocardiogram. There were left ventricular dilatation(enddiastolic diameter: 67 mm, endsystolic diameter: 58 mm), global hypokinesia, systolic dysfunction (ejection fraction 25%), and severe FMR on transthoracic echocardiography. Despite the optimal medical treatment, the patient was stil syptomatic and because of the high surgery risk, we decided to implant the percutaneous mitral annuloplasty device with the Carillon system (Cardiac Dimensions Inc., Kirkland, WA, USA) to the patient. Underfluoroscopy, 9F guiding catheter was introduced into the coronary sinus(CS) via right internal jugular vein. But we could not forward the delivery catheter over the hydrophylic wire. After that, we tried to use a agilis catheter to cannulate the distal part of the CS. But, it was gone towards the side branch. Next, we noticed that the patient was hypotensive and tachycardic and there was mild to moderate pericardial effusion with no cardiac tamponade. There was a perforation on distal part of the CS with left anterior oblique-caudal projection because of the side branch cannulation with steerable agilis catheter. After that, the procedure was stopped and the patient was transferred to the coronary care unit. The patient was monitored for 24 hours and there was no cardiac tamponad. Pericardial effusion was regressed. After 2 weeks later, the patient was transferred to the same procedure. Underfluoroscopy, 9F guiding catheter was introduced into the coronary sinus (CS) via right internal jugular vein again using a stiff wire and after the coronary sinus angiography with marked pigtail catheter, the distal anchor of the device was released. After the application of adequate tension, which resulted in a decrease in the degree of MR on echocardiography, no compression was seen on the CX artery. The proximal and distal anchors of the device were released into the CS with enough plication. After this procedure, the echo parameters of FMR further improved. Vena contracta diameter of the FMR and annular dilatation were also diminished. 2 weeks later the mitral annuloplasty procedure, CRT-D device was implanted to the patient through the Carillon device. The QRS duration was also regressed (136 msn). Six minute walking test value was also improved (before all the procedure 140 m, after 275 m). The patient's functional class was also improved. In conclusion, CS side branch perforation is not an obstacle to implant the Carillon device and CRT device.

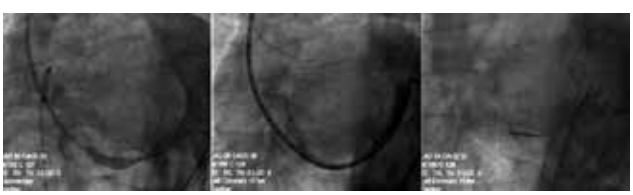

Figure 1.

OS-22

İskemik VT'ye neden olan multiple koroner arter fistülünün vasküler plug ile kapatılması

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Elli altı yaşında kadın hasta çarpıntı ve atipik anginal yakınma ile kliniğimize başvurdu. Hastanın özgeçmişinde hipertansiyon, 10 yıl önce konstrüktif perikardit nedeniyle operasyon, 6 yıl önce HSS(Hasta sinus sendromu) nedeni ile DDD-R pace maker takılması öyküsü mevcuttu. Çarpıntı ve anginal yakınma ile 2 yıl önce yapılan koroner angiografinin normal olduğu söylenmiş,ancak şikayetlerin giderek artması üzerine hasta kliniğimize başvurmuştu. Hastanın EKG'si normal sınırlarda ve yapılan EKO'da EF(Ejeksiyon fraksiyonu) %56 LV ve RV çap ve fonksiyonları normal, TYvelositesi: normal saptandı. Hastaya iskemi şüphesi ile yapılan MPS'de inferior duvarda efor iskemisi ile uyumlu bulgular saptandı. Çarpıntısı olan hastaya yapılan holter EKG'de bir çok kez Non-sustained VT atakları izlendi. hastaya koroner görüntüleme yapılmasına karar verildi. Radial yolla 6F sheath üzerinden KAG yapıldı. Sağ koroner arterde bir çok küçük fistülün ortak bir ampullada birleşerek pulmoner artere drene olduğu görüldü ve kalp takımı tarafından hastaya perkütan kapama yapılması kararı alındı. Femoral arter ve ven 6F sheath yerleştirildi, venöz sheath üzerinden multipurpose kateter ile pulmoner artere ulaşıldı. Kapama öncesinde pulmoner arter, sağ ventrikül ve fistul komşuluğundaki pulmoner arter bölgesinden kan gazı örnekleri alındı. Kateter içerisinden ilerletilen 260 cm'lik 0.038 inç hidrofilik klavuz tel ile fistüllerin drene olduğu ortak ampullaya ulaşıldı. Multipurpose kateter çıkarılarak 6F Judkins sağ koroner guiding kateter ilerletildi ve ardından klavuz tel çıkarılarak ampullanın en dar yerine 6 mm'lik Amplatzer vaskuler plug yerleştirildi. Sağ koroner arterden ve pulmoner arterden yapılan mükerrer enjeksiyonlarda fistülden akımın tamamen kesildiği izlendi. Kapama sonrası fistül komşuluğundan tekrar kan gazı örneği alındı ve kapama öncesi 96 olan oksijen saturasyonunun 73'e gerilediği izlendi. İzleminde semptomu olmayan hasta ikinci gününde eksterne edildi. Hastanın takibinde işlemden 2 ay sonra yapılan 3D BT'de fistülün tamamiyle kapalı olduğu izlendi.

Tartışma: Konjenital koroner fistüller embriyolojik dönemde kalbin beslenmesini sağlayan sinusoidlerin ortadan kalkmaması sonucu ortaya çıkarlar. Edinsel olanları daha çok kardiyak cerrahi sonrası oluşur. İki koroner arter arasında veya mediastinal başka bir yapı ile bağlantı oluşabilir. Koroner angiografi yapılan hastaların %0.1-0.2'sinde saptanır. Koroner fistüllerin %90'ından fazlası sağ kalbe veya ona bağlı damarlara açılır. Şant patofizyolojisi gözlense de nadiren önemli düzeyde olur. En sık semptom efor dispnesi ve yorgunluktur, angina nadir semptomdur. Fizik muayenede devamlı üfürüm duyulabilir. Kardiyomegali ve pulmoner kanlanmada artış görülür. Semptomatik koroner arter fistulleri çogunlukla perkutan yolla coilizasyon veya cerrahi olarak kapatılır. Bu vakada sağ koroner arterden pulmoner artere mutiple fistülü olan bir hastada retrograd yolla pulmoner arterden vaskuler plug ile perkütan kapama işlemi yapıldı.

OS-23

Patent foramen ovaleden geçen biatrial transit trombüs: İki vaka örneği

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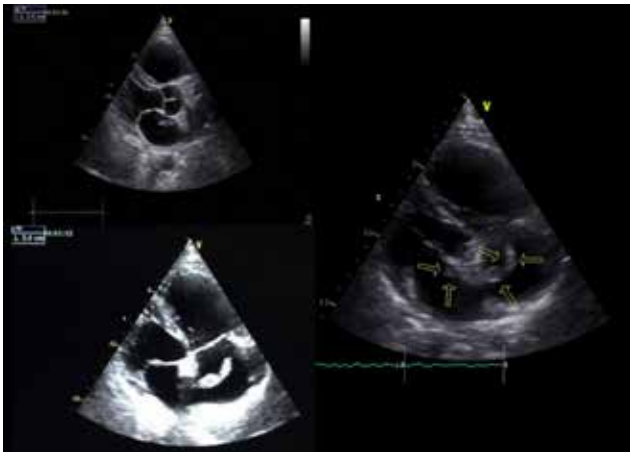
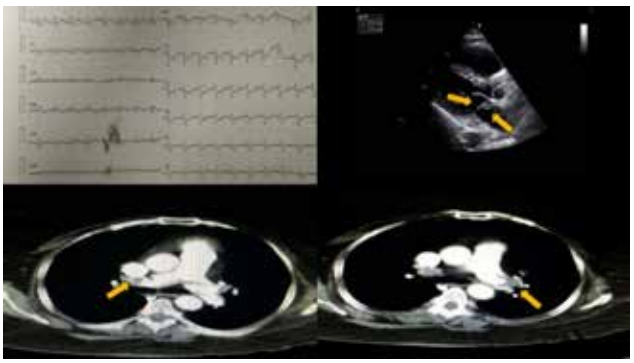
Elli bir yaşında erkek hasta acil servise nefes darlığı, göğüs ağrısı ve senkop şikayetleri ile başvurdu. Hastanın alınan anamnezinde derin ven trombüsü öyküsü olduğu, bir süre varfarin kullanımının ardından asetilsalisilik asitle takipte olduğu öğrenildi. Hastanın yapılan transtorasik ekokardiyografisinde sağ atriumdan patent foramen ovale aracılığı ile sol atriuma uzanan trombüs ile uyumlu görünüm izlendi. Sol atrial parça 2.1 cm, sağ atrial parça 3.4 cm ölçüldü (Şekil 1, video 1). Sistolik pulmoner arter basıncı 55 mmHg olan hastanın pulmoner hipertansiyonu trombüs kaynaklı emboliye bağlandı. Sistemik ve/veya pulmoner emboli riski nedeni ile hastaya trasnözefagial ekokardiyografi yapılamadı. Hemodinamik olarak stabil olan hasta kalp-damar cerrahisi ile konsülte edilerek ameliyat kararı alındı. 71 yaşında bayan hasta ani başlayan nefes darlığı ile acil servise başvurdu. Alınan anamnezinde 5 ay önce inoperabl serebral tümör tanısı olan hastanın bu kitleye bağlı sol hemiplejisinin olduğu öğrenildi. Fizik muayenesinde taşikardisi, triküspit odakta 3/6 sistolik üfürümü olan kan gazında hipoksi saptanan EKG'de S1Q3T3 olan hastaya pulmoner trombo emboli öntanısı ile çekilen bilgisayarlı tomografide bilateral ana pulmoner arterlerde trombüs saptandı. Bunun üzerinde hastaya transtorasik ekokardiyografi planlandı. Yapılan transtorasik ekokardiyografide sağ kalp boşlukları geniş olarak saptandı. 2-3. dereceden triküspit yetmezliği üzerinden düşülen Doppler'de sistolik pulmoner arter basıncı 65 mmHg olarak ölçüldü. Anevrizmatik interatrial septumdan sol ariyum yönünde uzanan diyastolde sol ventriküle doğru hareket eden 0,9x2 cm ebatlarında trombüsle uyumlu görünüm izlendi (Şekil 2, video 2). Mevcut pulmoner embolinin transit trombüsün sağ atrial kısmının kopması sonucu geliştiği düşünüldü. Sistemik emboli riski nedeni ile hastaya trasnözefagial ekokardiyografi yapılamadı. Hemodinamisi instabil olması ve inoperabl serebral tümör nedeni ile hastaya trombolitik tedavi uygulanamadı. Kalp-damar cerrahisi ile konsülte edilen hastaya çok yüksek riskli cerrahi kararı verildi ancak hasta ve hasta yakınları tarafından kabul edilmedi. Transit trombus zemininde gelişen masif pulmoner emboli veya serebrovaskuler olayla sonuçlanan vakalarda esas tedavi hemodinamik ve cerrahi risk değerlendirme sonrası antikoagulan infüzyonu, trombolitik tedavi veya cerrahi embolektomi ve PFO'nun tamiri olup bu tedavi seçenekleri bireysel bazda değerlendirilip hastaya göre karar verilmelidir.



Şekil 1. Birinci vakada patent foramen ovaleden geçmiş olan biatrial trombüsün görünümü ve büyüklükleri.



Şekil 2. İkinci vakada EKG'de görülen S1Q3T3, pulmoner arter dallarındaki trombüs ve patent foramen ovaleden geçmiş olan sol atriumdaki trombüsün ebatları.

OS-24

Sağ atriyumda iki başlı in-transit trombus

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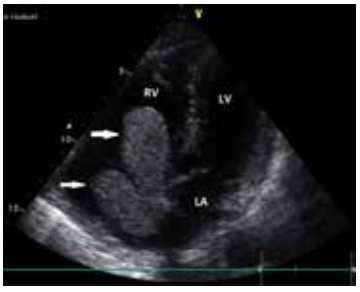
Seksen sekiz yaşında erkek hasta 2 haftadır olan bacak şişliği şikayeti ile kardiyoloji polikliniğinde değerlendirildi. Bilinen kardiyovasküler hastalığı olmayan hastanın renal kist nedeni ile 5 yıl önce operasyon öyküsü mevcuttu. Hemodinamisi stabil ve elektrokardiyografisi ise sinüs ritmi ve inkomplet sağ dal bloğu mevcuttu. Hastanın yapılan ekokardiyografisinde ise sol ventrikül ejeksiyon fraksiyonu %60 ve sağ atriyum içinde in-transit 2 başlı şekilde trombusle uyumlu görünüm izlendi (Şekil 1, Video 1). Ayrıca trombusun diyastolde triküspit kapaktan sağ ventriküle doğru hareketli olduğu, subkostal yapılan ayrıntılı incelemede ise trombusun inferior vena kava ağzına doğru uzandığı ve inferior vena kavanında karaciğer hattı boyunca trombus ile dolu olduğu gözlendi (Video 1, Video 2). Hasta kalp damar cerrahi bölümü ile konsulte edildi ve hastaya sağ atriyotomi yapıldı ve inferior vena kavaya uzanan 2 adet trombus çıkarıldı ve inferior vena kavadaki trombus çıkarıldı (Şekil 2). Hastaya venöz tromboz etyolojisi araştırılması planlandı. Akut pulmoner emboli dünyadaki en önemli ölüm nedenlerinden biridir. Pulmoner emboli vakalarının %3-23'de sağ atriyumda intransit trombus görülebilir. Bu vakalarda pulmoner emboli gelişirse diğer pulmoner emboli vakalarına göre oldukça fatal seyredebilir ve cerrahi olarak trombus eksize edilmelidir. Biz bu vakada pulmoner emboli gelişmeden tanı alan ve cerrahi olarak çıkarılan in-transit sağ atriyal trombus olgusunu sunmak istedik.



Şekil 1. Apikal dört boşluk incelemede sağ atriyumda sağ ventriküle doğru uzanan iki başlı trombus görünümü.

Şekil 2. Sağ atriyotomi sonrasında çıkarılan 2 adet trombus izlenmektedir.

OS-25

Pulmoner ven trombozu, nadir bir dispne nedeni

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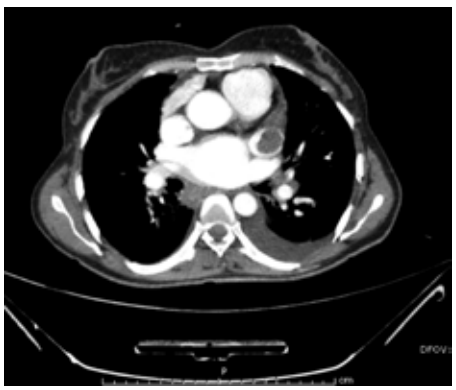
Giriş: Pulmoner ven trombozu (PVT) literatürde çok az sayıda vaka bildirilmiş nadir görülen pulmoner vasküler patolojidir. Genellikle atriyal fibrilasyon, mitral stenoz, pulmoner tümörler ve lobektomi komplikasyonu olarak ortaya çıkar. PVT öksürük, nefes darlığı ve plöretik göğüs ağrısı ya da ilerici ya da tekrarlayan pulmoner ödem kliniği ile prezente olabilmektedir. literatürde bazı nadir yayınlarda ciddi hemoptizinin tek başvuru sebebi olabileceği belirtilmiştir. Nonspesifik klinik ile başvuru olması nedeniyle nadir olarak tanı konmakta ve potansiyel ölümcül komplikasyonlara sebebiyet vermektedir. PVT klinik tanısı zordur ve genellikle pulmoner anjiyografi, transtorasik ekokardiyografi (TTE), transözofajiyal ekokardiyografi (TEE) ve BT gibi geleneksel tanı yöntemleri bir arada gerektirir. kardiyak kökenl, dispne düşünülen fakat standart medikal tedaviye yanıt vermeyen olgularda mutlaka ayırıcı tanıda PVT ye yer verilmesi uygun olacaktır.

Olgu Sunumu: 56 yaşında bayan hasta acile nefes darlığı ile başvurdu. Hastanın öyküsünde 10 yıl önce mitral kapak repair persistan atriyal fibrilasyon ve depresyon tanıları mevcut. hasta atriyal fibrilasyon tedavisi içi metoprolol 50 mg 1x1 ve asa 100 mg 1x1 almakta. fizik bakıda genel durum orta ateş 36.2 nabız 140, tansiyon 80/50, dakika solunum sayısı 40 puls oksimetre ile bakılan satğrasyon %87 dinlemekle sol akciğer alt zonda solunum sesi azalmış pa akcieğr grafisinde sol sinüste küntleşme mevcut. kan gazında hipoksi hipokarbi olan hastada ön tanı olarak pulmoner tromboemboli düşünüldü.acilde yapılan transtorasik ekokardiyografide ef 50 biatriyal dilatasyon romatizmal mitral kapak, orta derece mitral stenoz 14/7 gradiyent, kapak alanı pht 1.5 cm 2 planimetrik 1.4 cm^2 ölçüldü. tpab 60 mmhg olan hastaya kontraslı toraks bt planlandı. BT sonrası pulmoner arterler tamamen açık sol alt pulmoner vende trombüs ve total okluzyon saptandı. hasta koroner yoğun bakıma alındı ve heparin infüzyonuna başlandı. hastada 6 saat sonra satürasyon %90 üzerine çıktı ve dakika solunum sayısı 25 civarına indi. 24 saat sonra dispnenin tamamen gerilediğini gördük.

Sonuç: PVT tanısı zordur ve konvanssşyonel tanı araçlarını beraber kullanmayı gerektirir. etyolojide bazı kolaylaştırıcı faktörler olsada semptomları nonspesifik oluşu hekimi nadiren tanıya götürmektedir. Eğer standart tedaviye rağmen semptomlarında yeterince iyileşme sağlanamayan kardiyak dispne olgularında ayırıcı tanıda mutlaka pulmoner ven trombozuna yer vermek ve zaman kaybemeksizin antikoagülan tedaviye başlamak elzemdir. Eğer yeterince tedavi edilmezse pulmoner infarkt dışında sistemik emboli kaynağı hatta bazı yayınlarda stroke kaynağı olacağı vurgulanmıştır.



Şekil 1. Pulmoner ven trombozu. Sol alt pulmoner ven trombotik okluzyon.

OS-26

A young girl presented with cardiac thrombus: An unusual complication of inflammatory bowel disease

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Inflammatory bowel disease includes chronic inflammatory process of gastrointestinal tract, associated with extra-intestinal manifestations and complications. Thrombosis is a well-known complication of inflammatory bowel disease and a major cause of morbidity and mortality. A 16-year-old girl with history of Crohn's disease presented with ischemic stroke of the left middle cerebral artery and left anterior cerebral artery territory. Etiological workup disclosed mobile thrombus in the left atrium. Thrombus was removed successfully by the cardiovascular surgery team. Postoperative recovery was remarkable. After discharge, improvements were observed in the neurological deficits during the follow-up period. Patients with inflammatory bowel disease have an increased risk of thrombosis. Cerebral arterial thromboembolism is a less frequent complication. Early diagnosis is very important to consider therapeutic intervention. In this point, transthorasic echocardiaography is very useful and easily accessible tool for cardiac and pulmonary thrombosis.

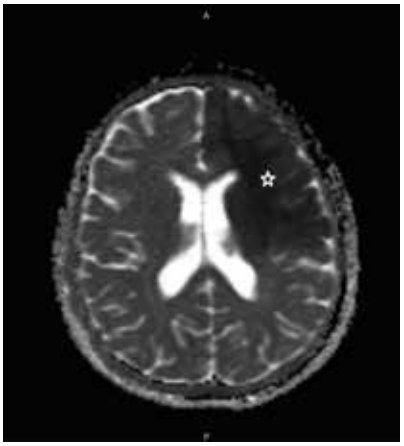

Figure 1. The cranial MRI of the patient revealing large ischemic lesions on the perfusion area of the left middle cerebral artery and left anterior cerebral artery without hemorrhage (white star).

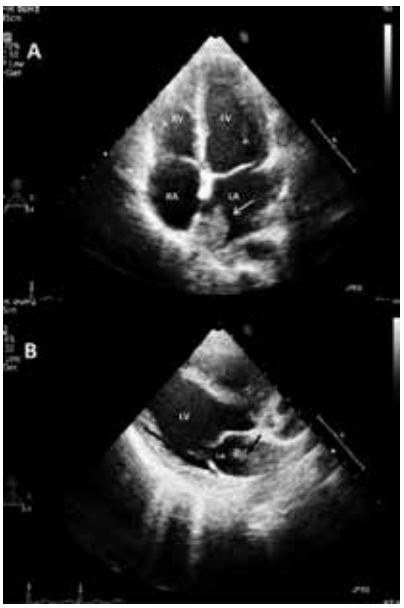

Figure 2. The two-dimensional echocardiogram revealing a mass in the left atrium in apical four chamber view (white arrow) **(A)** and parasternal long axis view (black arrow) **(B)**.

OS-27

Antithrombin III deficiency concomitant with atrial fibrillation results giant intracardiac thrombi in coronary artery disease patient

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Antithrombin III deficiency and atrial fibrillation respectively cause thromboembolic episodes result in morbidity and mortality. Primary protection is more important because the consequences are difficult to deal. Arterial thrombosis is far less common in antithrombin III deficiency. The prevalence of genetic antithrombin III deficiency is between 2-5/10000. On the other hand atrial fibrillation is a more frequent entity with increased frequency concomitant with age. Here we present 62 year-old asymptomatic male patient who had anterior myocardial infarction 6 months ago. After percutaneous coronary intervention to left anterior descending artery, he was discharged from another hospital with antiplatelet and antiagregant therapy. Even intracardiac defibrillator was implanted 2 months after the myocardial infarction.The electrocardiogram gained during the discharge was normal sinus rhythm. The patient applied to our outpatient clinic for a routine control, The transthoracic echocardiography conspicuously revealed thrombi in left ventricular apex, left atrium and right atrium aslo the electrocardiogram was atrial fibrillation (Figure 1) Transesophageal echocardiography was performed to put a precise diagnosis. Giant organized multiple thrombi in the left atrium accompanying thrombus in the left ventricle apex were detected (Figure 2). A giant thrombus hanging from superior vena cava to the right atrium was also revealed and intracardiac defibrillator was also thickened which was thought to be secondary to clot burden. (Figure 2, 3) Besides our patient was using klopidorel and acetyl-salicyclic acid. After immediate hospitalization, thrombophilia laboratory panel was obtained and heparin infusion started. Antithrombin III was interestingly detected as 55% of the reference level. Patient was consulted to the internal medicine department and heparin infusion treatment was carried on. After two weeks transesophageal echocardiography was again performed but the giant thrombi did not disappear and operation was offered to the patient. Patient refused the operation under informed consent and he was discharged from our hospital under warfarin, klopidogrel and acetyl-salicyclic acid therapy.

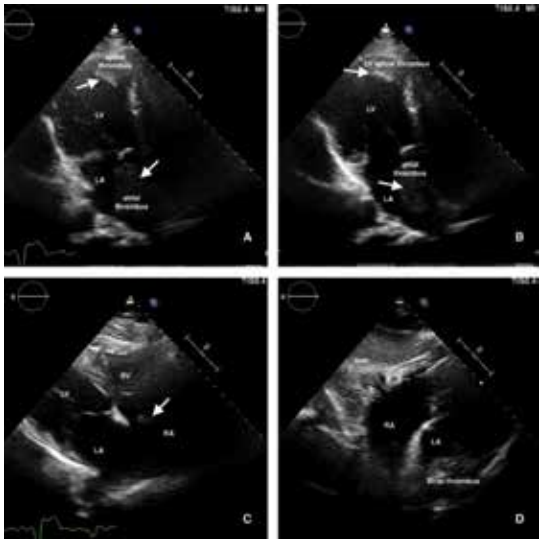

Figure 1. (A, B) Showing left ventricular apical and left atrial thrombi images of transthoracic echocardiography, **(C)** Showing right atrial thrombus in modified apical four-chamber view. **(D)** Shows left atrial thrombus in subcostal view.

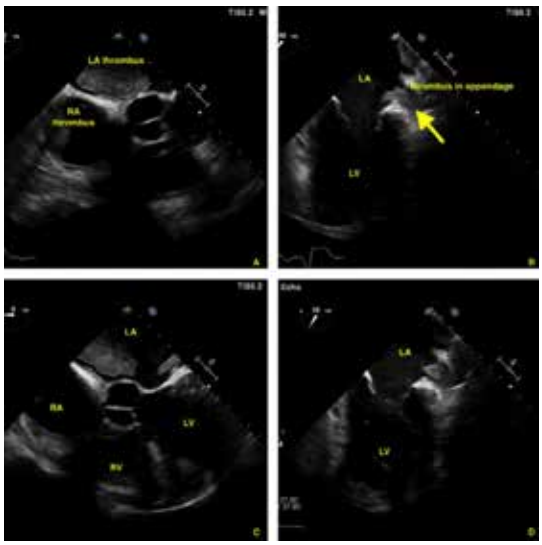

Figure 2. (A-D) Showing giant left atrial thrombi in transesophageal echocardiography.

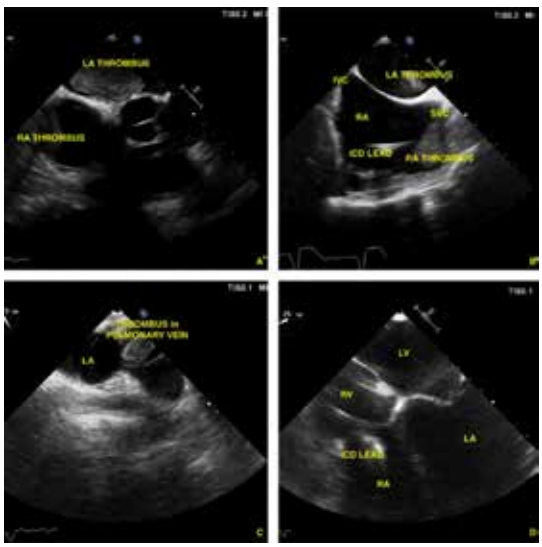

Figure 3. (A-D) Showing left atrial thrombus in transesophageal echocardiography, **(B)** showing left atrial thrombus and right atrial thrombus hanging from superior vena cava, **(C)** showing thrombus in pulmonary vein, **(D)** showing thickened ICD lead due to clot burden.

OS-28

Faktor V leiden mutasyonu olan hastada uzatılmış heparin ve vitamin-K antagonisti rejimleriyle erken mitral anuloplasti ring trombozunun başarılı tedavisi

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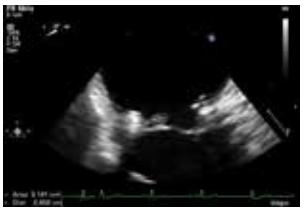
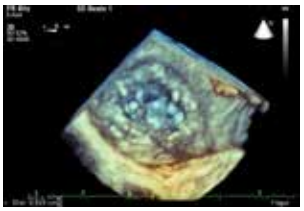
Yaklaşık 3 ay önce mitral ring annuloplasti operasyonu geçiren 62 yaşında kadın hasta sağ kolda uyuşma şikayetiyle polikliniğimize ayaktan başvurmuştur. Geliş inr:2.7 ve normal sinüs ritminde olup kardiyak muayenesinde ek bir özellik saptanmamıştır. Kalp hızı 70 atım/ dakika iken transtorasik (TTE) ve transesofageal ekoda (TEE) kapak alanı 1,5 cm^2 ve maksimum ve ortalama gradientler 10/ 5 mmHg ölçüldü. İki boyutlu ve 3 Boyutlu TEE' de mitral ring annuloplastinin mediyaline tutunmuş 9 mm büyüklüğünde mobil trombus izlendi (Video 1, Resim 1, Video 2, Resim 2). Yirmibir günlük intravenöz optimal heparin infüzyonu sonrası trombus kitlesinde % 50 azalma izlendi. Hiperkoagulabilite testlerinden homozigot faktor V leiden mutasyonu tespit edildi. Düşük doz aspirin ve etkin varfarin tedavisiyle hasta taburcu edildi. 4 ay sonraki kontrol TTE ve TEE' de sadece rezidüel trombus saptandı (Video 4). Ömür boyu etkin varfarin ve düşük doz aspirin tedavisi kararı verildi. Faktor V leiden mutasyonu gibi hiperkoagulabilite durumlarında trombus oluşumu için risk artışı vardır. Mitral ring anuloplastinin erken trombozunda heparin ve varfarin ile başarılı tedavi sağlanabilir.



Şekil 1. İki boyutlu TEE' de mitral annuloplasti ring trombusu.



Şekil 2. Üç boyutlu TEE' de mitral annuloplasti ring trombusu.

Diğer

OS-29

Takotsubo cardiomyopathy with left ventricular outflow trcat obstruction presented with sinus tachycardia

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45 years old female without known history of coronary artery disease presented at emergency department.She had complaints of dyspnea and chest pain. She mentioned complaints started with tachycardia few hours after emotional stress.On admission heart rate and blood pressure were 115 and 150/85 respectively. Physical examination revealed 2/6 systolic murmur on aortic area. ECG showed sinus tacycardia without any ST or T abnormality (Figure1). Echocardiography revealed left ventricular apical segment hypokinesia in contrast with basal segment hyperkinesia (Video 1). Color doppler displayed turbulent flow at left ventricular outflow tract (LVOT) whereas 93 mmHg gradient was measured with continuous doppler (Figure 2, Video 2). Systolic anterior motion of and mitral leaflet and subsequent mild mitral regurgitation was also remarkable. Troponin T level was 0.8 ng/mL. She underwent coronary angiography which showed normal coronaries. Ventriculography demonstrated apical ballooning and bazal segment hyperkinesia (Figure 3, Video 3). Acetylsalicilic acid 100 mg once and metoprolol 50 mg twice a day were prescribed. 3rd day ECG showed subtle terminal T wave negativity on V5-6 (Figure 4b). LVOT gradient disappeared on 4th day of admission albeit apical hypokinesia.Since patient's symptoms recovered and hospitalization period was uneventful she was discharged on 4th day. Control echocardiography on 21st day displayed recovered apical segment. Normal left ventricular outflow tract gradient was measured with continuous doppler. However ECG showed prominent T wave negativity (Figure 4c). Deep negative T waves disappeared and replaced with flat T waves on 70th day control (Figure 4d). Retrospectively analysing the patient's findings we established diagnosis of Takotsubo cardiomyopathy. Since pathopyhsiology of Takotsubo cardiomyopaty is still a unclear, definitive diagnosis could be sometimes challenging due to subtle and variable electrocardiographic presentations. Our case demonstrates TC with LVOT obstruction in a young female presented with tachcardia without ST segment elevation or T negativity. Follow up electrocardiographic and echocardiographic evaluations are essential to establish the true diagnosis in TC.

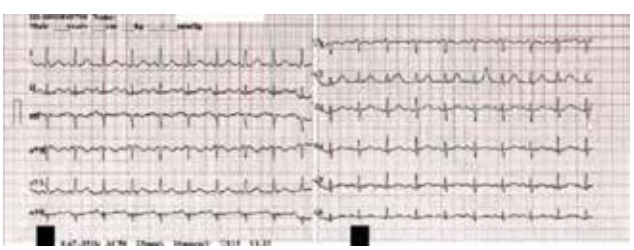

Figure 1. ECG of the patient on emergency service admission.

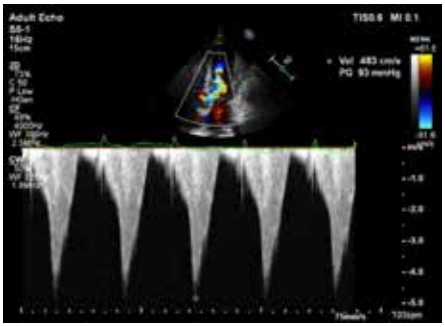

Figure 2. Transthoracic echocardiography apical 5 chamber view, left ventriculer outflow tract gradient was measured 93 mmHg.

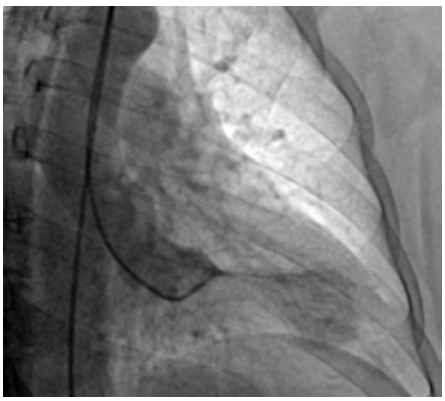

Figure 3. Ventriculography demonstrated apical ballooning.

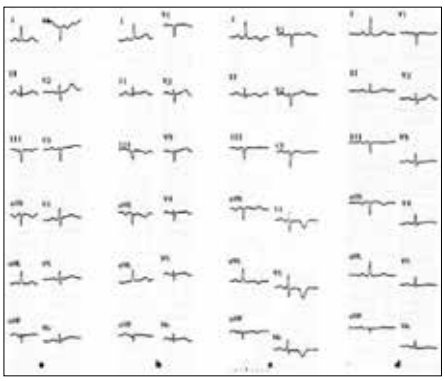

Figure 4. (A) 1st day ECG showed sinus tachycardia and subtle ST segment change in V4-5-6. **(B)** 3rd day ECG displayed subtle terminal T wave negativity in V4-5-6. **(C)** 21st day ECG showed prominent T wave negativity in V4-5-6 and I – AVL. **(D)** 70th day ECG, negative T waves disappeared, however they are still flat.

OS-30

Akciğer nakli sonrası ST değişikliği; Tako-Tsubo kardiyomyopatisi

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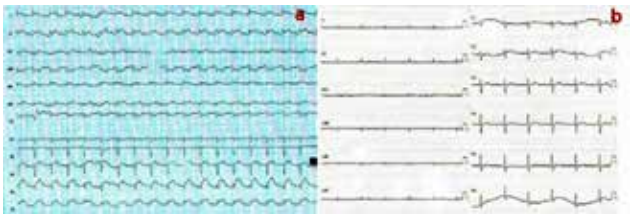
Giriş: Tako-Tsubo kardiyomyopatisi (TTK); epikardiyal koroner arter hastalığı, feokromasitoma ve myokardit olmadan, sol ventrikülde (SV) birden fazla segmentin geçici akinezi/diskinezisi ve EKG'de dinamik ST/Tdeğişikliğiyle karakterize akut kardiyak bir sendromdur. Yoğun bakım hastalarında sık görülmesi ve diğer kötü prognozlu akut kardiyak sendromlarla karışması nedeniyle, akciğer nakli sonrasında görülen dinamik EKG değişikliklerinin ayırıcı tanısında önem kazanmaktadır. Ancak, akciğer nakli ile ilişkili TTK daha önce az sayıda olguda bildirilmiştir.

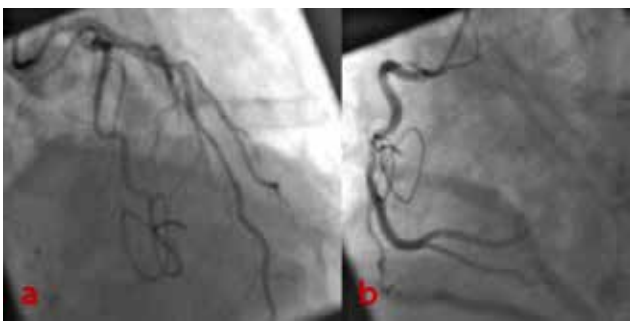
Olgu Sunumu: Amfizeme bağlı son dönem akciğer hastalığı ve sirkümfleks arterde stent öyküsü olan 61 yaşında erkek hastanın pre-op kardiyak değerlendirmesinde ekokardiyografide SV ejeksiyon-fraksiyonu(EF) %65 ve duvar hareketleri normaldi. Sağ kalp kateterinde ortalama pulmoner basınç19mmHg, pulmoner arter kama basıncı 10 mmHg idi. Kontrol koroner arter anjiyografisinde stentin açık olduğu ve koroner arterlerde başka anlamlı lezyonun olmadığı görüldü. Bilateral akciğer nakli sonrasında immünsüpresif protokol uygulandı. Entübe takip edilen hastanın post-op birinci günde mönitörde yeni gelişen ST yükselmesi görülmesi üzerine çekilen 12 derivasyon EKG'de inferior ve lateral derivasyonlarda ST elevasyonu görüldü. Ancak diğer derivasyonlarda ayna görüntüsü ST depresyonu gözlenmedi (Şekil 1a). Ardından sustained VT gelişti (Şekil 2) ve İV-amiadoron ile sinuzal ritme döndü. Yatakbaşı ekokardiyografide apikal, midventriküler segmentler akinetik; bazal segmentler ise hipokinetik olarak gözlendi ve sol ventrikül EF %30 hesaplandı. Acil koroner anjiyografi yapıldı ancak ST değişikliklerini açıklayacak yeni koroner patolojiye rastlanmadı (Şekil 3 a, b). EKG'deki ST değişikliği açıklayacak Troponin ve CK-MB yüksekliği saptanmadı. Takipte, EKG değişikliği ve SV duvar hareket bozukluğu geriledi ve EF'nin 15. günde %60'a yükseldiği gözlendi. Bu tablo TTK olarak yorumlandı.

Tartışma: Literatürde akciğer nakliyle ilişkili TTK sadece 2 olguda bildirilmiştir. Michel-Cherqui ve ark.nın olgu sunumunda akciğer nakli öncesi dönemde tanı alan TTK bildirilmiştir. Ghandrive ark.nın olgu sunumunda ise 7 yıl önce akciğer nakli uygulanmış, ancak solunum yetmezliği bulunan olguda bildirilmiştir. Her iki olguda da stres faktörü olarak hemodinamik instabilite, hiperkarbi ve hipoksi sorumlu tutulmuş. Olgumuz, post-op erken dönemdeki ilk olgu olup stres faktörü olarak erken dönem cerrahi stres, hipoansif atak sorumlu tutulabilir.



Şekil 1. (A) İnferior ve lateral derivasyonlarda ST elevasyonu olmasına rağmen diğer derivasyonlarda ayna görüntüsü ST depresyonu gözlenmedi; **(B)** Olgunun 6.ay kontrol EKG'si.

Şekil 2. SaO2 %94 olarak takip edilen olguda sustained VT atağı.



Şekil 3. (A, B) EKG'deki ST değişikliği açıklayacak yeni koroner patolojiye rastlanmadı.

OS-31

Bilateral koroner arter ostial darlığına neden olan sifilitik aortit vakası

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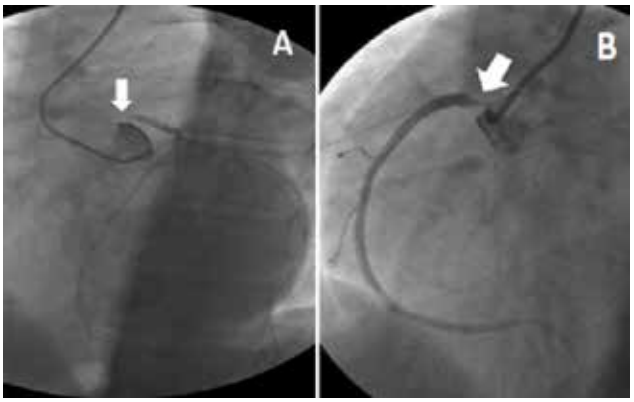
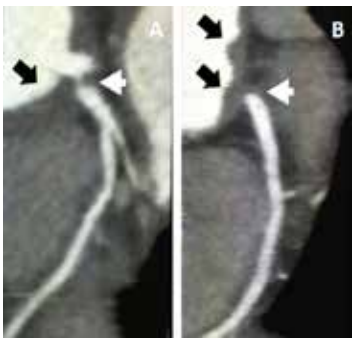
Koroner arterlerin diger kısımlarında darlık olmaksızın her iki koroner arterin ostial darlığı nadir görülen ve dikkatli inceleme gerektiren klinik durumdur. Tersiyer sifilis, daha çok tanısı gecikmiş olarak konulan hastalarda izlenen, günümüzde ender karşılaşılan hastalıktır. Bu evrede kardiyovasküler bulgular aort kökü tutulumu ve osteal koroner arter darlığı ile karakterizedir. Burada tipik anjina pektorisle başvuran ve koroner anjiyografide bilateral ostial koroner darlık saptanması üzerine yapılan incelemelerde sifilis tespit edilen olguyu sunacağız. Bilinen herhangi bir hastalığı olmayan, 2 paket/gün sigara kullanan 55 yaşında erkek hasta son 2 ayda ortaya çıkan tipik anjina şikayeti ile kliniğimize başvurdu. Anjinal yakınmalar nedeniyle efor kapasitesi kısıtlanan hastada son 10 günde her öğünden sonra mutlaka nitrogliserin kullanma ihtiyacı doğmuştur. Fizik muayenede aortik odakta 2/6 diastolik üfürüm dışında önemli bulgu saptanmadı. Elektrokardiyogramda normal sinus ritmi, sol ventrikul hipertrofisi ve yaygın ST segment depresyonu izlendi. Transtorakal ekokardiyografide inferior ve posterior duvarda ağır hipokinezi, sol ventrikül ejeksiyon fraksiyonu %50, aortik küspislerde önemli patolji olmadan orta aort yetersizliği tespit edildi. Koroner anjiyografide sol ana koroner arterin selektif kanülasyonu başarısız oldu ve nonselektif görüntülerde sol ana koroner arter ostiumunda %99, sağ koroner arter ostiumunda %90 darlık izlendi (Şekil 1). Akut aortik sendromların (AAS) ekartasyonu amacıyla çekilen kontrastlı toraks tomografide aort kökü ve asendan aortada en kalın yerinde 5 mm sirkümferensiyel kalınlaşma izlendi, diseksiyon ve ya intramural hematom ekarte edildi. Koroner BT anjiyografide koroner ostial stenoz varlığı

onaylandı, koroner arterlerin diger kısımlarında anlamlı darlık izlenmedi (Şekil 2). Aortit düşünülen hastada RPR (rapid plasma reagin) ve TPA testleriyle sifilis tanısı doğrulandı. Başarılı koroner arter baypas ameliyatı sonrasında hastanın kardiyak şikayetleri geriledi. Koroner arterlerin diğer kısımlarında hemodinamik anlamlı darlık olmadan yalnız koroner arter ostiumlarının bir veya her ikisinde önemli darlık izlenen, eşlik eden aort yetersizliği olan hastalar dikkatli inceleme gerektirmektedir. AAS ekarte edilen hastalar Takayasu arteriti ve sifilitik aortit (SA) gibi asendan aortayı tutan hastalıklar yönünden araştırılmalıdır. Sifilis hastalarının %26-da aort kökü ve koroner arter tutulumu bildirilmiştir. Bu hastalarda koroner lezyonlar genellikle ostial lokalizasyonlu olup, darlıklar %90 üzerindedir. Kardiyovasküler tutulum hastamızda olduğu gibi sifilisin ilk belirtisi ola bilir ve önceden bilinen sifilis tanısının veya diğer sifilis bulgularının olmaması SA tanısından uzaklaştırmamalıdır. SA-lı hastalarda koroner cerrahide safen greftlerinde de ostial darlık riski nedeniyle internal mammarian arterin kullanımı önerilmektedir. Günümüzde ender rastlansa da bu tip hastaların ayırıcı tanıda SA mutlaka düşünülmelidir.



Şekil 1. (A) Sol ana koroner ostial darlığının nonselektif görüntülünmesi (ok) **(B)** sağ koroner ostial darlığı (ok).



Şekil 2. Koroner BT tomografide asendan aortada kalınlaşma (siyah ok) ve koroner ostial darlıklar (beyaz ok) (**(A)** - sol ana koroner arter, **(B)**-sağ koroner arter).

OS-32

Diagnosis of endarteritis with PET-CT in a patients with aort coarctation

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Objectives: Infective endocarditis (IE) is a infectious disease that affects endarterit of large intrathoracic vessel and structures exposed directly to the blood flow including intracardiac foreign body matherials. In recent studies, it was shown that nuclear imaging (PET-CT) helps the definite IE diagnosis in possible IE patients according to Duke criterias. We are presenting a case in whom diagnosis of endarteritis was made with positron emission tomography–computed tomography (PET-CT) in a patients with aort coarctation.

Case Report: A 20 years old women was admitted to our cardiology departmen with complaints of fever and palpitation. On her background, she has iron defficiency anemia and aort coarctation. On physicial examination, there was pretibial oedema and erythema on both legs. On laboratories, iron defficiency anemia was diagnosed and two unites erythrocyte suspansion are given to the patient. On transthoracic echocardiography (TTE), there was bicuspid aortic valve, enlargment of coronary sinus and aort coarctation. TEE revealed bicuspid aortic valve but there was no vegetation on it. Metisilin sensitive staphylacoccus aureus (MSSA) is produced on blood cultures. We decided to perform 18 FDG PET-CT to make definite diagnose of IE according to 2015 ESC/ERS IE guidelines. It revealed increased intensive glucose uptake on dilated aortic segment adjacent to distal coarctation zone (Figure 1). There was no increased glucose uptake nor bicuspid aortic valve neither the other cardiac valves. The final diagnosis was endartertis of descending aorta and antimicrobial therapy was started according to susceptibility testing results.

Conclusion: IE is a deadly disease. Several reports have shown promising results for radiolabelled white blood cell (WBC) SPECT/CT and 18F-FDG PET/CT imaging in IE. The main added value of using these techniques is the reduction in the rate of misdiagnosed IE, classified in the 'Possible IE' category using the Duke criteria, and the detection of peripheral embolic and metastatic infectious events. This is a original case that PET-CT revealed endarteritis of descending aorta in a patient without prosthetic cardiac matherial.

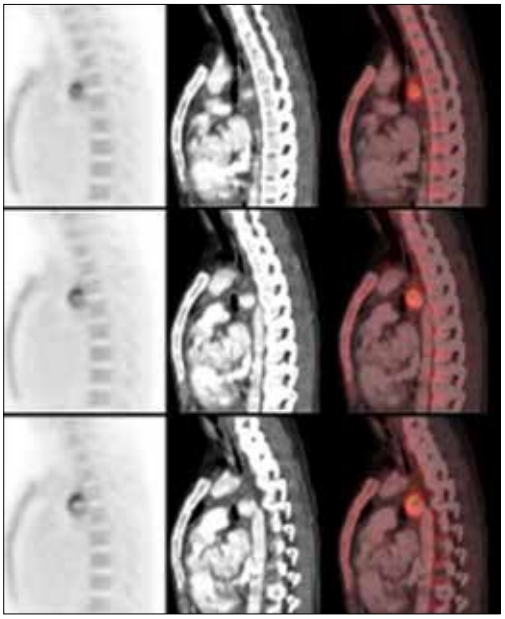

Figure 1. 18 FDG PET-CT revealed increased intensive glucose uptake on dilated aortic segment adjacent to distal coarctation zone.

OS-33

Interventricular septal lipoma presenting with atrial fibrillation

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A 27 years old female patient with no known cardiovascular disease was admitted with palpitation. Electrocardiogram showed atrial fibrillation with high ventricular rate. On physical examination, there was not a heart murmur. Five hours later, it was spontaneously returned to sinus rhythm. Laboratory tests were unremarkable. Transthoracic echocardiography revealed that a hyperechoic mass was originating from in the interventricular septum extending through the left ventricle (Figure 1). Cardiac MRI (magnetic resonance imaging) revealed solitary, smooth surface and unilobular mass arising from the endocardial surface of left ventricle. The signal intensity of the mass was consistent with adipose tissue (Figure 2). Resection of the mass was not considered because of its' asymptomatic course and small sized. Lipomas are the benign tumors account for 8.4% of the primary tumors of the heart and pericardium and MRI findings are characteristics for the diagnosis. They are usually found in the right atrium and left ventricle, interventricular septum is unusual location that only 2% of the cases of lipomas were found in interventricular septum.

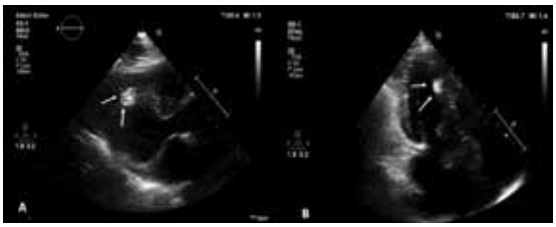

Figure 1. Parasternal short axis view **(A)** and apical four window view **(B)** of transthoracic echocardiography showing a hyperechoic mass was originating from in the interventricular septum extending through the left ventricle.

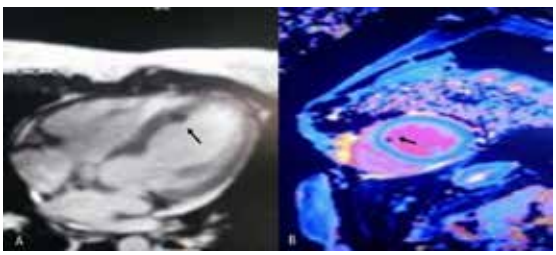

Figure 2. Long axis **(A)** and fat suppressed cardiac MRI images **(B)** are showing small lipoma (14x7 mm in diameter) originating from the interventricular septum. Video. Three dimensional echocardiography showing septal lipoma.

OS-34

A Non-Hodgkin lymphoma case presenting with constrictive pericarditis

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Extranodal involvement in non-Hodgkin lymphoma (NHL) occurs up to 33% of patients at some stages of the disease and it's most frequent in diffuse B-cell lymphoma. Cardiac involvement is clinically detected very rarely and it's usually a late manifestation of the disseminated disease. We reported a case with initial presentation as constrictive pericarditis clinic and finally having the diagnosis of disseminated NHL with secondary involvement of heart and pericardium. A 56-year old female presented with worsening dyspnea and fatigue for the last one month. She had a history of pericarditis 3 years ago, with massive effusion and she had pericardiocentesis, which revealed normal cytology and the etiology was thought to be viral. Her last control 6 months ago was all normal without any pericardial effusion. Her symptoms had started for one month and her dyspnea was NYHA class 3 on presentation to our clinic. Laboratory tests revealed only elevated lactate dehydrogenase level, 737 U/L (120-246 U/L), Cardiomegaly was evident in chest X-ray and echocardiography revealed echogenic non homogenous mass all around heart muscle in the pericardial cavity, with accompanying fibrinous minimal pericardial effusion. All the parameters were consistant with constrictive pericarditis. For further clarification of the pericardiac mass, thorax CT and cardiac MRI were done and they revealed lobulated conglomerate mass, probably lymph nodes, around heart, invading into right atrium and pericardium (Figure 1). Because the patient showed rapid progression of the clinical picture she was admitted by cardiovascular surgery team for the aim of decompression of the heart. After thoracotomy, firm, rigid,and lobulated white mass was seen in front of the right ventricle (Figure 2). It had firmly invaded the myocardium so complete resection was impossible, but decompression was achieved. Patient recovered postoperatively and pathology revealed diffuse large B-cell lymphoma (DLBCL). Further diagnostic work up revealed disseminated disease, so the diagnosis was secondary cardiac involvement. Chemotherapy regimen was started at the hematology clinic and she is still being followed up by cardiology with minimal pericardiac mass, without any hemodynamic effect. As far as we searched, our case is the first in the literature having the diagnosis of secondary cardiac NHL and presenting with constrictive sypmtoms. Patient had diffuse lymph node involvement all over her body at the time of diagnosis, but cardiac involvement was the dominant and aggresive site since it was the presenting symptomatic region. To conclude, NHL can present with cardiac symptoms, such as fatigue and dyspnea, and it can be the initial presenting symptom in an extranodal disease. Echocardiographic examination is the mainstay of the diagnosis for both effusive conditions and tumor involvement. Constrictive pericarditis signs at echocardiography can be the initial presentation of a NHL case, although very rare.

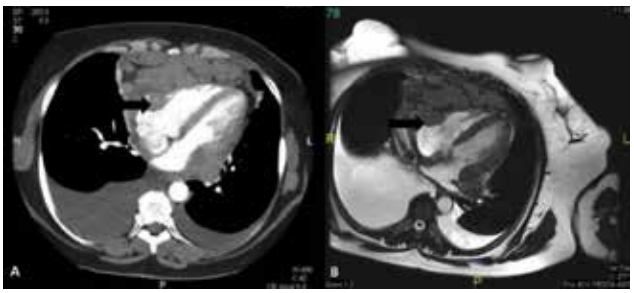

Figure 1. Thorax CT at left and cardiac MRI at the right side. Both revealed lobulated conglomerate mass around heart, invading into pericardium and right atrium (shown with black arrows). Bilateral pleural effusion was also seen.

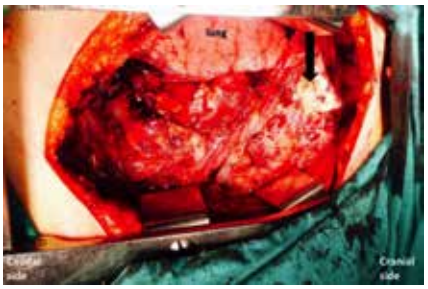

Figure 2. Post-thoracotomy view. Firm, rigid, and lobulated white mass was seen in front of the right ventricle (shown with black arrow).

OS-35

A giant left ventricular pseudoaneurysm presenting with transient ischemic attack 7 years after acute myocardial infarction

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Left ventricular pseudoaenurysm (LVPA) is a rare and dangerous clinical status which commonly occurs after transmural infarction. LVPA develops when myocardial rupture is contained by pericardial adhesions and thrombus formation. Untreated pseoudoaneurysms have a very high risk of rupture. Thus surgery is the preferred therapeutic option [1]. We present a patient with a giant inferior LVPA after myocardial infarction who survived for seven years and complicated only with transient ischemic attack. A 75-year-old male patient was admitted to emergency department with a complaint of temporary verbal aphasia. Seven years earlier, the patient had suffered an inferior wall myocardial infarction and treated with urgent left anterior descending artery (LAD) - saphenous vein graft (SVG) and right coronary artery (RCA) - SVG bypass surgery. In postoperative course, left ventricular systolic impairments were observed with a reduction of ejection fraction to 48% and hypokinesia of the inferior wall. He had not a cardiac symptom or control coronary angiography (CAG) in the meanwhile. His symptoms were completely resolved before the admission and neurological examination was normal. A cranial multi-detected computed tomography (MDCT) was performed to explain the neurological symptoms. MDCT scan revealed a location of ischemic stroke at chronic stage in left occipital lobe (Figure 1). His electrocardiogram showed ST segment elevation and deep Q waves in leads D3 and aVF which were consistent with an old inferior wall myocardial infarction (Figure 2). Echocardiographic examination revealed the presence of a large (max. dimensions: 67x75 mm, entrance size: 38 mm) thrombosed LVPA of the inferior wall (Figure 3, Video 1, 2, 3). Remaining walls had normal contractility with a global EF of 38%. CAG demonstrated the normal functioning of the LAD-SVG and left circumflex artery and total occlusion of the RCA. RCA-SVG stump was not visualized. Contrast-enhanced MDCT of the heart confirmed the LVPA arising the inferior wall (max. dimensions: 65x71x85 mm) and total occlusion of the RCA-SVG from the origin. In a consultation with cardiovascular surgeon team, an urgent surgery decision was made. However, the patient and his relatives denied the therapy. Therefore, anticoagulation with warfarin and low molecular weight heparin were administered. After reaching the target level, the patient was discharged and called for a control after a month. Control echocardiographic examination revealed the persistence of the thrombus inside the LVPA. We present the latest pseudoaneurysm in the literature secondary to AMI. Furthermore; we present a unique case report with illustrative and demonstrative images via echocardiography and MDCT.

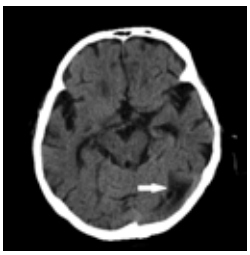

Figure 1. Electrocardiogram shows ST segment elevation and deep Q waves in lead D3 and aVF which is consistent with left ventricular aneurysm.

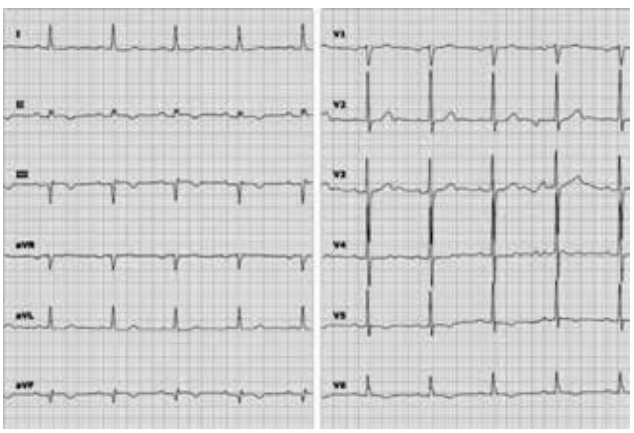

Figure 2. (A) TTE, Apical modified 4 chamber view shows a near-normal examination. **(B, C)** Parasternal short axis view from TTE shows a big (67x75 mm) and partially thrombosed LVPA. There is a narrow gap (38 mm) between LV and LVPA.

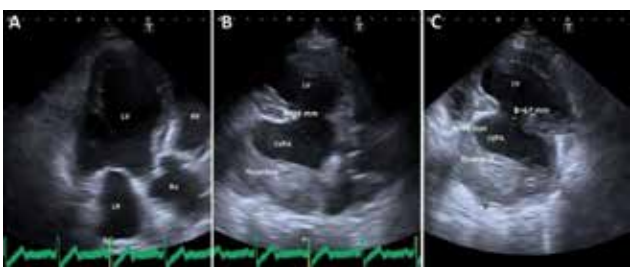

Figure 3. Horizontal, sagittal and coronal cardiac CT scans show thrombosed LVPA. LVPA has a thin outer layer.

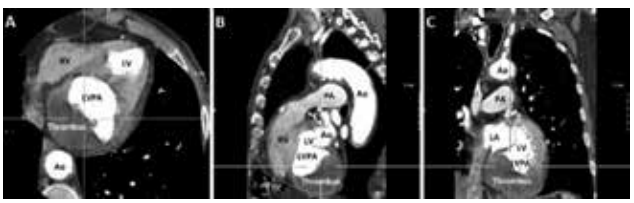

Figure 4. Coronal and horizontal cardiac CT scans show totally occluded RCA-SVG from the anastomosis point.

OS-36

Postpartum spontan koroner arter diseksiyonu

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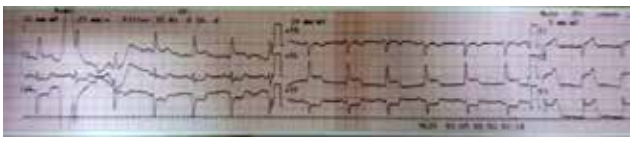
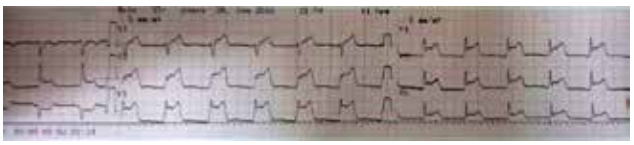
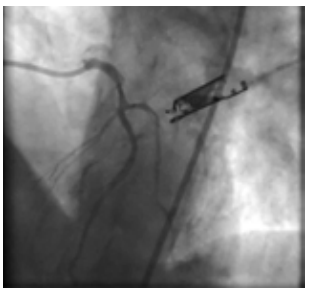
Spontan koroner arter diseksiyonu;, iskemik kalp hastalıkları arasında nadir rastlanan bir durumdur. Akut koroner sendrom, kardiyojenik şok ve ani kardiyak ölüm şeklinde karşımıza çıkabilir. Olguların %75-80'i kadındır ve bunlardan 1/3'ü son trimester gebeler ve doğum sonrası ilk 3 ay içindeki kadın hastalardır.Teşhisin erken konulması ve tedaviye erken başlama artmış sağ kalımla ilişkilidir. Koroner anjiografi tanı için sıklıkla kullanılan yöntemdir. Biz de ateroskleroz için risk faktörü taşımayan 36 yaşındaki postpartum SKAD vakasını paylaştık. Otuz altı yaşında kadın hasta ani başlayan göğüs ağrısı, nefes darlığı şikayeti ile başvurdu. Bilinen kardiyovasküler hastalık öyküsü yoktu. 1 hafta önce sezaryan/sectio ile doğum yapmıştı ve gebelik süresince preeklampsi nedeniyle tedavi görmüştü. Fizik muayenesinde; genel durum kötü; bilinç bulanık, kan basıncı 120/80 mmHg; solunum sesleri kaba idi, bilateral akciğer bazallerinde raller mevcuttu. Kalp sesleri ritmik ve taşikardikti. Nabız 110/dakika idi. Elektrokardiyografide anterior derivasyonlarda 3-4 mm ST elevasyonu vardı. Ekokardiyografisinde; apikal septum, apeks, anterior mid ve apikali hipokinetikti. Sol venrikül ejeksiyon fraksiyonu %30'du ve azalmıştı. Hasta katater laboratuarına alındı. Sağ femoral arterden girilerek selektif sağ-sol koroner anjiyografi yapıldı. Anjiyografide LAD proksimalinde diseke lezyon ve medialinde plaklar vardı. Sağ koroner arter plaklı idi. LAD proksimalindeki diseke lezyona 3.0x9 mm stent yerleştirildi ve tam açıklık TIMI 3 akım sağlandı. Hasta koroner yoğun bakım ünitesine alındı. Tedavi ve takip sonrası şikayetleri gerileyen hasta medikal tedavisi düzenlenerek taburcu edildi.



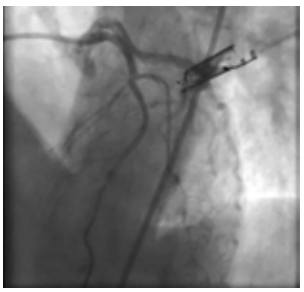
Şekil 1. Hastanın acil servisteki EKG'si.



Şekil 2. Hastanın acil servisteki EKG'si (devam).



Şekil 3. Koroner anjiyografi sol sistem görüntüleme lad proksimal disseksiyonu.



Şekil 4. PCİ sonrası lad proksimal lezyonunun görüntüsü.

OS-37

Sağ koroner arter kronik total oklüzyonuna sirkumfleks arterden uzanan epikardiyal bağlantılardan retrograd yaklaşımla başarılı perkütan girişim

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Beş yıl önce üç damar koroner baypas operasyonu geçiren 56 yaşındaki erkek hasta 4 aydır devam eden eforla artan göğüs ağrısı şikayetiyle polikliniğimize başvurmuştur. Miyokardiyal perfüzyon sintigrafide sol ventriküler EF %40 ve infero-septal alanda reversibl perfüzyon defekti saptanması üzerine uygulanan koroner anjiyografi de sol ön desendan (lad) arter 1. diyagonal sonrası %100 tıkalı, sirkumfleks (cx) arter proksimali %80 darlık, 2. obtus marjinalis sonrası cx tam tıkalı izlendi. Sağ koroner arter (rca) proksimali sonrası tam tıkalı olup, köprü kollateral ile antegrad dolum ve sol sistemden retrograd dolum izlendi (Video 1). Sol internal mammaryan arter grefti hariç baypas greftleri tam tıkalı izlendi. Rca lezyonu kronik total oklüzyon (kto) olarak değerlendirildi. Perkütan koroner girişim kararı verildi. Sağ femoral artere 7 F kılıf ile girildi ve 10000 IU iv heparin bolus uygulandı. Rca ostiumuna 7 F 4.0 sağ guiding kateterle selektif oturuldu. Proksimalde penetrasyon icin yeterli güdük olmadığından ve oklüzyon bölgesinin her iki tarafında yandallar çıktığı için anterograd yoldan corsair mikrokateter desteğiyle miracle 6 ve conquest 9 tellerle gerçek lumene geçilemedi. Sol femoral artere 7 F kılıf ile girildi. Sol ana koroner ostiumuna EBU 3.5 kateterle selektif olarak oturuldu. Cx' den rca'ya uzanan epikardiyal bağlantılardan Sion tel ile retrograd yaklasımla geçildi. Corsair mikrokateter oklüzyon bölgesine kadar ilerletildi. Miracle 6 ve Conquest 9 teller ile geçilemedi. Conquest 12 tel ile oklüzyon geçildi. Tel asendan aortada periferik snare ile yakalandı ve sağ guiding kateter içine alındı ve 2.5x20 mm standart balon 16 atm'de şişirilerek tel tuzaklandı. Corsair mikrokateter ile lezyondan geçilerek sağ guiding kateter içine kadar ilerletildi. RG-3 tel ile eksternalize edildi. Ardından 2.0x20 mm standart balon ile 10 atm'de predilatasyon uygulandı, ve 2.25x32, 2.5x38 ve 2.75x16 mm everolimus salınımlı promus stentler 16 atm'de implante edildi. Başarılı açılım sağlandı (Video 2). Takiplerinde göğüs ağrısı ve ekg değişikliği olmayan hasta antiplatelet tedavisi düzenlenip taburcu edildi. Koroner baypas sonrası nativ damarların kto tedavisi sıklıkla uygulanmaktadır. Kto'da antegrad yaklaşımın başarı oranı sınırlı olduğundan, iyi kollateral kanallar varlığında retrograd yaklaşım sıklıkla gerekmektedir.

OS-38

Akut koroner sendrom nedeni trombotik sol ana koroner arter darlığının düşük doz, yavaş infüzyon t-PA ile başarılı tedavisi

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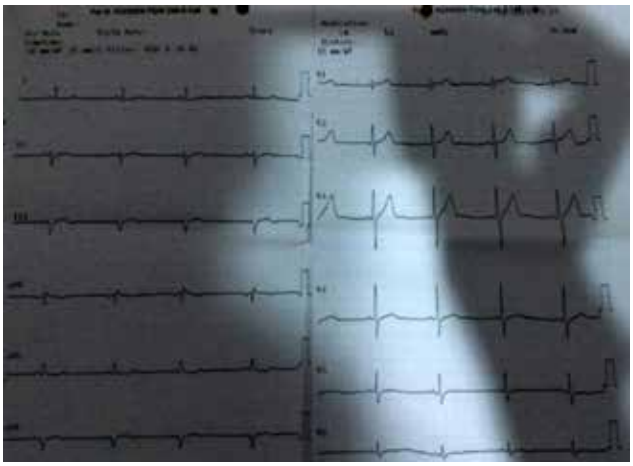
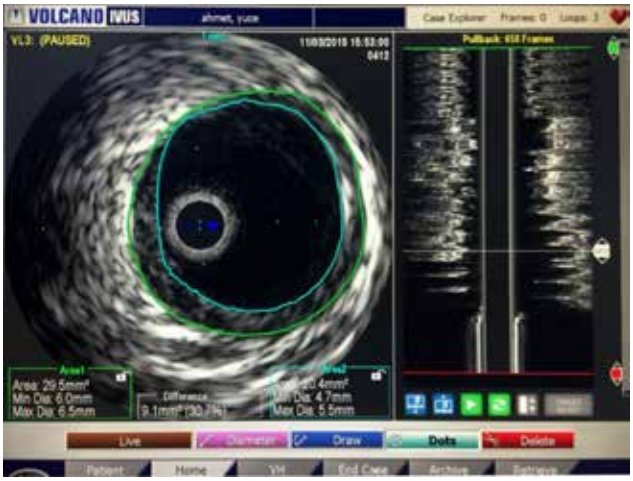
Kırk üç yaşında erkek hasta 2 saattir aralıklı varolan tipik göğüs ağrısı, EKG'sinde D2, D3, aVf derivasyonlarında bifazik T dalgası (Şekil 1), troponin I değeri 4.24 ng/ml olup, ST yükselmesi olmayan miyokard enfarktüsüyle kliniğimize refere edildi. Öyküsünde sigara ve aile öyküsü dışında ek risk faktörü, madde kullanımı olmayan hastanın koroner anjiyografisinde sol ana koroner arterde semi-mobil koroner akıma izin veren trombüs izlendi. (Video 1). Hemodinamisi stabil olan, ağrısı tekrarlamayan hastaya antikoagülan ve antiagregan tedaviye ilave gp 2b-3a inhibitörü tirofiban infüzyonu 15 cc/h, 80 saat verildi. Kontrol koroner anjiyografide sol ana koronerde trombüsün aynen durduğu görüldü (Video 2). Trombüs aspirasyonu, direk stentleme, bypass operasyonu, trombolitik tedavi uygulanması, risk-yarar oranları ve en uygun reperfüzyon tedavi stratejisi tartışıldı. Literatürde tam doz trombolitik tedavi uygulanan hastalar da görüldü. Hastaya 25 mg tissue plasminogen activator (t-PA) 6 saatte verildi. 24 saat sonra tekrarlanan koroner anjiyografisinde trombüsün kaybolduğu ciddi lezyon olmadığı görüldü (Video 3). Asetilsalisilik asit, tikagrelor, atorvastatin, nebivolol, perindopril verilerek taburcu edilen hastanın 1 ay sonra yapılan kontrol koroner anjiyografisinde ve intravasküler ultrasonografisinde ciddi darlık oluşturmayan plak izlendi (Şekil 2, Video 4). Hastanın protrombotik etiyoloji açısından detaylı değerlendirilmesi sonrası protein C ve S eksikliği dışında ek patoloji saptanmadı. Protez kapak trombüsleri ve koroner embolilerde düşük doz, yavaş infüzyon trombolitik tedavinin güvenli ve efektif olduğu bildirilen vakalar olmasına rağmen, izole trombotik lezyonlarda, bizim vakamızdaki gibi sol ana koroner lezyonunda da düşük doz, yavaş infüzyon t-PA tedavisinin güvenli ve efektif olabileceği akılda tutulmalıdır. Hasta 8 aydır komplikasyonsuz medikal olarak izlenmektedir.



Şekil 1.



Şekil 2.

OS-39

Dekstrokardi olgusunda sol ana koroner diagonal bifurkasyon olgusu

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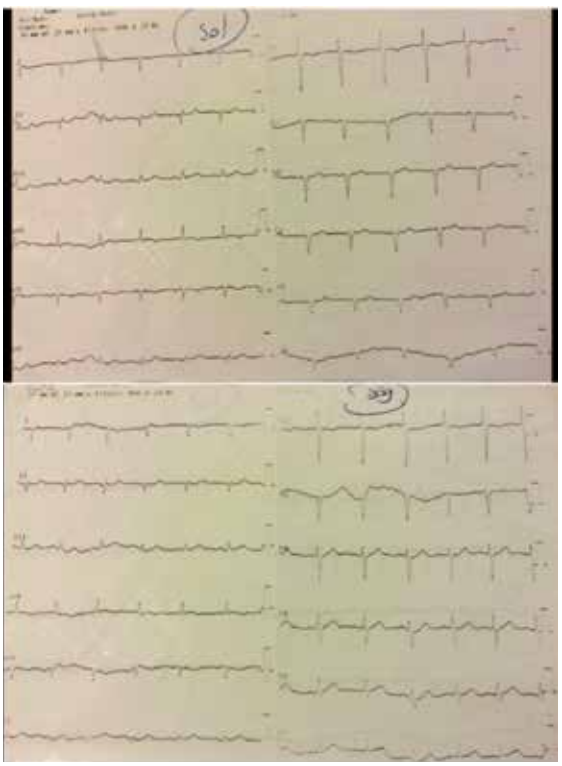
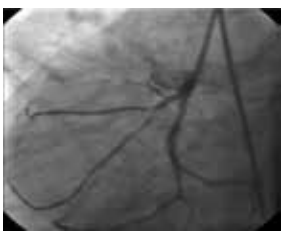
Giriş: Sitüs inversus ile beraber dekstrokardi nadir olan bir konjenital malformasyondur. Bu hastalarda koroner arter hastalığı sıklığı normal popülasyon ile aynı olduğu düşünülmektedir. Literatür incelendiğinde bu hastalarda az sayıda perkütan koroner girişim bildirilmiştir. Koroner anjiografi ve perkütan girişim tekniği zor olmamakla beraber ayna imajı tekniği, farklı kateter kullanımı ve alışılmamış kateter manipülasyonları gerektirebilir. Bilgimiz dahilinde literatürde tek olan dekstokardi olgusunda bifukasyon koroner stent vakasını tartışmayı amaçladık.

Olgu Sunumu: Kırk sekiz yaşında bilinen dekstrokardi ve sitüs inversusu olan hasta, sabaha karşı uykudan uyandıran, bir saat süren ve sağ kola yayılımı olan göğüs ağrısı olması üzerine acil servise başvurdu. Brakiyal tansiyon arteryel 135/70, nabız 73 atım/dk, ateş 36.6 C idi.Fizik muayanede kalp tepe atımı sağ 5. interkostal aralık midklavikular hatta palpe edildi, S3 ve üfürüm duyulmadı. Ekg d1 ve aVL p negatif, aVR'de pozitif R dalgası, V2-6 arası QS formasyonu izleniyordu. Sağ ekg'de r progresyonu doğal olup iskemik ST-T değişikliği izlenmedi (Şekil 1). Hastanın takibinde kontrol troponin-t 0.045 ng/dl'den 0.208 ng/dl'ye yükselmesi üzerine hasta akut koroner sendrom ön tanısı ile koroner yoğun bakım ünitesine interne edildi. Hastanın yapılan transtorasik ekokardiyografi incelemesinde sol ventrikül duvar hareket bozukluğu,major kapak patolojisi ve ek konjenital anomali izlenmedi. Hastaya ikili antiaggregan(asetilsalisilik asit+klopidogrel) yüklemesi yapılarak kateter laboratuvarına alındı. Hastada sağ femoral arter girişim yolu kullanıldı. Sağ Judkins(JR) 4 diagnostik kateter kullanılarak sol anterior oblik (LAO) 45 açı ile sol koroner sinüsten çıkan sağ koroner arter görüntülendi. Ardından aynı kateter counterclokwise rotasyonu ile sağ koroner sinüsten çıkan sol ana koroner ostiumuna oturuldu. LAO 30, CRANİAL 30 açı'da medina 0;1;1 LAD-D1 lezyonu izlendi. Hastaya V stent tekniği ile bifurkasyon kararı alındı. sheath 7f ile değiştirili Lad lezyona 2.25x24 mm, D1 osteal lezyona eş zamanlı 2.5x18 MM Resolute Des implante edildi. Kissing balon yapılarak işleme son verildi (Şekil 3).

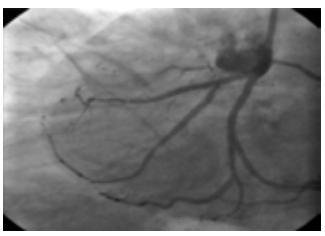
Tartışma: Sitüs inversus dektrokardi hastalarında angina retrosternal olup sağ kola yayılım şeklinde olabilir. Bu atipik durumun mekanizması açık olmamakla beraber nöral aksın gelişiminden kaynaklanabilir. Anjiografi ve girişim yapılırken ayna imajı tekniği ve judkins kateter kullanılmıştır. Örneğin RAO kaudal poz dekstrokardisi olan hastada "spider" görüntüsü vermektedir. Akut koroner sendrom ile gelen hastada dektrokardi farklı aortik ark ve koroner çıkış yapısı, kateter manüplasyonu ve görüntüleme açılarının farklılığı nedeni ile kapı balon süresi ve kullanılan opak miktarı artmaktadır. Uygun tekniklerin bilinirliğinin artması işlem süresini ve kompliasyonlarını azaltabilir.



Şekil 1. Hastanın çekilen sol ve sağ perikordiyal EKG'si.



Şekil 2. Lad-diagonal lezyonu.



Şekil 3. Stent sonrası AP kranial görüntü.

Girişimsel kardiyoloji / Koroner

OS-40

Iatrogenic aortic dissection during percutaneous coronary intervention

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Background: Aort dissection during coroary procedures is a very rare complication occuring more often in interventions than diagnostic procedures. Iatrogenic aortic dissections during coronary procedures can be treated either by surgically or percutaneously. In this report we are presenting a case of iatrogenic aortic dissection completely sealed by coronary stent implantation.

Case Report: 68 years old male patient with a history of hypertension presented with CCS class 3-4 angina pectoris. Coronary angiography revealed total occlusion at proximal RCA. 6F jr4 guiding catheter was engaged to coronary ostium and floppy guidewire was advanced. Distal flow was maintained after wire crossing. At first we aimed to implant stent to distal RCA, however control angiogram iatrogenic RCA ostial dissection spreading to ascending aorta (Figure 1, Video 1). We first deployed DES to distal RCA and then implanted 3x20mm DES to proximal RCA to seal the origin of the dissection. After stent deployment, control angiograms revealed no more contrast filling to the false lumen. Immediate CT angiogram demonstrated aortic dissection flap terminating proximal to the arcus aorta (Figure 2). Since the patient was asymptomatic and assuming that we successfully sealed the origin of the dissection, medical follow-up was decided. 3 days later and 1 month later control CT angiograms revealed complete resolution of the dissection (Figure 3). Iatrogenic aort dissection is a rare but a severe complication of coronary interventions. Sealing the origin of the dissection by stent implantation van solve the problem, but if the dissection spreads to arcus aorta, and if the patient is unstable emergent surgery is the choice of treatment. Operators must be very careful in catheter or guidewire manipulation especially if there's proximal lesions. The patient was asymptomatic throughout the procedure and during the hospitalisation and discharged without complication.

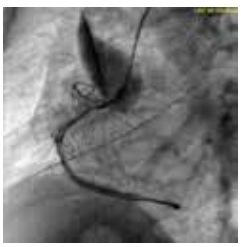

Figure 1.

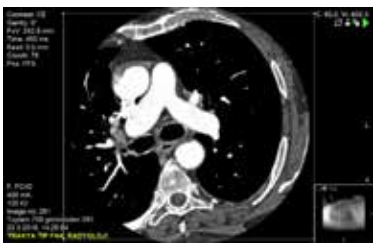

Figure 2.

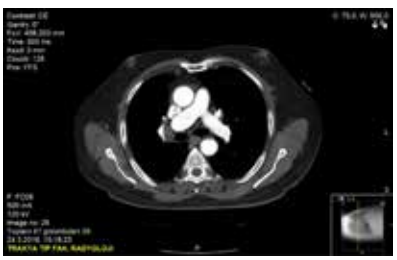

Figure 3.

OS-41

Akut anteriyor miyokard infarktüsüyle başvuran 34 yaşında 36 haftalık gebe hastanın başarılı basamaklı tedavisi

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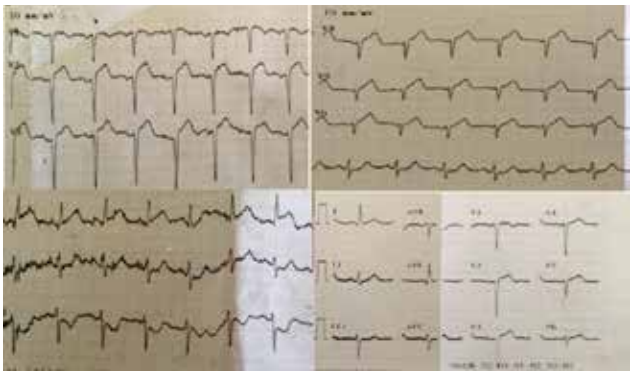
Giriş: Gebelikte miyokard infarktüsüyle başvuran hastaların çoğunda altta spontan koroner arter diseksiyonu yatmaktadır. Tedavi yaklaşımı olarak konservatif izlemden, perkutan koroner girişim ve ivedi bypass operasyonuna kadar giden vakalar literatürde bulunmaktadır.

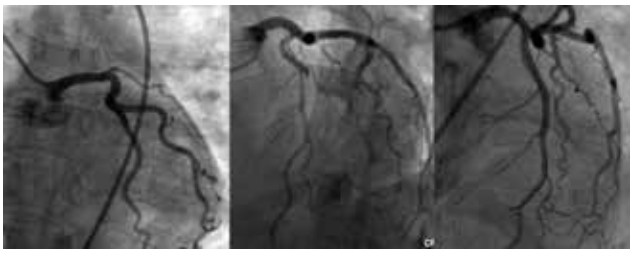
Olgu Sunumu: 34 yaşında bayan hasta tipik vasıflı göğüs ağrısının 2. saatinde hastanemize başvurdu. 36 haftalık gebe olup, koroner arter hastalığı risk faktörü yoktu. EKG'sinde D1, aVL, V1-V6 derivasyonlarında ST elevasyonu olup (Şekil 1), akut anteriyor miyokard infaktüsü tanısıyla hastaya asetilsalisilik asit 300 mg, klopidogrel 600 mg, enoksaparin 0.6 ml intravenöz yapılarak, primer perkutan koroner girişime alındı. Koroner anjiyografisinde LAD görüntülenemedi. (Şekil 2a, Video 1) LAD ostealden güdüğü görülemeyecek şekilde total tıkalı olabileceği düşünüldü. Göğüs ağrısı ve ST segment elevasyonu devam eden, ekokardiyografisinde anteriyor duvar, septum ve apeks hipokinetik (Video 2) izlenen hastaya ağrısının 3. saatinde 100 mg alteplaz (tPA) 90 dk'da intravenöz uygulandı. Hastanın göğüs ağrısı geçti, ST segment elevasyonu düzeldi. 24 saat sonra yapılan kontrol koroner anjiyografisinde LAD ostealden sirküler diseksiyon izlendi. (Şekil 2b, Video 3) Hastada perkutan koroner girişim, diseksiyonun ilerlemesi ve komplikasyon açısından yüksek riskli değerlendirilerek, öncelikli konservatif yaklaşım, doğumun yaptırılması, sonrasında perkutan koroner girişim planlandı. Hasta kadın doğuma konsülte edilerek 3 gün sonra sezaryan ile doğum yaptırıldı. Peroperatif yakın hemodinami takibi yapıldı, göğüs ağrısı olmadı. Hasta ve bebek açısından komplikasyonsuz doğum gerçekleştirildi. Hasta doğumdan 1 hafta sonra anjiyografi laboratuvarına alınarak LAD'ye ostealden 3.0x38 mm ilaç salınımlı stent implante edildi. Nonkompliyan 3.0x15 mm balon ile postdilatasyon yapıldı. Komplikasyon olmadı. (Şekil 2c, Video 4) Hasta asetilsalisilik asit, klopidogrel, metoprolol tedavisi altında taburcu edildi.

Tartışma: Gebelikte diseke koroner lezyonlara erken müdahaleyle, diseksiyonun ilerlemesi ve ivedi bypass operasyonuyla sonuçlanan olgular literatürde mevcuttur. Diseke alanda intramural

hematomu arttırma riski nedeniyle trombolitik tedavi genellikle önerilmemektedir. Bizim vakamız, başlangıçta görüntülenemeyen ancak total düşünülen lezyona trombolitik tedaviyle başarılı reperfüzyon sağladıktan sonra, ostealden itibaren diseke ve zor LAD lezyonuna öncelikle medikal izlem, sonrasında perkutan koroner girişimin başarılı ve basamaklı uygulanmasıyla sıradışıdır. Gebelikte ST segment elevasyonlu miyokard infaktüsüyle başvuran, anjiyografide lezyon görüntülenemeyen semptomatik hastalarda trombolitik tedavinin uygulanabilirliği açısından da değerlendirilmelidir.



Şekil 1.



Şekil 2.

OS-42

Akut miyokarditle seyreden hipereozinofilik sendromlu hasta

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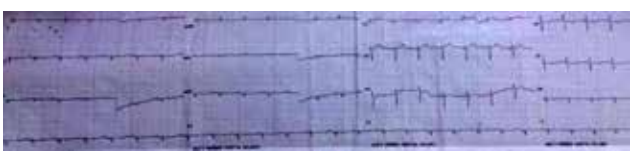
Hipereozinofilik sendrom (HES); sekonder nedenler dışlandıktan sonra persistan hipereozinofili (>1500/mm^3) ile seyreden, çoklu organ tutulumuna karşılık lösemi kriterlerinin yokluğu ile karakterize lökoproliferatif bir hastalıktır. Erkeklerde daha sık görülen HES un tüm formlarında kardiyak tutulum sık görülmekte ve mortalitenin esas sebebi olduğu bilinmektedir. HES in kardiyak tutulumu nekrotik, trombotik ve fibrotik evreleri içerir. Kalpte restriktif ve dilate kardiyomiyopati, kapak yetersizliği, septal hipertrofi, konstriktif perikardit ve nadir olarak nekrotizan miyokardit yapabilir. Tedavi edilmemesi halinde mortalitesi oldukça yüksektir. Hastanemizde eozinofilik akciğer hastalığı araştırılırken akut nekrozitan miyokardit gelişen ve sonrasında hipereozinofilik sendrom tanısı alan bir hasta sunulmuştur. 5 ay öncesinde KOAH tanısı alan 63 yaşında erkek hasta inhaler bronkodilatatör tedaviye rağmen öksürük ve nefes darlığının devam etmesi üzerine göğüs hastalıkları servisine yatırılmış. Başvuru sırasında CBC de 17200/mm^3 lökositoz saptanıp, %65 eozinofili (11200/mm^3) mevcutmuş. Eozinofilik akciğer hastalığı düşünülerek araştırılan hastanın yatışında yapılan EKO normal sınırlardaymış. Hipereozinofili yapacak sekonder nedenler araştırılırken, yatışının 9. gününde ani başlangıçlı bir saat süren retrosternal bölgede baskı tarzında göğüs ağrısı gelişmiş. Tarafımıza danışılan hastanın başvuru EKG sinde, sinüs ritmi ve V4-6 T dalga negatifliği varken; göğüs ağrısı sırasında çekilen EKG de sağ dal bloğu paterninde geniş QRS li sinüs ritmi saptandı (Şekil 1, 2). Hasta akut miyokard enfarktüsü düşünülerek koroner yoğun bakım ünitesine alındı. Hastaya aspirin, klopidogrel, heparin tedavisi başlandı. Yoğun bakıma kabulünde göğüs ağrısı azalan hasta konservatif olarak takip edildi. Kabulünde kan basıncı 120/70 mmHg, nabız:115/dk, sO2 %90 olup kardiyak oskültasyonda S3 ü mevcuttu. Akciğer muayenesinde ise bilateral orta zonlara kadar ince ralleri mevcuttu. Konjestif kalp yetmezliği tanısı ile hastaya eş zamanlı furosemid tedavisi de başlandı. Yapılan EKO da sol ventrikül global hipokinetik ve EF' nin %23 olduğu görüldü. Kan biyokimyasında Troponin T 2942 ng/L (0-14), proBNP ise 9165 pg/ml (0-110) saptandı. Kompanzasyon sonrası KAG da normal koroner arterler saptandı. Sol ventrikülografide ise ventrikül sistolik fonksiyonunun belirgin bozulmuş olduğu izlendi (Video 1–3). Mevcut tablo göğüs hastalıklarıyla beraber değerlendirildiğinde hastanın HES e bağlı akut nekrozitan miyokardit geçirdiği düşünüldü. Hastaya 80 mg/gün metilprednizolon tedavisi başlandı ve takibinde kalp yetmezliği semptomlarının düzeldiği ve proBNP nin belirgin azaldığı görüldü. Tedavinin 6. gününde CBC de eozinofilinin belirgin gerilediği (%9-1100/mm^3) izlendi. 10. günde yapılan EKO da sistolik fonksiyonlarda düzelme gözlenen hastanın EF si %35 saptandı. Hasta betabloker, ACE inhibitörü, aldesteron antagonisti ve steroid tedavisiyle 1 ay sonra kontrole gelmek üzere taburcu edildi.



Şekil 1. Hastanın kardiyak tutulum öncesi EKG'si.

Şekil 2. Hastanın göğüs ağrısı sırasındaki EKG'si.

OS-43

Myocarditis induced after heavy nimesulide usage

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A 47-year-old male patient came to emergency department with a complaint of chest pain which got worse in the last 24 hours. The patient denied any history of cardiac disease or complaint and he had no classic risk factors. Physical examination gave normal findings. Electrocardiography was quite normal. Normal findings except a slight hypokinesia of lateral wall was detected on echocardiography. The troponin level was 20.98 ng/ml. WBC was 12.400/mm^3 and C reactive protein was 12.40 mg/L. The patient informed that he did not have any symptomatic infection in the last month. On the other hand, the patient mentioned about heavy nimesulid usage (four to five pills per day) for his back pain for nine days. The chest pain was atypical, however, increased troponin levels made us to decide to perform the coronary angiography that revealed completely normal coronary arteries. So the diagnosis of myocarditis was established. On the fourth day of hospitalisation, we could discharge the patient with good clinical status. Many drugs like phenothiazines, amphetamines, cocaine, anthracyclines, alcohol, lithium, tricyclic antidepressants and clozapine can cause acute myocarditis (1). Nimesulide exerts it's effects mainly via COX-2 inhibition. Dinchuk et al demonstrated that half of COX-2 -/- deficient mice had diffuse myocardial fibrosis involving both left and right ventricles (2). He mentioned that COX-2 deficiency might be involved in cardiomyopathy. Moreover, Takahashi et al. stated that inhibition of cyclooxygenase-2 could enhance myocardial damage in a mouse model of viral myocarditis (3). Furthermore, Steer et al remarked that up-regulation of COX-2 expression is considered to play a crucial role for compensatory mechanism in the mouse model of heart failure induced by encephalomyocarditis virus infection (4). To our knowledge, myocarditis as a complication of nimesulide has not been described in the English literature before. We consider that the possible pathophysiological mechanism in this patient with myocarditis is unique and reasonable. That is why we consider that this article would raise awareness of the possibility of myocarditis as a side effect of heavy nimesulide usage and draw physicians' attention on this issue.

OS-44

LVAD'li hastada fungal endokardit

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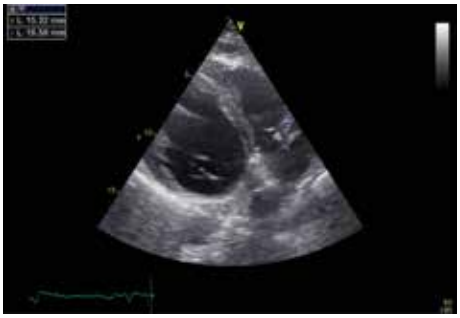
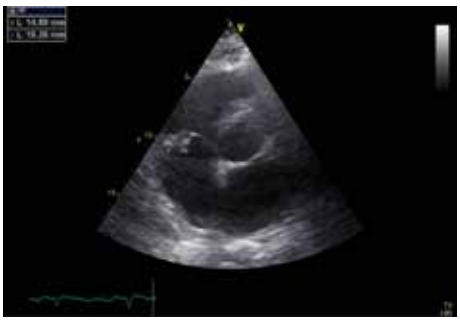
Giriş: İnfektif endokardit geniş intratorasik damarların endarteritini veya kan akımına doğrudan maruz kalan kalp içi yabancı cisimleri de içeren kardiovasküler yapıların endovasküler mikrobik enfeksiyonu olup ölümcül bir hastalıktır. En sık mitral ve aort kapakları tutmakla birlikte son dönem kalp yetmezliği hastalarında yaşam kalitesini artırmak ve kalp nakline kadar diğer organ fonksiyonlarını korumak için kullanılan LVAD'li hastalarda da görülebilmektedir. Sağ kalp endokarditleri ise nadir görülmektedir, en sık intrakardiyak defibrilatörü olanlarda ve iv ilaç bağımlılarında görülür. İnfektif endokarditin en sık sebebi bakteriyel enfeksiyonlardır. Daha nadir olarak fungal enfeksiyonlar da özellikle immunsüprese,damar içi cihazı olanlarda görülebilir. Fungal endokarditlerin mortalitesi daha yüksektir (>%50). Transtorasik ekokardiyografi; infektif endokardit tanısında önemli role sahiptir. Biz bu olgumuzda 3 ay önce LVAD takılan kalp yetmezliği tanılı hastada gelişen sağ kalp fungal endokarditini sunacağız.

Olgu Sunumu: Elli sekiz yaşı bilinen HT,dilate kardiyomiyopati ve ICD öyküsü olan hastada, efor kapasitesinde azalma olması üzerine yapılan TTE tetkikinde LVEF 16, RVEF 25, SPAP 63, TY 3-4, MY 3 saptandı. Hastaya izlemde transplantasyona köprüleme amacıyla LVAD implantasyonu ve triküspit devega annuloplasti işlemi uygulandı. Hasta LVAD implantasyonundan yaklaşık 3 ay sonra, 3 gündür olan ateş, halsizlik ve nefes darlığı ile acil servise başvurdu. Geliş EKG'si sinüs taşikardisiyle uyumluydu. Fizik muayenesinde cihaz nedenli kalp sesleri değerlendirilemedi. Rutinlerinde beyaz küredе, CRP, sedimentasyonda yükseklik saptanması üzerine hastaya yapılan TTE tetkikinde 28x16 mm büyüklüğünde yüksek ihtimalle ICD leadinden köken alan sağ atriyumla sağ ventrikül arasında hareketli vejetasyonla uyumlu görünüm saptandı. Transtorasik ekokardiyografi görüntüleri net olarak değerlendirilebildiği için transösafajial ekokardiyografi planlanmadı. Mevcut bulgularla hasta kalp ve damar cerrahisine yatırıldı. Hastanın ciddi risk faktörleri olduğu ve infekif endokarditin LVAD ilişkili olabileceği göz önüne alınarak ampirik antibiyoterapi başlandı. Kan kültürlerinde candida dublinensis üremesi gözlendi. Hastanın cerrahisi yüksek riskli olduğu için öncelikle antibiyoterapi ile medikal izlemi uygun görüldü. Hastaya kaspofungin, linezolid, piperasilin-tazobaktam tedavisi uygulandı. 45 günlük antibiyoterapi sonrası yapılan TTE tetkikinde triküspit kapakta vejetasyon lehine bulgu saptanmadı. Hasta LVAD fonksiyonları normal ve semptomları gerilemiş bir şekilde taburcu edildi.

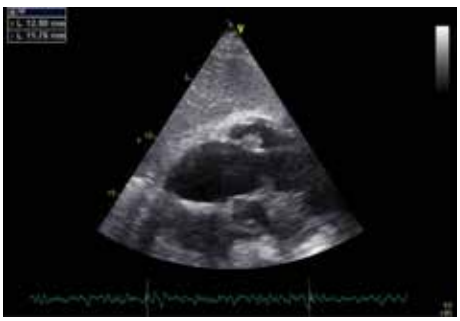
Tartışma: LVAD'in klinik sonuçları iyi olmakla beraber, infektif komplikasyonlar halen bu hastalar için yüksek risk oluşturmaktadır. İnfektif endokardit ise bu hastalarda en korkulan enfeksiyondur. LVAD'li hastalarda infektif endokardite bağlı cihazda ciddi obstrüksiyon, sepsis görülebilir. Mortalitesi yüksektir. LVAD ilişkili fungal endokardit yüksek riskli olması nedeniyle hızlı ve dikkatli tedavi edilmelidir.



Şekil 1. TTE A4B görüntüsünde triküspid kapak ilişkili 1.5x1.6 cm boyutlarında vejetasyon lehine görünüm izlenmektedir.



Şekil 2. TTE PSKA Görüntüsünde triküspid kapak ilişkili 1.5x1.6 cm boyutlarında vejetasyon lehine görünüm izlenmektedir.



Şekil 3. TTE Subkostal Kesit Görüntüsünde triküspid kapak ilişkili 1.3x1.2 cm boyutlarında vejetasyon lehine görünüm izlenmektedir.

OS-45

Protez mitral kapakda fungal endokardite bağlı alt ekstremite arterde emboli olgusu

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Altmış yedi yaşında erkek hasta, eforla nefes darlığı, halsizlik yakınması ve ani başlayan sağ bacakta ağrı ile kliniğimize başvurdu. Anamnez ve fizik muayenede, sağ bacakta ağrı, solukluk, sağ ayak dorsalis pedis ve tibialis posteriyor arterlerde nabızları alınmıyordu. Alt ekstremite dopler ultrasonografik incelemede, sağ popliteal arter düzeyinde tam tıkanıklık bulguları saptandı. Ayrıca yapılan transtorasik ekokardiyografik incelemede, biyoprotez mitral kapak ucuna tutunmuş hareketli kitle, (Video 1) saptandı. Yapılan transözefegiyal ekokardiyografik incelemede, biyoprotez mitral kapak liflet ucuna tutunmuş, hareketli dev vejetasyon görüldü (Video 2–5). Hastaya infektif endokardit tanısı konuldu, kan kültürü alındı, vejetasyonun sağ popliteal artere emboli olabileceği düşünülerek ve kritik bacak iskemi bulguları olması sebebiyle cerrahi olarak embolektomi operasyonu yapıldı. Çıkarılan materyal mikrobiyolojik inceleme sonrası, fungal etyoloji (candida) saptandı. Fungal endokardit tanısıyla, antifungal tedavi başlandı, kalp damar cerrahi ortak konsey kararı ile hastaya mitral kapak replasmanı (MVR) operasyonu yapıldı. Operasyon sonrası klinik stabil seyreden ve antifungal tedavisi tamamlanan hasta şifa ile taburcu edildi. Bir yıldır poliklinik takiplerinde, nüks görülmedi ve halen klinik stabil durumu devam etmektedir.

OS-46

Tüberküloza bağlı koroner yapışıklık

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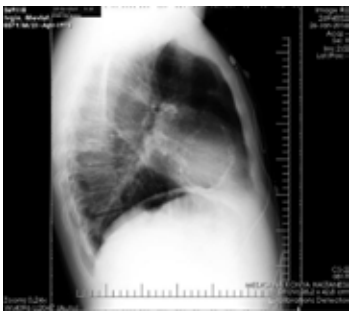
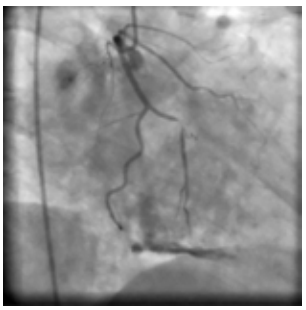
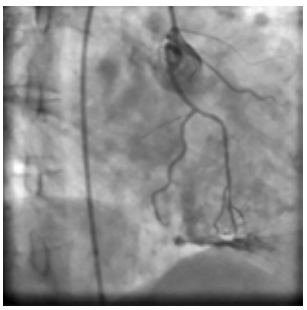
Otuz altı yaşında erkek hasta yaklaşık 2 aydır olan olan göğüste istirahat halinde sıkıştırır vasıfta tipik göğüs ağrısı ile başvurdu. Ateroskleroz için risk faktörü taşımayan aile öyküsü ve sigara anamnezi olmayan hastaya iskemi araştırması yapıldı. Efor testinde iskemi izlenmesi üzerine hastaya koroner anjiyografi planlandı. Hasta anamnezinde tüberküloz için 10 yıl önce tedavi gördüğünü belirtti. Fizik muayenesinde patolojik bulgu yoktu. Çekilen yan akciğer grafisinde kalbi çepeçevre saran perikardiyal kalsifkasyon halkası mevcuttu (Şekil 1). EKG'de prekordiyal derivasyonlarda yaygın t negatifliği vardı. Hastaya koroner anjiyografi yapıldı sol koroner sistem normal idi. sağ koroner arter orta bölgesinde % 95 darlığa sebebiyet veren parikardın koroner artere yapışıklığı gözlendi.(Şekil 2, video 1) Bu bölge tel ile geçildikten sonra 2.25x15 mm ilaç kaplı stent implante edildi. tam açıklık sağlandı (Şekil 3). İşlem sonrası hastanın şikayetleri gerileldi. Tüberküloz perikarditi tüberkülozun önemli bir komplikasyonudur. Genellikle tanısı gecikmeli veya atlanmış şekilde konulur. Konstrüktif perikardit olarak karşımıza çıkması artmış mortalite ile ilişkilidir. Pulmoner tüberkülozun %1-2'sinde ortaya çıkan perikarditin bir kısmı konstrüksiyona sebep olmaktadır. Anti-TBC tedavi ile birlikte periardiyektomi bu hastalarda önerilen tedavi modalitesidir. Hastamızın AC grafisinde ve KAG görüntülerinde kalbi halka şeklinde saran kalsifikasyonla karakterize TBC perikarditinin sekeli izlendi. Hastanın ekokardiyografisinde sistolik fonksiyonları normal, tip 1 diyastolik disfonksiyon mevcuttu. Koroner anjiyografisinde ise literatürde çok rastlanmayan ciddi darlığa sebep olan perkardiyal koroner yapışıklık mevcuttu. damar lümen çapı küçük olduğu için işlem esnasında rüptür veya yırtılma riski mevcuttu. Greft stent hazır bulundurularak ilaç kaplı sent implante edildi. hastaya perikardiyektomi için bilgi verildi ve yakın takibe alındı. Vakayı sunmaktaki amacımız siz olsanız ne yapardınız? sağ sol kalp kateterizasyonu ve perikardiyektomi esnasında koronere müdehale alternatif olabilirmiydi?



Şekil 1. Çekilen yan akciğer grafisinden kalbi çepeçevre saran tüberküloza bağlı kalsifikasyon halkası.



Şekil 2. KAG da ciddi darlığa sebep olan perikardiyal yapışıklık izlenmekte.



Şekil 3. 2.25x15 ilaç kaplı stent sonrası açıklık.

OS-47

Kalp yetersizliği ve sol ventrikül destek cihazı olan hastada cihaz ilişkili endojen endoftalmit

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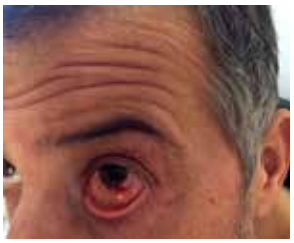
Giriş: Sol ventrikül destek cihazı temel olarak, cilt altına yerleştirilmiş bir pompa olup, ileri kalp yetmezliği hastalarında yakın zamana kadar "transplantasyona köprü"amacıyla kullanılmıştır. Günümüzde, refrakter kalp yetmezliği hastalarında sağ kalımı ve hayat kalitesini olumlu etkileyen uzun dönem bir tedavi gözüyle de görülmektedir. LVAD uygulanması invazif bir işlem olup cilt altında aktif bir pompa bulunmasını gerektirmektedir. Cihaz, sol ventrikül vasıtasıyla sistemik dolaşımla temas halinde olduğundan sistemik infeksiyona gidiş kolay olmaktadır. Endoftalmi, gözici dokuların bakteriler, mantarlar ya da parazitlerce invaze edilmesidir. Eğer patojen vücuttaki bir dokudan vitreusa ulaşmışa endojen endoftalmi terminolojisi kullanılır.

Olgu Sunumu: 48 yaşında erkek hasta 2006' da idiyopatik dilate kardiyomiyopati tanılı ve EF %28 ölçülmesi üzerine 2013'te BIV-ICD implantasyonu sonrası 3 yıldır dekompanse kalp yetmezliği nedeniyle mükerrer hospitalizasyon ve maksimum medikal tedaviye refrakter olması üzerine sol ventrikül destek sistemi uygulandı. Hasta Aralık 2015' te ateş, nefes darlığı nedeniyle, işlevsel durumu NYHA II- III olarak yatırıldı. Ateş nedeniyle taranan odaklarından drive line girişi hiperemik, infeksiyözbelirteçlerinden CRP: 152.5 mg/L, prokalsitonin 2.8 ng/ mL bulundu. Yatışının ikinci gününde sol gözde ani görme kaybı tarif eden hastada endojen endoftalmi tespit edildi (Şekil 1, 2). Drive line'dan ve kandan alınan kültürlerde metisilin dirençli koagulaznegatif stafilokok üredi, vitreustan alınan kültürde üreme olmamakla beraber oküler muayene bulguları tipik endojen endoftalmiyle uyumlu olan hastaya multipl intravitreal vankomisin, seftazidim ve amfoterisin-B injeksiyonları ve sistemik antibiyoterapi başlandı. Antibiyoterapi altında kontrol için alınan 2 set kan kültüründe üreme olmadı. Kaynak kontrolü için drive line revize edildi. İkinci injeksiyondan sonra hastanın görme alanı totale yakın geri kazanıldı.

Tartışma ve Sonuç: LVAD transplantasyon adayı olan son dönem kardiyak yetersizlikte, akut ve kronik kalp yetmezliğinde uzun dönem,ya da kardiyotomi sonrası yetmezlikte; pozitif inotroplar/ intraaortik balon tedavisi altında kardiyak indeksi 2 lt/dk/m^2'nin altında olan ya da sistolik kan

basıncı 80 mmHg altında kalan hastalarda; sağ ventrikül ve böbrek fonksiyonları yeterli olan hastalarda genel olarak güvenle uygulanan bir tedavidir.Ancak batın içi adezyon, perforasyon ve herniler, derin hemorajiler, tromboemboli ve inmeler, sağ ventriküler yetmezlik ya da olgumuzdaki gibi ciddi infeksiyonlarla ilişkili olabilmektedir. Literatürde LVAD ilişkili infeksiyon oranları %30-50 arasında bildirilmiştir ancak bunların arasında hematojen yolla endojen endoftalmit yoktur. Sağkalımı LVAD işlevine bağlı olan bu hasta grubunda ateş,nötrofili- lökositoz, püy gibi bulguların varlığında infeksiyonodakları taranmalı; hastanın kliniğine göre etkilenen alana yönelik tedaviler uygulanmalıdır.

Şekil 1.



Şekil 2. Endojen endoftalmit.

OS-48

A rare complication in a patient with atrial fibrillation: Acute splenic infarct presenting as epigastric and left upper quadrant abdominal pain

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Introduction: Splenic infarction (SI) occurs when the splenic artery or its branches become occluded, either by an embolus or by in situ thrombosis. SI is a rare event but should be considered in predisposed patients or those with any combination of suggestive clinical features, especially abdominal pain. Computed tomography (CT) evaluation is diagnostic and the outcome is good.

Case Report: A 65-year-old female patient with a history of heart failure suffered from shortness of breath. Atrial fibrillation (AF), 129 beat/mn was seen in her electrocardiography (ECG). Past medical history, there was no information about AF. She was hospitalized and anticoagulated with subcutaneous heparin. Two days after admission, a sudden onset of left upper quadrant and epigastric cramping pain developed and she vomited once after finishing dinner. She denied fever, night sweating or heat intolerance. A dull abdominal pain persisted but no rebounding pain or muscle guarding was noted.The bowel sounds were normoactive but there were crepitant rales in basal lungs and minimal pretibial edema. Transthorasic echocardiography (TTE) showed us EF 35% with dilated left ventricula and left atria, moderate (2/4) mitral regurgitation and mild tricuspite (1/4) regurgitation with TAPSE 16. Any thrombus or vegetative pattern was seen in TTE. A plain abdominal X-ray revealed distended intestinal lumen. The patient presented with anemia with a hemoglobin level of 10.3 g/dL. White blood cell (9.3 u/L) and high levels of C-reactive protein (13 mg/dL) and Troponın I (0.04 ng/ml) were also noted. With this preliminary information, ileus and suspected intra-abdominal infection, were the tentative diagnoses. We also drew two sets of blood cultures, and ceftriaxone 2 g daily was prescribed. Abdominal ultrasonography showed gallbladder sludge without remarkable changes of the pancreas, spleen and kidneys. Because of the persistent abdominal pain, we performed contrast enhaced abdominal CT which showed a low-density area within the spleen (Figure 1, 2, 3a and 3b). There was no growth of bacterias or other patogens in blood cultures and no fever during hospitalization. İn detailed physical examination, there was no peripheral signs of infective endokarditis. We consulted the patient to general surgery. General surgeon explained the indication of splenectomy. She was treated with anticoagulative and antibiotic theraphy for 7 days and then underwent on operation for splenectomy. After the surgery the patient was anticoagulated with warfarin and discharged.

Discussion: In conclusion, patients with pre-existing valvular heart diseases, heart failure and AF are prone to embolisation. Some rare complications like SI can be happened by arterial embolisation. A "full" or "typical" picture is often not found and diagnostic suspicion must depend on either the recognition of predisposing factors or attention to suggestive presenting features leading to an early use of a CT scan whenever SI is suspected.

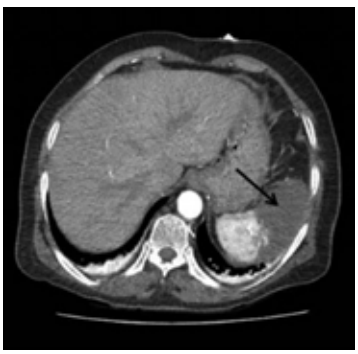

Figure 1. The infarct area greater than 50% of spleen is seen in axial plane CT angiography (arrow).

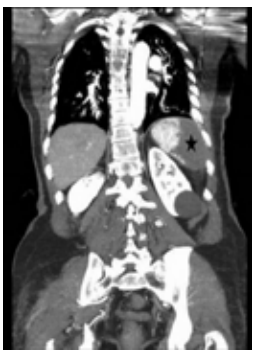

Figure 2. The infarct area greater than 50% of spleen in coronal plane (asteriks).

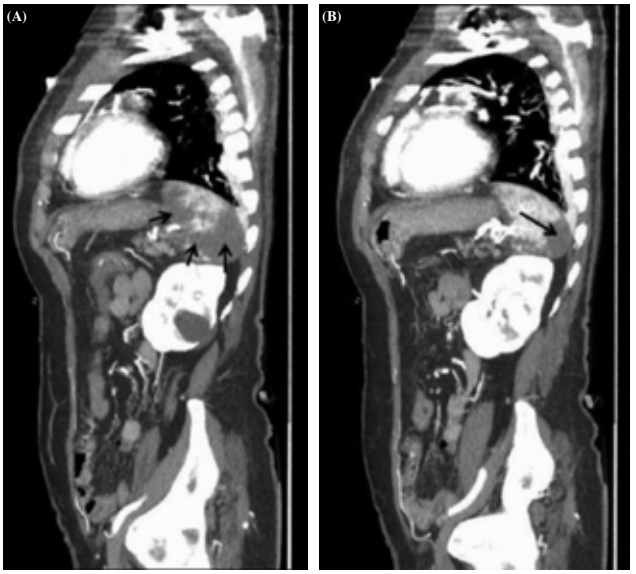

Figure 3. (A) The splenic infarct area is seen in sagittal plane (arrows). **(B)** The splenic infarct area is seen in sagittal plane (arrow).

OS-49

Intermediate artery stenting after implantation of the mitral annuloplasty device due to the device compression

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We report a case who was intermediate artery stented after the implantation of percutaneous mitral annuloplasty (PMA) device with the Carillon system due to coronary compression. A 75- year-old male with nonischemic heart failure, hypertension, atrial fibrillation, diabetes mellitus and severe FMR was referred to our clinic. He was severely symptomatic (NHYA class II-III). There were left ventricular dilatation, global hypokinesia, systolic dysfunction (ejection fraction 25%), and severe FMR on transthoracic echocardiography. There was moderate tricuspid regurgitation and pulmonary artery systolic pressure was 60 mmHg. Left ventricular enddiastolic and endsystolic diameter were 60 and 52 mm, respectively. Vena contracta diameter was 11 mm and left atrium was 50 mm. There were no significant stenosis on coronary angiography. We decided to perform PMA with the Carillon system to the patient because of the high surgery risk. Underfluoroscopy, 9 F guiding catheter was introduced into the coronary sinus. After the coronary sinus angiography with marked pigtail catheter, the distal anchor of the device was released. After the application of adequate tension, which resulted in a decrease in the degree of MR on echocardiography. No compression was done on the mid part of the CX artery. The proximal anchor of the device was released. Before completion of the procedure, coronary angiography was performed to ensure lack of coronary artery compression by the device. After releasing the device, we saw a compression on proximal part of the intermediate artery. A drug eluting stent (2.5x15 mm) was implanted to the comppressed area on the intermediate artery. There were no chest pain, ECG abnormality or stent deformation. The echo parameters of FMR further improved. Vena contracta diameter of the FMR was 11 mm before the procedure and after procedure it was 5 mm. Annular dilatation was also diminished. After one month the implantation of PMA device and stents, the patient was no symptomatic. Left ventricular dilatation was dimished and mild degreee FMR was observed. It was complication related with coronary sinus based annuloplasty with Carillon system for FMR and coronary stenting is a solution for compression of coronary artery due to the Carillon device in our case.

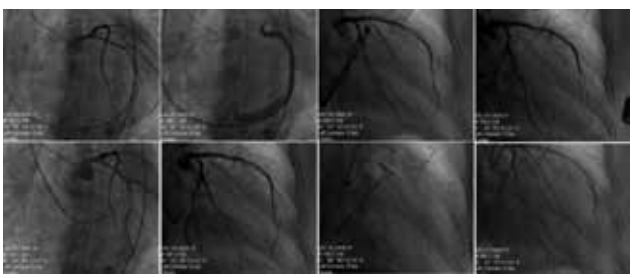

Figure 1.

OS-50

Percutan coronary intervention to single coronary artery in patient with inferior and anterior myocard infarction

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The single coronary artery is a benign and very rare coronary artery abnormality. Anomalous origin of the right coronary artery originating from the left anterior descending artery has been reported previously in just a few cases. In this article, we presented percutan coronary intervention to single coronary artery with anterior and inferior myocard infarction.After PCI we saw that right coronary artery was origined from the midportion of the left anterior descending artery.

Introduction: The term single coronary artery defines coronary arteries originating from a single coronary ostium in the aorta. It is a very rare coronary artery abnormality.The single coronary artery incidence ranges from 0.024% to 0.066% in several series (1,2). This article is first case that shows the three prioporties simultanously:single coronary artery anomalous, the right coronary artery (RCA) originated from the middle of the left anterior descending artery (LAD) and performing a percutan coronary intervention to single coronary artery with anterior and inferior myocard infarction,

Case Report: A 65-year-old male patient presented with a complaint of increasing angina with a duration of three hour. The initial electrocardiogram demonstrated acute anterior and inferior myocardial infarction. After the patient was accepted acute myocardial infarction, the patient was immediately transferred to the cardiac catheterization laboratory and coronary angiography via left trans femoral approach was performed. But coronary angiography was performed diffucult because RCA did not seen at aortography. The left coronary artery angiography revealed normal left main coronary artery(LMCA), total occlusion in the LAD after the D1 branch, mild atherosclerotic disease in the left circumflex coronary artery(LCx) (Fig. 1, Video 1). We decided on PCI in order to restore perfusion in the LAD. After a 6 Fr guiding catheter was placed into the left coronary ostium, balloon dilation was performed in the occluded lession in the LAD using a 2.5 mm balloon. After the baloon dilatation we injected contrast and we saw that the RCA was origined mid LAD. Both RCA and LAD have stenotic lessions. We decided to perform PCI at first RCA then LAD. After the floppy wires were advanced RCA and LAD, 3.0 mm drug-eluted stent (DES) was implanted to RCA. Then 3.0 mm DES was implanted LAD. Lastly, we performed final kissing balloon and ended PCI successfully (Fig. 2, Video 2).

Conclusion: In this Case Report, we presented a very rare anomaly of RCA with unique origin and course which was detected during the PCI.

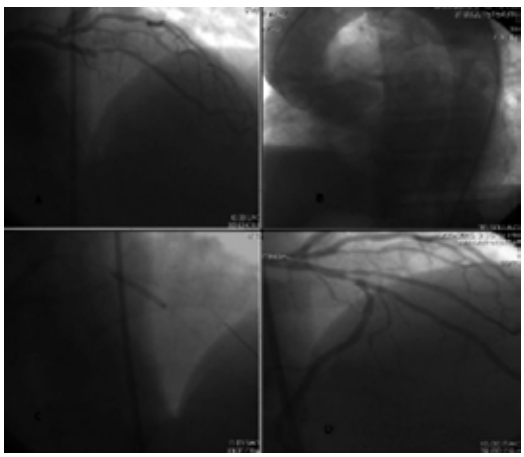

Figure 1. A: LCA anjiogram in left anterior oblique projection with caudal angulation, revealing that normal LMCA, total occlusion of the LAD after the D1 branch B: Aortography C: First baloon anjioplasty for LAD D: LCA anjiogram in left anterior oblique projection with caudal angulation after the baloon anjioplasty.

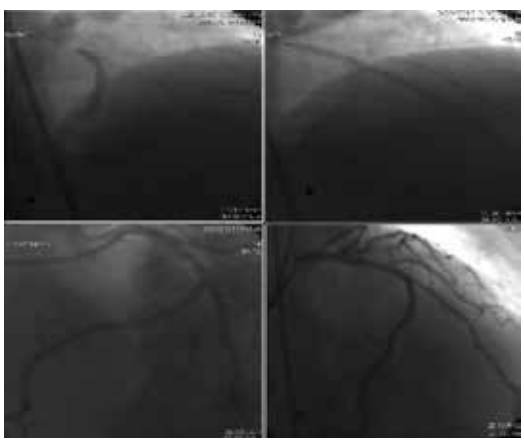

Figure 2. A: Stent implantation for RCA B: Stent implantation for LAD C: LCA anjiogram in left anterior oblique projection with caudal angulation after the Stent implantation D: LCA anjiogram in right anterior oblique projection with caudal angulation after the Stent implantation.

OS-51

Medikal tedaviye dirençli vazospastik angina hastasında koroner stent tedavisi

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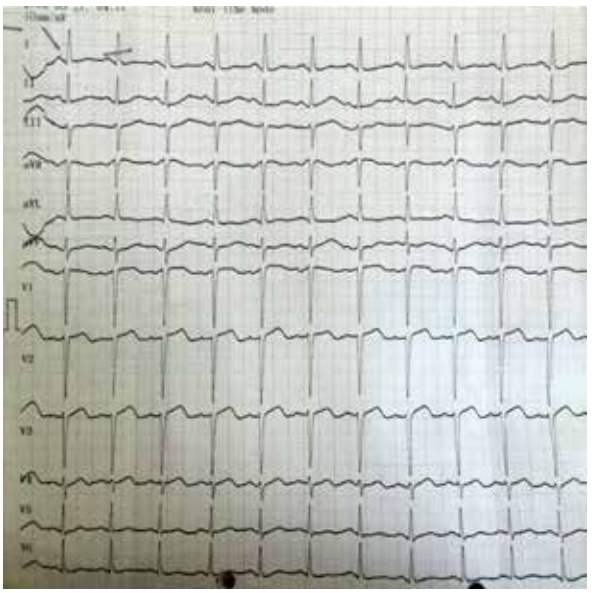
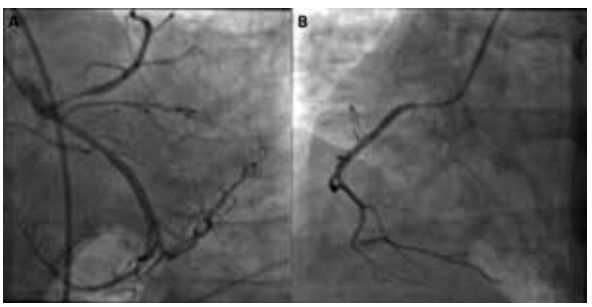
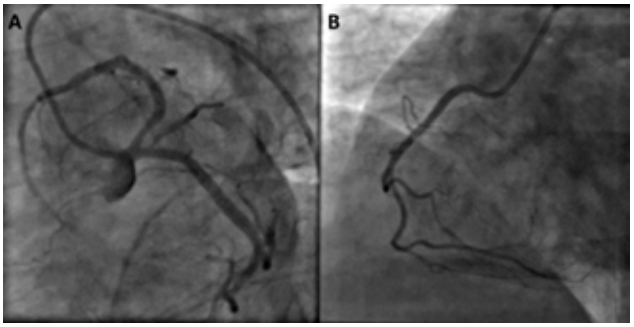
Vazospastik angina (varyant angina, prinzmetal angina) sıklıkla istirahatte gelen ve nitratlara cevap veren göğüs ağrısı epizodları ve eşlik eden ST segment elevasyonu ile karakterize nadir bir klinik durumdur. Epikardiyal bir koroner arterin >%90 geçici daralması ile angina ve iskemik EKG değişiklikleri oluşur. Nadir, ancak ciddi komplikasyonlarından birisi de ventriküler aritmiler ve ani kardiyak ölümdür. Sunacağımız vaka, koroner anjiyografi ile vazospastik angina tanısı almış, kalsiyum kanal blokeri ve nitrat tedavisi altında devam eden iskemiye bağlı dirençli ventriküler taşikardi gelişmiş olan bir hastadır. Tedavide, koroner anjiyografide saptanan vazospastik plaklı bölgeye stent yerleştirilerek tedavisi tamamlanmış ve takibinde asemptomatik izlenmiştir. Hipertansiyon ve yeni tanı Tip 2 diabetes mellitus tanıları olan 46 yaşında kadın hasta son 1 aydır olan, efordan bağımsız tipik göğüs ağrıları nedeniyle koroner anjiografi önerilerek servisimize kabul edildi. Sol ön inen arter (LAD) proksimal ve sağ koroner arter (RCA) proksimalde %50-60 darlık saptanması üzerine yapılan intrakoroner nitrogliserin sonrası darlıklar kayboldu, LAD proksimali plaklı olarak izlendi. Hastada vazospastik angina düşünülerek medikal tedavisi amlodipin 10 mg, isosorbid mononitrat 50 mg 1x1 ve atorvastatin 20 mg 1x1 olarak düzenlenerek taburcu edildi. Şikayetleri gerilemeyen hasta taburculuğunun 10. gününde artan sıkıştırıcı göğüs ağrısı olması üzerine acil servise başvurdu. İzleminde EKG'de V2-V3'de 1 mm ST elevasyonu ve QT uzaması (Şekil 1) izlenen hastanın intravenöz nitrat tedavisine yanıtı olmadı. Koroner anjiyografi planlanan hastanın ani gelişen ve hemodinamiyi bozan monomorfik ventriküler taşikardi atakları olması üzerine kardiyoversiyon yapıldı. İzlemde dirençli ventriküler taşikardi ve hemodinamik bozulma nedeniyle lidokain tedavisi başlanarak ve entübe edilen hasta koroner anjiyografi laboratuarına alındı. LAD proksimalde %95 darlık ve RCA ostiumda izlenen %90 darlık, (Şekil 2a, b) verilen intrakoroner nitrogliserin sonrası düzelmeke beraber LAD proksimal plaklı olarak izlendi (Şekil 3a, b). Hastada koroner vazospazmın amlodipin ve isosorbid mononitrat altında olması ve iskeminin dirençli ventriküler taşikardiye sebep olması nedeniyle LAD ve RCA proksimaldeki bölgeye ilaç salınımlı stent implante edildi (Şekil 4a, b). Hastanın takiplerinde göğüs ağrısı şikayeti olmadı ve verapamil 240 mg, uzun etkili nitrat ve 80 mg atrovastatin tedavisi altında asemptomatik olarak izlemine devam edilmektedir. Vazospastik anginada ilk basamak tedavi uzun etkili kalsiyum kanal blokerleri ve nitratlardır. Maksimum medikal tedaviye dirençli olgularda stentleme uzun vadeli sonuçları net olmasa da tedavi yöntemlerinden birisidir. Vazospastik angina ciddi ventriküler aritmi ve ani kardiyak ölüm ile sonuçlanabilen klinik bir durumdur. Sunduğumuz vaka, medikal tedaviye rağmen malign aritmik iskemi yaratması nedeniyle koroner stentleme ile tedavi edilmiş nadir bir vakadır.



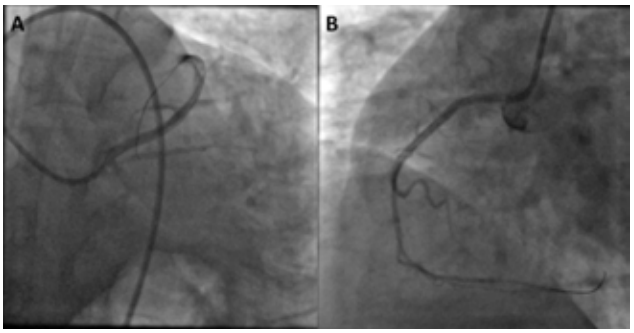
Şekil 1. Hastanın başvuru EKG'sinde V2-V3 de ST elevasyonu ve QT uzaması görülmektedir (QTc:0,50 msn).



Şekil 2. Koroner anjiyografide LAD proksimalde %95 darlık ve RCA ostiumda izlenen %90 vazospastik darlıklar.



Şekil 3. İntrakoroner nitrogliserin sonrası vazospazmda düzelme, plaklı LAD proksimal segmenti izlenmektedir.



Şekil 4. Koroner stentleme sonrası LAD ve RCA proksimal segmentin görüntüsü.

Diğer

OS-52

STEMI responsible of stomach ulcer to the cardiologist

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Mechanical complications of MI are rare but their severity imposes their research clinically and can be confirmed by echocardiography. The management of these complications is very delicate and usually surgery. We present clinical case of 83 years old man with an important chest deformation, presented to our emergency unit for MI. We transfer the patient to the cath-lab, the angiogram in femoral access found a thrombotic total occlusion of the proximal LAD treated by primary PCI. After thromboaspration we found that the 0.014 wire is over a septal artery on wich there is an aneurysmal pocket, we rewired the real LAD and performed PCI. Echo on bed in cardiac care unit found an increasing septal aneurysm and the angio control found a worsning of the aneurysm confirming a septal artery perforation and and important septal wall dissecting hematoma treated by 3 inflations (for 15 mn each one) of small NC balloon in the septal artery to clotte the hematoma. Results After the third inflation the hematoma clotted and the echo picture got the same density as all myocardium without any mechanical complication (pericardial effusion or septal defect). Wall dissecting hematoma is very rare complication during PCI, it has been also described after VSD repair and cardiac resynchronisation therapy implantation. We speculate a septal artery perforation by wire and Export and blood erruption in necrotic myocardium. For late evolution modalities, the haematoma may expand, rupturing into adjacent structures, or resolve. In case of large hematoma, we propose to achieve: Prolonged balloon inflation for 10-15 min at 2-6 atm to clotte the hematoma. Covered stent. Rarely, one or more conventional stent (BMS/DES) to exclude perforred artery. Artery embolization using coils. Surgical treatment.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

OS-53

Intracranial stent implantation

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Introduction: Intracranial stent placement is applied as a rescue therapy for inadequate or failed response to thrombolysis and mechanical thrombectomy in acute stroke patients. Although TICI score is high, there is increased intracranial bleeding risk with baloon expandable stents. This case report describes an intracranial stent implantation in a patient with acute stroke.

Case Report: A 71 year old woman admitted to emergency department with weakness in the right arm and leg. Past medical history included hypertension, diabetes mellitus, Alzheimer disease. Cranial CT showed secondary changes due to brain atrophy. There was decreased flow through left middle cerebral artery on brain CT angiography. The diagnosis was acute ischemic stroke. The patient was taken to catheterisation laboratory. There was total occlusion of distal left internal carotid artery before T segment. After passing total occlusion with Whisper LS guidewire, mechanical thrombectomy was performed with Solitaire TM 6.0x30 mm. Although we repeated the procedure, there was still 90% stenosis at the distal part of left internal carotid artery (Figure 1). 2.25x18 Endeavor drug eluiting stent was implanted (Figure 2). Finally improved blood flow through occlusion was observed (Figure 3).

Discussion: Acute ischemic stroke is a major cause of mortality and morbidity in Turkey. Catheter based treatment gives promising results. There is sometimes challenging situations like inability to seat with guiding catheters, failed response to thrombolysis and mechanical thrombectomy. Intracranial stent implantation is an alternative treatment. Further investigations needed to show long term results.

Conclusion: Intracranial stent implantation is an alternative treatment strategy when there is inadequate or failed response to mechanical thrombectomy.

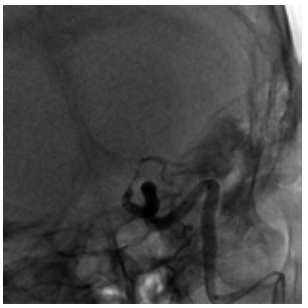

Figure 1. There was 90% stenosis of distal ICA despite repeated thrombectomy.

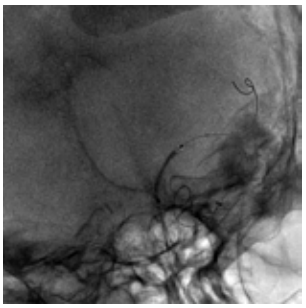

Figure 2. DES was implanted at the distal portion of ICA.

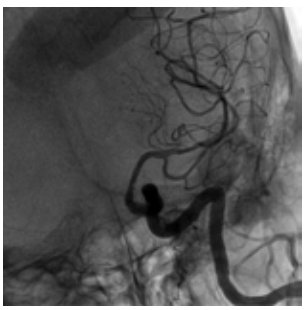

Figure 3. After implantation, there was increased blood flow through stenosis.

OS-54

Perkütan koroner anjiyoplasti sırasında gelişen akut strok: Hızlı revaskülarizasyon, iyi prognoz

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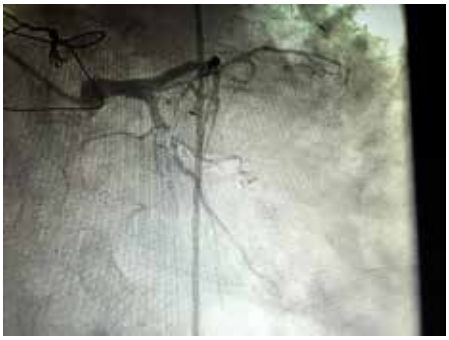
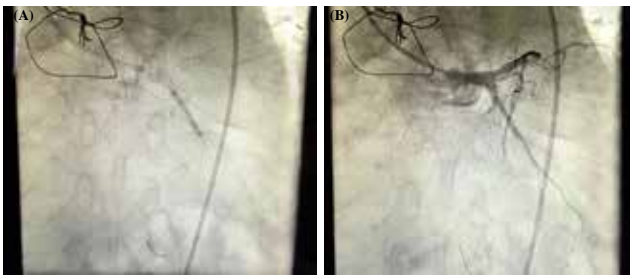
Giriş: Koroner anjiyografi ve girişimler sırasında strok gelişme insidansı düşük olmakla birlikte (%0.2-%0.4), bu durum mortalite ve morbiditesi yüksek bir sağlık sorunudur. Böyle bir durumda erken uygulanan serebral anjiografi ve revaskülarizasyon bu ciddi durumda bile iyi sonuçlar elde edilmesine olanak sağlamaktadır. Bu vakada 79 yaşında ciddi aort darlığı ve kalp yetmezliği olan bir hastada CX arterine uygulanan perkütan girişim sırasında sağ middle serebral arterin(MCA)'in akut oklüzyonuna bağlı strok gelişmiş ve takibinde zaman kaybetmeksizin revazkülarizasyon sağlanmıştır.

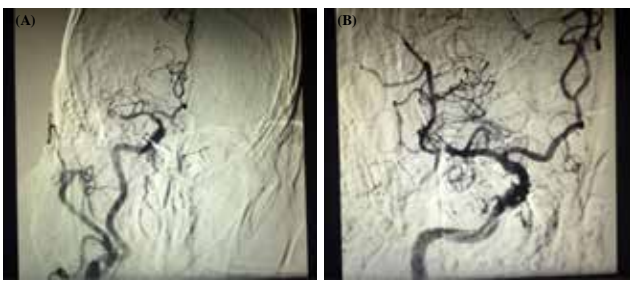
Olgu Sunumu: 79 yaşında geçirilmiş koroner bypass ameliyatı, diyabet, kalp yetmezliği ve ciddi aort darlığı olan hasta nefes darlığı şikayeti ile başvurmuştu. NYHA sınıf III dispnesi olan hastanın transtorasik ekokardiyografisinde ciddi aort darlığı (ortalama gradient: 46 mmHg), EF: %28, hafif-orta derece mitral yetersizliği ve hafif-orta derece aort yeterizliği saptandı. Hastaya transkateter aortik kapak implantasyonu (TAVI) hazırlığı yapılması planlandı ve bu plan doğrultusunda koroner anjiografi uygulandı. Koroner anjiografide RCA konüs dalı sonrası %100 (antegrad kolleteraller ile distal doluş mevcuttu), LAD 1. diyagonal sonrası %100, CX 1. obtuse marjinal dalı sonrası %90 tromboze darlık (Şekil 1), 1 adet Aort-Safen grefti %100 tıkalı, LIMA-LAD açık olarak saptandı. Hastanın CX %90 darlığa girişim kararı alındı ve sol ana koronere extra backup kateteri ile oturuldu. Sonrasında hastaya kateter içinden 10000 IU fraksiyone olmayan heparin bolus olarak uygulandı. Lezyondan 0.14 inç kılavuz tel ile geçildikten sonra öncelikle lezyona 2.0x15 mm balon ile PTCA yapıldı. Sonrasında sirolimus kaplı 2.75x18 mm stent lezyona implante edildi (Şekil 2a, b). Kontrol görüntü alındığı sırada hastada ani başlayan bulantı ve bilinç bulanıklığı gelişti. Yapılan nörolojik mauyenede akut sol hemipleji saptandı. Hastaya akut strok tanısı konulduktan sonra aynı seansta hemen selektif serebral anjiografi uygulandı ve sağ MCA'nın %100 tıkalı olduğu saptandı (Şekil 3a, b). Sağ internal karotis arter Envoy kateter ile kanule edildi. Sonrasında 0.14 inç tel ile lezyondan geçildi. Tel üzerinden gönderilen mikrokateter lezyonun distalinde bırakıldı. Mikrokateter içerisinden gönderilen prESet 6-30 mm geri alınabilir stent (Şekil 4) ile trombektomi işlemi yapıldı (Şekil 5a, b). Aynı işlem üç kez tekrarlandı. İşlem süresince 1 mg/dk infüzyon hızında 5 mg'lık tPA uygulamaları 4 kez yapıldı. Toplamda intraarteriyal 20 mg tPA uygulandı. İşlem sonrası tam rekanalizasyon sağlandı ve işleme son verildi (Şekil 6). Yoğun bakımda 24 saat intravenöz heparin infüzyonu ile takip edilen hastaya ikili antiplatelet tedavi başlandı. 6 saat sonra bilinç bulanıklığı düzelen hasta 5. gününde solda 4/5 oranında motor kuvvet kaybı ile taburcu edildi.



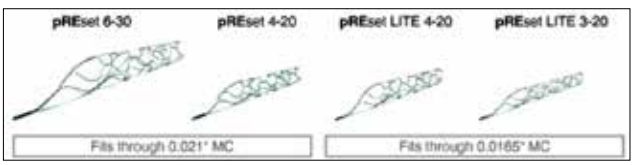
Şekil 1. CX 1. OM sonrası tromboze %90 darlık.



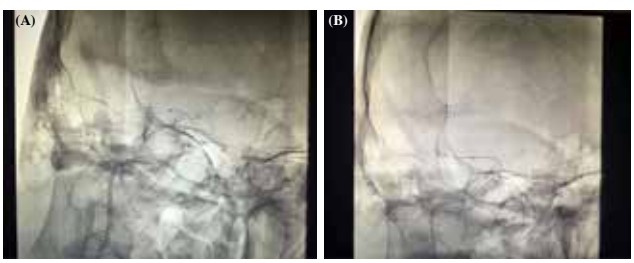
Şekil 2. (A) CX lezyonuna stent yerleştirilmesi. **(B)** CX'de stent implantasyonu sonrası sağlanan akım.



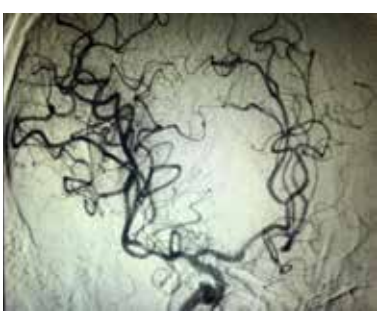
Şekil 3. (A) Sağ MCA %100 oklüzyonu. **(B)** Sağ MCA %100 oklüzyonu.



Şekil 4. prESet 6-30 mm geri alınabilir stent.



Şekil 5. (A) prESet 6-30 mm geri alınabilir stent ile trombektomi. **(B)** prESet 6-30 mm geri alınabilir stent ile trombektomi.



Şekil 6. Sağ MCA'da sağlanan tam açıklık.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

OS-55

Karotis anjiografi sırasında gelişen akut inme vakasına kardiyolog tarafından başarılı mekanik embolektomi uygulaması

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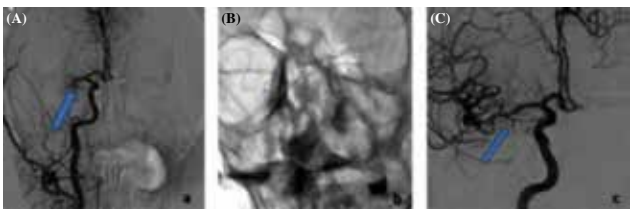
Perkutan koroner- karotis görüntüleme veya girişimsel işlemlere bağlı iskemik strok bilinen bir komplikasyondur. Girişimlere bağlı sıklıkla geniş serebral arterleri ilgilendiren iskemik strok görülür ve en önemli sebep kateter manipülasyonu sonrası aortadan ateromatöz debris embolizasyonudur. Akut iskemik strok tedavisinde fibrinolitik uygulama veya kateter-temelli trombolizis-trombektomi yapılabilmektedir. Ancak girişime bağlı gelişen strok için kateter-temelli tedavi uygulaması, strok merkezleri ve nöroradyolog sayısının az olması sebebiyle mümkün olamamaktadır. Bu sebeple girişime bağlı gelişen akut iskemik strok vakalarına tecrübeli kardiyologların müdahalesi önem kazanmaktadır. Altmış yedi yaşında erkek hasta 2 hafta önce geçirdiği geçici iskemi atak (TIA) sonrası çekilen dopler usg de sol karotiste %50 darlık saptanması üzerine karotis anjiografi amaçlı merkezimize başvurdu. 2011 de koroner stent hikayesi var. Elektrokardiyografi sinüs ritminde, kalp hızı 76/dk. Hipertansiyon, hiperlipidemi ve diabetes mellitus sebebiyle ilaç kullanmakta. Sağ femoral yoldan sheat takılarak 2500 Ü heparin bolus yapıldı. Hastaya konvansiyonel koroner anjiografi yapıldıktan sonra sağ 4J kateter ile sağ karotis arter ve vertebral arter görüntülendi. Tip3 arkus aorta mevcut hastaya 4JR ile angajman mümkün olmayınca 5F Simmons kateter ile denendi. Çeşitli manevralar yapılarak sol karotis artere oturulduktan sonra görüntüleme yapıldı ve anlamlı darlığa neden olmayan plak saptandı. Sol karotis görüntüleme sırasında hastada konuşma bozukluğu, takibinde sol hemipleji gelişti. Nöroloji ile konsülte edilmesi sonrası intrakranial görüntüleme yapılmasına karar verildi. Sağ karotis arterden görüntüleme sonrası sağ orta serebral arterde (MCA) akut tıkanıklık izlendi (Şekil 1a). Stiff wire desteği ile 6F shuttle yerleştirildikten sonra, road map kullanılarak, koroner choise floppy wire ile MCA daki akut tıkanıklık geçildi. Koroner trombüs aspirasyon cihazı ilerletilerek tekrarlayan trombüs aspirasyonları yapıldı ve akım sağlandı (Şekil 1b, c). Hemodinamisi stabil seyreden hastanın 10 dakika sonra kontrolünde akımda yavaşlama izlenmesi üzerine trombüs aspirasyou tekrarlandı. Sonraki kontrollerinde akımın sebat ettiği gözlendi. Hasta yatağına alınırken sol ekstremite hareketlerinin geri döndüğü izlendi. Takip eden saatlerde nörolojik fonksiyonları tamamen normalleşen hasta taburcu edildi ve semptomsuz takibi devam etmektedir.



Şekil 1. (A) Sağ orta serebral arterde (MCA) akut tıkanıklığa bağlı kesilen akım izlenmekte. **(B)** MCA wire ile geçilerek embolektomi yapılmaktadır. **(C)** Embolektomi sonra rekanalize olan MCA görülmekte.

Diğer

OS-56

Hypereosinophilic syndrome with cardiac involvement and multiple cerebral embolisms

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The hypereosinophilic syndromes are defined as overproduction of eosinophils and eosinophilic infiltration and tissue demage in multiple organs. Hypereosinophilia is defined as absolute eosinophil count is greater than 1500 cells /microliter in peripheral blood sample or histopathologic confirmation of eosinophilic tissue infiltration. We report a case of hypereosinophilia with cardiac involvement and multiple cerebral embolisms. A 28 years old male have been presented with complaints of atypical chest pain and weakness for three months. His physical findings were not remarkable except of digital splinter hemorrhage (Figure 1a). Electrocardiogram showed sinus rhythm with T wave inversion and ST segment depression in inferolateral leads (Figure 1b). Transthoracic echocardiography revealed normal left ventricular ejection fraction and modest mitral regurgitation with very thick hyperechoic endocardium especially in left ventricular apical region (Figure 1c). In complete blood count, white blood count and eosinophils count were 36000 cells / microliter and 29000 cells /microliter respectively. He became apathic and disorientated three days after admission. Diffusion brain MRI indicated multiple embolisms including bilateral parietooccipital lobes (Figure 1d). Mature eosinophils was seen in peripheral blood smear and bone marrow sample (Figure 2a and b). We don't find any cause of secondary eosinophilia such as parasitic infection,churg-Strauss syndrome. Thoracaabdominal CT indicates splenomegaly and supraclavicular lenfadenopathy. Supraclavicular lenf node biopsy was nondiagnostic for lymphoma and another tumors. Tumor markers are negative. He was diagnosed as an idiopathic hypereosinophilic syndrome. He was treated with 100 mg/day intraveneous methyl prednisolone but eosinophil count was not dropped. We performed transesophageal echocardiography. We saw mobile echoic mass in left ventricule and the patient was transferred to cardiovascular surgery clinic for operation. Diagnosis of hypereosinophilic syndrome is based on 3 criteria (1) an eosinophil count of more than 1500 cells per microliter for at least 6 months, (2) no other evident cause for eosinophilia, and (3) organ involvement signs by eosinophilic infiltration. Hypereosinophilic syndrome is a heterogeneous disease which can either be completely asymptomatic or involve multiple organ systems. Cardiac involvement includes progressive subendocardial fibrosis with mural thrombus formation, which can lead to peripheral emboli and restrictive cardiomyopathy.

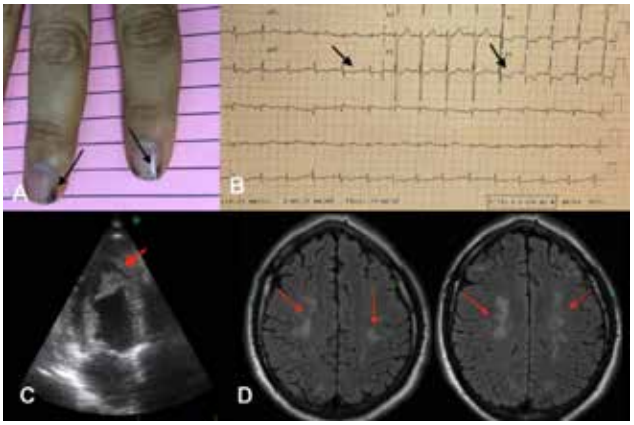

Figure 1. Digital splinter hemorrhage **(A)**, sinus rhythm with T wave inversion and ST segment depression in inferolateral leads **(B)**, very thick hyperechoic endocardium especially in left ventricular apical region **(C)** and multiple embolisms including bilateral parietooccipital lobes **(D)**.

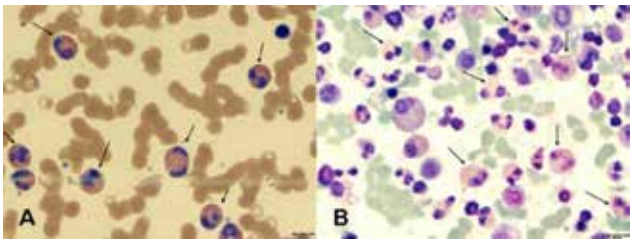

Figure 2. (A, B) Mature eosinophils in peripheral blood smear and bone marrow sample.

Pulmoner hipertansiyon / Pulmoner vasküler hastalık

OS-57

P-ANCA associated microscopic polyangitis: A neglected diagnosis underlying the pulmonary hypertension associated with chronic thromboembolic pulmonary artery disease

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Pulmonary arterial hypertension (PAH) is a severe condition characterized by chronic obstruction of small pulmonary arteries leading to progressive right heart failure. Inflammatory mechanisms may play an important role either in the etiology or progression of pulmonary hypertension (PH). Although, connective tissue disease is a well-known cause of PH, systemic vasculitis are generally overlooked as a cause of PH. Here-in we describe a case of PH associated with microscopic polyangiitis which is a rare necrotizing vasculitis characterized with auto antibodies against the constituents of neutrophil cytoplasm.

Case Report: A 75-years old woman was admitted with newly developed dry cough, dyspnea and lower extremity pain. She had a diagnosis of pulmonary embolism for two years and chronic atrial fibrillation. She was not receiving any anti-coagulant treatment. on physical examination, we find that lower extremity dimension differences. Her laboratory assessment showed pancytopenia with high pro-BNP level and high D-dimer value. She had moderate tricuspid regurgitation, systolic pulmonary arterial pressure of 60 mmHg associated with right ventricular dilatation and normal left ventricular systolic functions on echocardiographic examination. Doppler ultrasonography revealed new onset deep vein thrombosis in the left lower extremity. Computed angiographic tomography demonstrated middle and lower segmental perfusion defects in the territory of left pulmonary artery. Etiologic assessment of recurrent thrombosis for a possible vasculitis revealed that she was positive for ANCA profil mpo +++ and EANCA-P 1/320(+). So she was put on corticosteroids and anticoagulation with the diagnosis of microscopic polyangiitis associated with pulmonary hypertension.

Discussion: Although rare, vasculitis should be kept in mind in patients with PH and recurrent thrombosis even in the old patients. Microscopic polyangitis, is a rare p-ANCA associated vasculitis that could cause thromboembolic events leading to PH. Moreover, it could also cause diffuse hypoxic necrotizing interstitial lung disease and eventually hypoxic PH. the positive p-ANCA levels reveals the diagnosis. And in these patients corticosteroids and specific anti-inflammatory treatment are needed for the resolution thrombi.

OS-58

Cardiac tamponade as the initial presentation of systemic lupus erythematosus

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A 50-year-old woman admitted to our emergency department with a compliant of ever-increasing shortness of breath for 5 days. Her medical history was unremarkable except hypertension. On examination, blood pressure was 88/54 mmHg, pulse rate was 128 per minute and body temperature of 36.5 degree Celsius. Jugular venous distension was obvious. Furthermore, there was rash on her face. Respiratory examination revealed right sided crackles and and decreased breath sound at bases of right lung field. Laboratory investigations revealed anaemia with low platelet count (91.000/mm^3). ESR was 49 mm/h and CRP was 11 mg/dl. Moreover, creatinine level was 1.98 mg/dl. Electrocardiogram showed low voltage. Echocardiogram confirmed features of cardiac tamponade that was large circumferential effusion with early diastolic right ventricle collapse. Emergency pericardiocentesis with subxiphoid approach was then performed and 500 ml of pericardial fluid was drained. After pericardiocentesis, her symptoms' resolved. Her serum was strongly positive for antinuclear antibody as well as positive anti-double stranded DNA antibody. Serum complement levels were low. Pericardial fluid was exudative in nature. Pericardial fluid cytology showed cells of an inflammatory response with no microorganism identified by Gram stain or Ziehl-Neelsen stain. PCR for tuberculosis was negative. There was no evidence of malignancy. The diagnosis of SLE was established based on the positive clinical and immunologic findings.The patient was started high dose prednisolone along with non-steroidal anti-inflammatory drugs and hydroxychloroquine. Pericarditis is very common in SLE and has been shown to occur in 60% of patients but cardiac tamponade is truly rare both as the initial manifestation and throughout the disease course. Physicians must consider SLE as the differential diagnosis of cardiac tamponade and perform convenient tests to diagnose connective tissue diseases. Because, immunesuppressive treatment is the mainstay therapy of connective tissue disorders that should be diagnosed correctly, thus, recurrence of pericardial effusion or even pericardial tamponade could be prevented.

OS-59

Cardiac tamponade concomitant peripheral arterial thrombosis

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Pancreatic adenocarcinoma accounts for about 85% of pancreatic cancer. Pancreatic adenocarcinoma represents diverse, nonspecific clinical manifestations, including an abdominal mass, epigastric pain, abdominal discomfort, poor appetite,and anorexia. We report here a patient with pancreatic adenocarcinoma presenting arterial thrombosis and cardiac tamponade. A 51 years old man was admitted to our emergency department with progressively increasing dyspnea and right leg pain. His past medical history was unremarkable except for active smoking 1 pack of cigarettes daily. Physical examination showed vital signs as follows: body temperature of 37.2°C, blood pressure of 110/85 mm Hg, heart rate of 115 beats per minute, and respiratory rate of 19 breaths per minute. Oxygen saturation was 90% on inhaled room air. His lung sounds were clear bilaterally and heart sounds were muffled. A resting ECG showed sinus tachycardia. The peripheral vascular examination was performed that there was no pulse on the right side below the knee, all others could be palpated. A lower extremity Doppler ultrasound and echocardiography were immediately performed. Echocardiography revealed a large pericardial effusion (3.4 cm) with causing compression of right heart chambers (Figure1). The results from the Doppler showed that there was a monophasic flow pattern (most likely an artefact) below the right popliteal artery. The Cardiovascular Surgery department was consulted, it was decided to make an lower extremity angiography. In the angiography we saw that there was a huge thrombus, which caused a subtotal occlusion in the right main arteria ilaca and total occlusion after the right popliteal artery (Figure2). The patient who suffered from intense pain in his leg by that time was referred the cardiovascular department for a trombectomy and pericardiocentesis. During operation 700 ml of haemorrhagic fluid was drained from the pericardial space and the thrombuses from the iliac and popliteal artery were removed with a trombectomy catheter. Postoperation all peripheric pulses were palpated again and the perfusion of the extremities was normal. Also a second echocardiography was performed again that there is no pericardial effusion. The pericardial fluid samples were analyzed for diagnosis at biochemistry and patology laboratory. The results from the pathology showed that an adenocarsinoma metastasis, the primary origin was from the pancreas. In conclusion; although pancreatic adenocarcinoma usually represent nonspesific clinical manifestations, cardiac tamponade concomitant peripheral arterial thrombosis may rarely be first presentation of pancreatic adenocarcinoma.

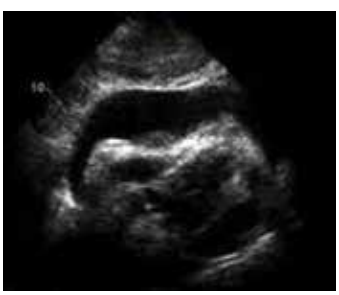

Figure 1. Cardiac tamponade.

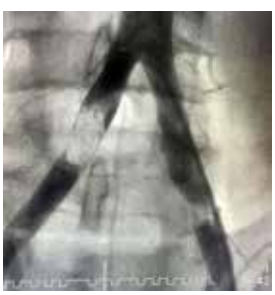

Figure 2. Thrombosis.

OS-60

Akut süperior mezenterik arter tromboemboli: Başarılı perkütan translüminal anjiyoplasti ve trombolitik tedavi

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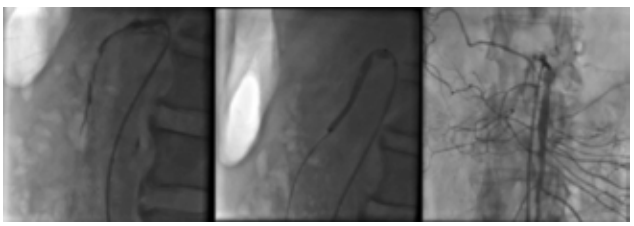
Süperior Mezenter Arterin (SMA) kısmi ya da tamamen tıkanması masif barsak nekrozu ile sonuçlanabilir. Hastalığın kliniği ve semptomlarının spesifik olmaması tanı ve tedavide gecikmelere neden olur. Akut mezenterik iskemi (AMI) ölümcül bir vasküler acil olup mortalitesi %50–%80 arasındadır. Süperior Mezenterik Arterin (SMA) kısmi ya da tam tıkanması sonucu intestinal iskemik hasar gelişebilir.İskemik hasar klinik şikayetlerin spesifik olmamasına ve tanının gecikmesine bağlıdır. Erken tanı akut intestinal iskemiye bağlı morbidite ve mortaliteyi azaltır.

Olgu Sunumu: Altı saat önce başlayan nefes darlığı ve karın ağrısı olan 75 yaşında erkek hastanın son üç saatten beri göbek çevresindeki şiddetleyen ağrı, bulantı-kusma ve çarpıntı şikâyetleri ile acil servise başvurmuştur. Koroner bypass, kalp yetmezliği, hipertansiyon ve diabetes mellitus hikayesi olan hastanın fizik muayenesinde tüm batında yaygın hassasiyet vardı. Defans ve rebound gibi peritoneal irritasyon bulguları yoktu. Fizik muayenesinde; her iki akciğer bazalında ince ralleri olan hastanın kan basıncı: 180/110 mm/Hg, nabız: 160 atım/dk. ve disritmik saptandı. Ateş: 37.3 C°, solunum sayısı: 32/dk idi. Laboratuvar bulgularında; Hemoglobin: 15.7 g/dl, Beyaz küre sayısı: 17 000 U/L, kreatinin: 1.69 mg/dl, ALT:15 IU/L, AST: 15 IU/L, CK-MB: 32 U/L, Troponın I: 1.88 ng/ml olarak bulundu. EKG de yüksek ventrikul hızlı atrial fibrillasyon (Kronik AF). Genel durumu kötü olan hastaya sol femoral arter yoluyla invaziv arter monitorizasyonu sağlandı. Sağ femoral ven yoluyla santral venöz kateter yerleştirilen hastaya iv nitrat, tedavileri başlandı. Ekokardiyografide sol ventrikül dilate ve ejeksiyon fraksiyonu %15-20 olarak bulundu.Hastanin devam eden karın ağrısı nedeniyle yatak başı yapılan ultrasonografik incelemesinde SMA' de renkle dolum saptanmadı. Hastaya SMA anjiosu yapıldı. Ön-arka projeksiyonda yapılan aortagrafide her iki renal arterde dolum sağlanmışken SMA'de akım izlenmediği görüldü. Sağ amplatz guiding kateter ile SMA kanüle edilerek yapılan selektif anjiyografide SMA proksimalinde ciddi darlık ve damarda yoğun trombüs izlendi (Şekil 1). 0.035'' hidrofilik guide wire ile lezyon geçildi ve 5x60 mm balon ile anjiyoplasti işlemi yapıldı. Yoğun trombüs aspirasyon kateteri ile trombektomi yapıldı. Damar tam açıldı (Şekil 1). Yoğun trombüs yükü sebebiyle direkt SMA'e yerleştirilen kateter ile 15 mg bolus ve 35 mg infüzyon şeklinde, heparin infüzyonu ile beraber 1 saat içinde, toplam 50 mg rt -PA trombolitik tedavi uygulandı. Hastanın karın ağrısı tam olarak geriledi. Hasta ileri takıp için yoğun bakıma alındı.

Tartışma: Akut mezenterik iskemi şüphesi olan hastalarda anjiyografi öncelikli tanı yöntemidir. Anjiyografiyle tıkanıklığın yeri ve kollateral dolaşımın varlığı gösterilebilir. Fibrinolitik tedavinin etkinliğinin değerlendirilmesinde anjiografik olarak pıhtının lizisi, karın ağrısının ortadan kalkması ve abdominal muayenenin normal olması önemlidir.



Şekil 1. Süperior mezenterik arter ön-arka anjiografi görüntüleri.

OS-61

Treatment of severe subclavian artery stenosis with jailed balloon, filter protection and reactive hyperemia techniques

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Gaziantep Dr. Ersin Arslan State Hospital, Gaziantep

59 year old male patient suffered from dizziness, vertigo and left arm claudication. One year ago cranial MRI revealed diffusion abnormalities in left cerebellum. Left arm arterial pulses were weak ECG and echocardiography was normal DSA showed critical stenosis of left subclavian artery at the level of left vertebral artery orifice. Aspirin, clopidogrel and LMW-heparin was prescribed and after one week intervention was planned. Spider filter was placed to left brachial artery and apposition of filter was checked by angiography. Stenosis was crossed with glidewire and exchanged with distal filter. Blood pressure cuff was inflated for maintaining reactive hyperemia. 8x40 mm self-expandable stent was deployed after inflation of jailed 3.0x15 mm balloon at v1 segment of vertebral artery. Aim was to prevent plaque shift to vertebral artery. Firstly pressure cuff, secondly jailed balloon and finally postdilation baloon were dilated. Jailed balloon was easily retrieved after the procedure. Procedural steps: blood pressure cuff inflated, jailed balloon inflated, postdilation of stent was done, jailed balloon deflated, blood pressure cuff deflated, reactive hyperemia was achieved, postdilation balloon was deflated, filter was retrieved. This case shows successful and safest treatment of severe subclavian artery stenosis involving vertebral artery orifice. Three methods of protection was used: 1. Distal antiembolic filter. 2. Jailed and inflated balloon at v1 segment. 3. Reactive hyperemia maintained by blood pressure cuff inflation and deflation a sequential treatment approach was used.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

OS-62

Treatment of an iatrogenic left subclavian artery dissection by the left radial artery approach in a patient with CABG

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Iatrogenic dissection of the subclavian artery during cardiac catheterization is a rare complication that is occasionally referred in literature, because of low incidence. We present a case of stent treatment of a the left subclavian artery dissection caused by wire in a patient with a left internal mammary artery graft. Treatment was performed by angioplasty and stent placement, using a retrograde left radial approach.

Case Report: A 67-year-old man with a history of hypertension, diabetes mellitus, hypercholesterolemia had coronary artery bypass graft surgery that included a LİMA graft to the LAD. Due to exertional chest pain, left heart catheterization was performed from right femoral aproach. For the imaging of the LIMA, diagnostic JR4 catheter and 0.035-inch hydrophilic wire were pushed forward from the left subclavian artery and wire was not progressed. Contrast media injection was showed us the dissection of the subclavian artery. The dissection was rapidly progresse until total occlusion (Figure 1a). A 6-French sheath was introduced into the nonpulsatile radial artery with Doppler technique. A 0.014 inch guidewire (300 cm) was passed through the dissected area of the left proximal subclavian artery to the descending aorta (Figure 1b). Guidewire was captured with a endovascular snare system (12-20 mm) from the 6-French JR4 guiding catheter and radio femoral wire loop has been occured. From the femoral sheath, dissected segment of the subclavian artery was passed through with a 5 -Fr diagnostic catheter and 0.035 inch hydrophilic guidewire was placed to brachial artery. Then the diagnostic catheter was replaced with a right guiding catheter.Percutaneous angioplasty was performed with a 5x40 mm balloon (Figure 2a). Following balloon deflation, a balloon expandable stent 8x59 mm was positioned in the proximal subclavian artery.The stent was deployed and angiography revealed that the stented area to be satisfactory with a good antegrade flow and absence of residual dissection (Figure 2b). He was discharged without any complication.

Discussion: The complication of iatrogenic subclavian artery dissection during diagnostic and interventional cardiac procedures is described as an infrequently condition and usually occurs when attempting to cannulate internal mammary artery grafts. The management of the dissection can range from either a conservative or interventional approach according to the side branch vessels involved and distal flow. The importance of this complication and the urgency for immediate treatment was determined by the degree of dependence of the coronary circulation to the grafted LIMA. The retrograde approach from the radial artery is preferable to antegrade approach from the femoral artery, possibility of failure to cross the dissected area, due to getting through the false lumen with the wire. Correct placement of the stents is very important, that distal part of the stent can not extend to origin of the vertebral artery and LİMA.

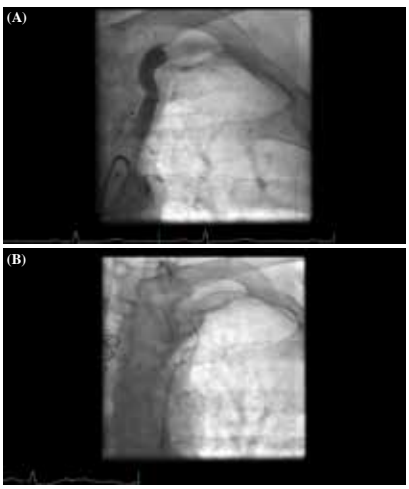

Figure 1. (A) Antegrade cannulation of the left subclavian artery results in a dissection with TIMI 0 flow distally. **(B)** Passing through the dissected segment of the subclavian artery to the descending aorta with a 0,014 inch guidewire.

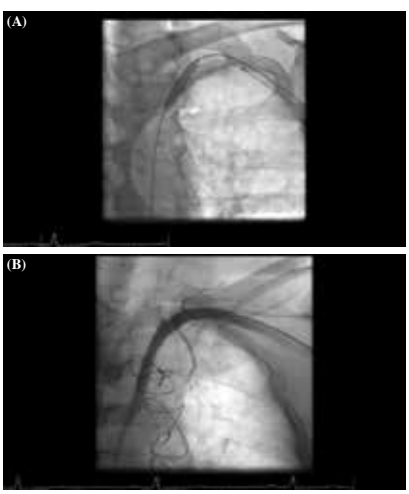

Figure 2. (A) Angioplasty of proximal subclavian artery with a balloon. **(B)** Stent placement of subclavian artery with a good antegrade flow and absence of residual dissection.

OS-63

Right truncus brachiocephalic stenosis presented with digital necrosis

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A 39 year-old female patient presented with severe pain and digital necrosis on the fingers of her right hand. During her physical examination, no pulse was detected in her right radial or ulnar arteries; her fifth finger had been amputated from its distal phalanx (Figure 1a). The patient was smoker 18 years/pocket and had been implanted with a coronary stent due to acute coronary syndrome. Echocardiography revealed depressed left systolic function without any thrombosis in the heart. Electrocardiography showed normal sinus rhythm. The patient was taking clopidogrel 75 mg, acetylsalicylic acid 100 mg, ramipril 5mg, furosemide 40 mg, spironolactone 25 mg daily, rosuvastatin 20mg daily and beta blocker. Angiography of the upper right extremity was performed and revealed 98% percent occlusion of the right truncus brachiocephalic artery without any antegrade collateral flow (Figure 1b). Next we performed percutaneous peripheral intervention and a balloon-expandable 8x19 mm stent was implanted in her right truncus brachiocephalic artery (Figure 1c). Post-intervention control angiography showed patency of both radial and ulnar arteries (Figure 1d). Soon after revascularization her complaint of pain progressively improved; additionally, other signs of necrosis were resolved (Figure 1e). Routine laboratory tests were unremarkable and specific vasculitis and other rheumatological diseases were not found. She had no symptoms and her fingers were completely normal at her sixth-month follow-up visit. Ischemic symptoms at distal edges of an extremity, even together with necrosis, must be evaluated with angiography and time allowed after revascularization for healing before amputation is undertaken.

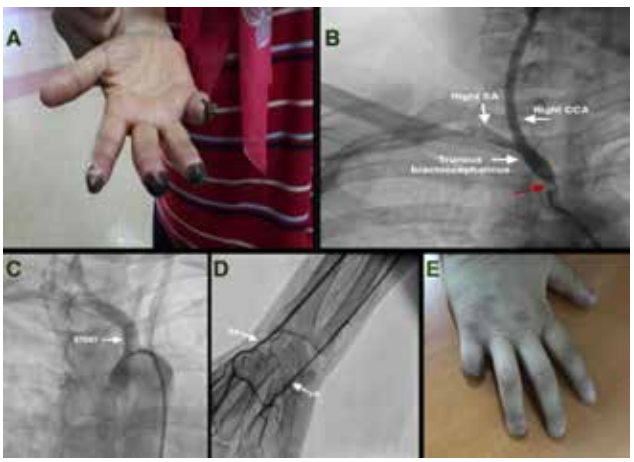

Figure 1. (A) Figure shows digital necrosis in fingers and fifth finger had been amputated from its distal phalanx. **(B)** Angiography of upper right extremity shows 98% percent occlusion of right truncus brachiocephalic artery. **(C)** Balloon-expandable 8x19 mm stent to right truncus brachiocephalic artery was implanted. **(D)** Angiography shows patency of both radial and ulnar arteries. **(E)** After revascularization, necrosis was resolved.

OS-64

Extraction of fragmented catheter material through contralateral femoral sheath following unsuccessful snare attempt during left superficial femoral artery stenting

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Increasing number of interventions in cardiology have resulted a parallel incease in complications such as broken catheter tip,balloon or guide wire embolisation.The most commonly used and effective method for the percutaneous retrieval of such broken fragments is using snare. However in cases where this technique has been a failure, operators have to make prompt decisions for the retrieval of such broken fragments during the procedure. A 56-year-old male was taken to catheterization laboratory to have an implantation of self expandable 'Supera' stent (Abbott Vascular, USA) to occluded left superficial femoral artery. The Destination 6 French-45 cm guide sheath (Terumo, Japan) was inserted via right femoral artery to the left common femoral artery. 5x200 mm Supera stent was firstly implanted following 4x120 mm, 5x60 mm balloon predilatation. Since there was a suboptimal result,we planned to implant second 7x60 mm Supera stent while stent delivery catheter tip was broken during the procedure. We tried to grasp the piece which was still on the wire with loop snare (Amplatz GooseNeck Snare Kit, ev3 Europe, Paris, France. Multiple attempts failed to pull back the disconnected segment into the sheath. So,we placed a 6 French sheath contralateral femoral artery. The 0,018" Control wire was snared via contralateral sheath establishing through-and-through femoral access. Even if we brought out the wire we were unable to extract the broken segment in the wire. We decided to push the catheter tip forward by placing a 3,5x20 mm balloon and finally succeeded to extract the broken piece from contralateral sheath. The dissection occured in left common iliac artery and common femoral artery was treated by implanting 7x150 mm Protégé EverFlex (ev3 Endovascular Inc, Plymouth, Minn) and 8x100 mm S.M.A.R.T.® Flex stent (Cordis Corporation) after placing a second control wire from Destination catheter. The puncture sites were closed with 6 Fr Angioseal" (St. Jude Medical) and the procedure was ended. Excessive elongation might be associated with breakage of delivery catheter tip as shown in our case and care must also be taken not to extend the stent too much during deployment of self expandable Supera stent.

Figure 1. Broken self expandable Supera catheter delivery tip.

Diğer

OS-65

A very rare complication of a very common invasive cardiac procedure: Ischemic renal infarction due to the disrupted aortic thrombus after coronary angiography

Çağın Mustafa Üreyen[1], Yunus Cem Baş[1], Görkem Kuş[1], Murat Esin[1], Emre Asiltürk[1], Özkan Kayhan[1], Gamze Korkmaz[1], Seren Ay[1], Sibel Üreyen[2], Göksel Çağırcı[1], Şakir Arslan[1]

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A 63-year-old female patient applied to emergency department with a complaint of typical angina. After giving the dual antiplatelet therapy, the patient was taken to cath-lab one day after admission. The decision of maximal medical therapy was made after coronary angiography. The patient described a flank pain, however, it was supposed to be due to bed rest after coronary angiography. Twenty four hours after discharge, the patient came to emergency department with a complaint of macroscopic hematuria and increasing left flank pain. The computerized tomography (CT) demonstrated a thrombus of 3x1 cm in size in the aortic arch (Figure 1). It also showed contrast free areas in the low and middle part of the left kidney parenchyma indicating acute infarction of the left kidney (Figure 2). Renal function returned to normal after a slight rise of creatine and the pain disappeared after 2 days, hence, the patient was discharged with warfarin. Renal infarction is a relatively rare disease with the incidence of 0.004-0.007%, giving rise to overlooking in most cases. Renal infarction is commonly found in patients with atrial fibrillation, intra-cardiac thrombus, valvular heart disease or infective endocarditis, thromboembolism, homocysteinemia, malignancies, coagulapathy and a history of trauma and invasive interventions. In our case, thromboembolism was thought to be caused by disrupted aortic thrombus, which can be seen very clearly in CT pictures. The coronary angiography was supposed to be the responsible procedure causing to disrupt the thrombus in the aorta. The CT pictures of abdomen showed wedge-shaped left renal density consistent with an acute renal infarction. The thrombus image in left renal artery was remarkable. Our case demonstrated a very rare complication of coronary angiography, which is the most common invasive procedure in cardiology practice. To the best of our knowledge, any case of renal infarction after a coronary angiography with a possible pathophysiological mechanism of thrombus disruption from arcus aorta to renal artery has not been reported before. We consider that it is an important aspect for physicians for this mostly overlooked pathology especially in patients presenting with abdominal/flank pain after invasive cardiovascular procedures.

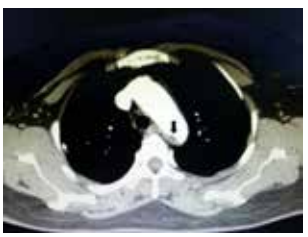

Figure 1. Thrombus in the aortic arch.

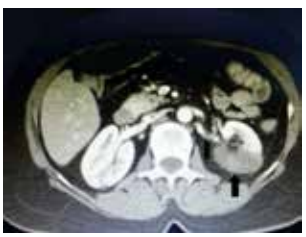

Figure 2. Black arrows demonstrating acute renal infarction and renal artery thrombus.

Diğer

OS-66

Akut assendan aort disseksiyonunda diastolde sol ventriküle sarkan flep

Doğaç Çağlar Gürbüz[1], Ersin Çağrı Şimşek[2]

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Asendan aort diseksiyonu genelde akut başlangıçlı semptomların görüldüğü, mortalitesi yüksek bir hastalıktır. Burada diyastolde sol ventirküle prolabe olan flep nedeniyle aort yetmezliği gelişen akut asendan aort diseksiyonu olan bir hastayı sunduk. Kırksekiz yaşında erkek hasta, 1 haftadır olan göğüste sıkıntı hissi ile kardiyoloji polikliniğine başvurdu. Bilinen hipertansiyon öyküsü olan hastanın kardiyak hastalık öyküsü yoktu. Fizik muayenede sol sternal kenarda 3/6 diastolik üfürüm duyuldu. EKG de iskemi belirtileri olmayan hastada ventriküler ekstrasistol dışında patolojik bulgu yoktu.Yapılan ekokardiyografide; asendan aorta çapı 56 mm, aort içerisinde sol ventriküle diastolde prolabe olan ve ciddi aort yetmezliği neden olan flep saptandı (Şekil 1a-c) Aort kapağı normal kalınlıkta ve üç yaprakçıklıydı. Hasta asendan aort diseksiyon tanısıyla acil operasyona verildi. Post-op kardiyak tamponat gelişen hasta tekrar operasyona alındı. Takibinde genel durumu kötüleşen akut böbrek yetmezliği ve kardiyojenik şoka giren hasta post op 41. günde ex oldu. Diseksiyon flebinin aortadan prolabe olması sonrası gelişen aort yetmezliği aort diseksiyonun mortalitesini arturan bir komplikasyondur. Acil cerrahi müdahale gerekir. Ancak bizim vakamızda olduğu gibi başarılı cerrahi müdahelye rağmen postop komplikasyonlar nedeniyle bu hastalar kaybeledilebilir.

OS-67

Acute lower extremity paralysis after successful lower extremity endovascular intervention

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61 year old man presented at outpatient clinic complaining of severe claudication. He had history of diabetes, hypertension and smoking. Physical examination revealed absence of right femoral, poplietal, dorsalis pedis pulses and weak left lower extremity pulses. Blood pressure and heart rate were measured 140/95 and 80 beat/min respectively. ECG was normal. Computed Tomography (CT) angiogram showed total occlusion of right common iliac artery, 80% stenosis of left iliac artery and collateral circulation supplying right external iliac artery. Percutaneous intervention was decided. Patient lied in a supine position and he was prepped and draped in a sterile fashion. Left and right common femoral arteries were punctured and 6F sheats were placed. Left iliac stenosis was stented with 7.0x59 and 7.0x39 balloon expandable stents (Omnilink Elite, Abbott Vascular), first. Then, 0.35 Glidewire and Navicross microcatheter (Terumo) were advanced to the distal cap of right iliac occlusion respectively. Occlusion was passed with Glidewire and then microcatheter was taken out. Angioplasty was performed with 7.0x40 mm balloon with 10 atm (FoxCross, Abbott Vascular). 9.0x59, 8.0x59, 8.0x39 mm balloon expandable stents (Omnilink Elite, Abbott Vascular) were implanted successfully (Figure 1a). 300 cc contrast medium was used during procedure. After completion of procedure the patient was not able to pass from the angiography table to transport stretcher. After couple of failed attempts, we realized that the patient can not elevate his right leg. Physical examination revealed abdominopelvic distention and patient's inability to flex thigh. He was hemodynamically stable with 130/80 mmHg blood pressure and 85 beat /min heart rate. Hemotacrit was stable with a level of 33%. Despite stability of the patient, hemotama compressing lumbar nerve plexus remained the most important pathology to exclude. Contrast enhanced abdominal CT scan did not show any hemotama. However significant bulging of lateral wall of urinary bladder was apparently displayed (Figure 1b). Volume rendered 3D images depicted compression of femoral nerve between a pouch like bulging of bladder and iliac stents (Figure 1c). 14 F foley catheter was placed and 500 ml of urine drained. Six hours after foley catheter placement, patient's complaint disappeared and he easily flexed his thigh. Two days after procedure he underwent a thorough abdominal USG examination after drinking 1500 ml water without urination. Examination showed normal findings and excluded any anatomic pathology including diverticula or pseudodiverticula. He was discharged on 3rd day of procedure and followed up uneventfully.

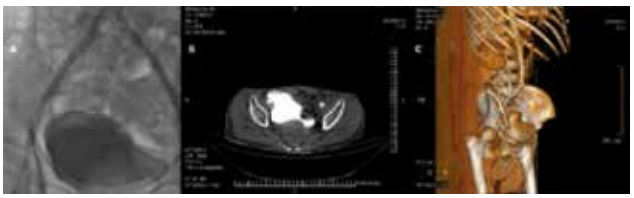

Figure 1. (A) Final angiogram displaying patent right iliac artery lumen and stents. **(B)** Computed tomography showing bulging of bladder on iliac stents. **(C)** 3D volume rendered image depicts right displacement of bladder on stents resulting femoral nerve compression.

OS-68

Endovascular treatment of brachial fistula stenosis

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67 years old male was admitted our clinic because of ineffective hemodialysis. From his past history we learned that he was taking hemodialysis for eleven months but during last month hemodialysis could not be effective because of the low flow. After patients examination due to the weak fistule improvement we decided to make angiography.There was 90 percent stenosis after 5 cm from anastomosis on the venous segments (Figure1, Movie 1). Predilatation with 2.5x25 mm and 5.0x20 mm balloons were performed. 4.5x18 mm bare metal coronary stent was successfully implanted (Movie 2). After stent implantation was inflated 5.0x12 non-compliant balloon for post dilatation (Figure 2, Movie 3). After three months follow up patient has no complaint and hemodialysis treatment goes on without any problem. We would like to demonstrate a brachial fistula stenosis case and its alternative treatment option.

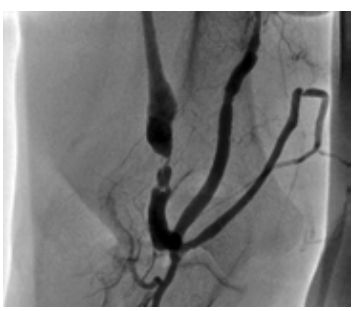

Figure 1.

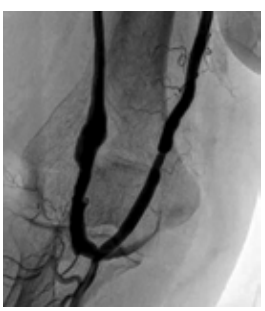

Figure 2.

OS-69

Endovascular treatment of venous occlusion presenting superior vena cava syndrome in a patient with Wegener Granulomatosis

Erdem Karaçöp, Ahmet Bacaksız, Asım Enhoş, Özge Özden Tok, Aydın Nadir, Muharrem Nasifov, Abdulkadir Yıldız, Mustafa Ahmet Huyut, Nuray Kahraman Ay, Mehmet Ergelen, Ömer Göktekin

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Introduction: Superior vena cava syndrome (SVCS) is usually associated with chronic upper extremity deep vein thrombosis and has a high mortality rate. Clinical presentation is arm and facial swelling, dyspnea, visual disturbances and stridor. It is more likely due to malignant etiologies (lung carcinoma, lymphoma) and less commonly rheumatologic disease. İmmediate diagnosis and treatment is essential for survival. This report describes an endovascular tratment for superior vena cava syndrome in a patient with Wegener Granulomatosis.

Case Report: An 80 year old woman admitted to emergency department with right arm and facial swelling. Her medical history included Wegener Granulomatosis and end stage renal failure. She was on hemodialysis treatment for eight years. Physical examination revealed unilateral edema and pain on right upper extremity and neck. Bilateral rales were heard at the basal portion of lungs. Electrocardiography showed atrial fibrillation, heart rate:98/bpm, increased magnitude of QRS on V4-V6. Echocardiography revealed left ventricular hypertrophy, EF: 58%, diastolic dysfunction, moderate mitral regurgitation. Contrast venography showed total occlusion of superior vena cava and right subclavian vein (Figure 1). After predilatation, 8.0x10 mm stent was implanted in subclavian vein and 9.0x40 stent was implanted in superior vena cava (Figure 2, 3). Finally improved blood flow through occlusion was observed (Figure 4).

Discussion: Superior vena cava syndrome is a clinical emergency. The underlying pathology is usually venous thrombosis. It is associated with malignancy, hypercoaqulable states, dialysis access. Venography is the gold standart for evaluation of subclavian vein and superior vena cava. The traditional treatment strategy is radiotherapy and chemotherapy for malignant etiologies. Surgery is rarely indicated. Disadvantage of radiotherapy is high reccurence rate. Endovascular treatment of superior vena cava occlusions is lifesaving and must be classified as first- line therapy.The technique should be taken into account for both malignant and benign disease. It has high succes rate.

Conclusion: Endovascular treatment of superior vena cava and subclavian artery occlusions gives promising results and could be regarded as first line treatment.

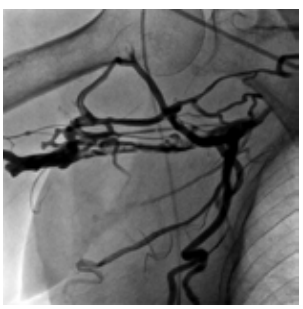

Figure 1. There was total occlusion of subclavian ven in venography.

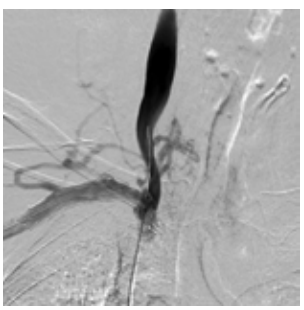

Figure 2. After stent implantation in subclavian vein.

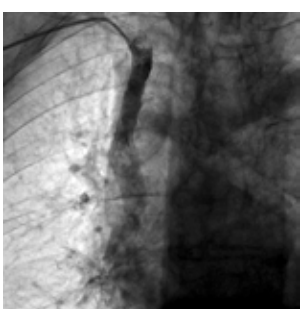

Figure 3. Stent implantation in superior vena cava.

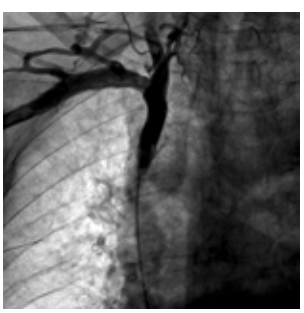

Figure 4. Improved blood flow after stent implantation.

OS-70

Hemodiyaliz hastasında kronik brakiyosefalik ve subklavyan ven trombozlarına endovasküler stentleme

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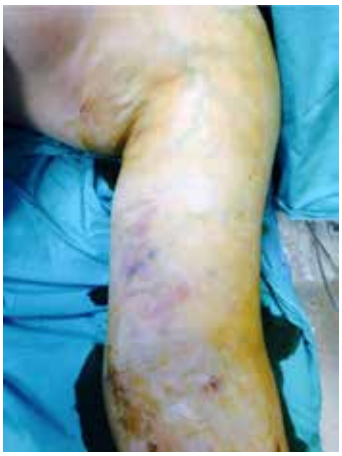
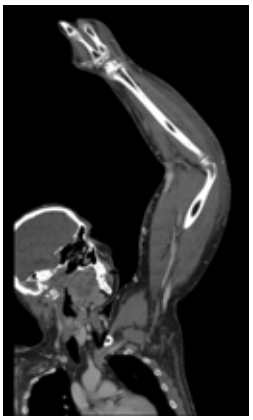
Yaklaşık 20 yıldır diyabetik nefropatiye sekonder böbrek yetersizliği nedeniyle hemodiyaliz uygulanan 47 yaşında erkek hasta 3 aydır artarak devam eden sol kolda şişlik ve hareket kusuru şikayetiyle hastanemize başvurmuştur. Sol üst kol çapı 35 cm ölçüldü (Şekil 1). Sol brakiyal arter sefalik ven arasında bir yıl önce yeni fistül açılmıştır. Sol üst ekstremite bilgisayarlı tomografik anjiyoda sol subklaviyan venin proksimal kesimi ve sol brakiyosefalik ven kronik trombozla uyumlu oklüde izlendi (Şekil 2). Hasta arteriyovenöz fistülün kapatılmasını kabul etmedi. Perkutan girişim kararı verildi. Sağ femoral vene 8F kılıf yerleştirildi. Unfraksiyone heparin 10000 IU (100 IU/ kg) iv bolus uygulandı. Venografide, 7F MP kateterle sol brakiyosefalik venin oklüde olduğu izlendi

(Video 1) ve güdüğe kadar kateter ilerletildi. 0.035 inç stiff body ile oklüzyon geçilmeye çalışıldı, ancak penetrasyon sağlanamadı. 5F Bern kateter denenmesine rağmen yine oklüzyon geçilemedi. 0.035 inç telin arka sert kısmıyla oklüzyon penetre edildi. 90 mm kılıfsız ve 0.035 inç minie mikrokateter ile oklüzyon geçildi (Video 2). Ardından, 7.0x100 mm ve 8.0x80 mm periferik balonlar (simeks) 8 atm'de şişirildi (Video 3). Distal bölgeye 10.0x80 mm self-ekspandable nitinol stent (Epic) (Video 4) ve proksimal bölgeye de 9.0x37 mm balon ekspandable stent (Express) (Video 5) 10 atm'de implante edildi. Daha sonra, 10x40 mm periferik balon (simeks) ile 10 atm'de overlap bölgesine postdilatasyon uygulandı (Video 6). Başarılı açılım sağlandı (Video 7). Ertesi gün, sol koldaki şişlik geriledi. Sol üst kol çapı 25 cm ölçüldü (Şekil 3). Medikal tedavisi düzenlenip hasta taburcu edildi. Üst vena kava, brakiyosefalik, subklavyan ve juguler venlerin ciddi darlıklarını kapsayan santral ven hastalığı diyaliz hastalarının yaklaşık %23'ünde görülmektedir. Tedavi gerektiği durumda, güncel kılavuzlar perkutan transluminal anjiyoplastiyi ve lüzum halinde endovaskuler stentlemeyi önermektedir.



Şekil 1. İşlem öncesi sol koldaki ödem.



Şekil 2. Bilgisayarlı tomografide sol brakiyosefalik ve subklaviyan venlerin oklüzyonu (okbaşı).

Şekil 3. İşlem sonrası sol koldaki ödemin gerilemesi.

OS-71

Recurrent aortic dissection treated by uncovered multilayer flow modulator stent

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Introduction: Repair of Stanford type B aortic dissection should be considered in patients with refractory pain under medical treatment and in whom with uncontrolled hypertension. In this paper, we reported a case of recurrent aortic dissection treated by thoracic endovascular aortic repair (TEVAR) using uncovered multilayer flow modulator (MFM) stent.

Case Report: A 55-year-old man who had history of vascular surgery five years ago due to dissection of ascending aorta was admitted to emergency service with acute chest pain. A new dissection starting from arcus aorta extending to celiac trunk and suprarenal abdominal aorta and dilatation of the proximal ascending aorta were shown in computed tomography (Figure 1). The heart team consisting of cardiologist, cardiovascular surgeon, and anesthesiologist decided to endovascular treatment because redo aortic surgery was considered highly risky with 9 of logistic EuroSCORE. The patient was conservatively treated three days. After reaching private stents, two MFM stents (Cardiatis, Isnes, Belgium) were successively implanted with a 3-cm overlap from the ascending aorta (due to instructions of manufacturer) to bifurcation of abdominal aorta, covering the entire aneurismal segment of the proximal aorta with safety margin of the stent (Figure 2). The patient was discharged at postoperative third day.

Discussion: Type B aortic dissection is treated either by medical or by invasive procedures including surgery and endovascular repair. Recently, TEVAR is favorable option thanks to advances in stent technology. Covered stent is mostly used type for TEVAR. However, covered stent is inconvenient if dissected area covers an important organ artery. Nowadays, MFM stents have been widely used in this scenario. Sultan et al have described the mechanisms of action of MFM, in detail. The main advantage of these stents is to decrease wall stress with rapid romotion of endothelialization and maintenance of side branch patency.

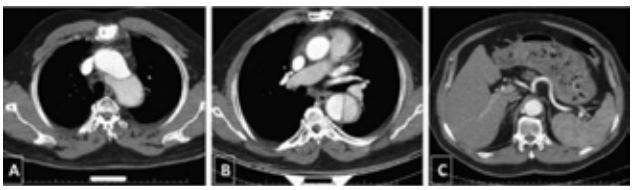

Figure 1. Computed tomography images showing aortic dissection starting from arcus aorta **(A)** and extending to infrasplenic artery **(C)**.

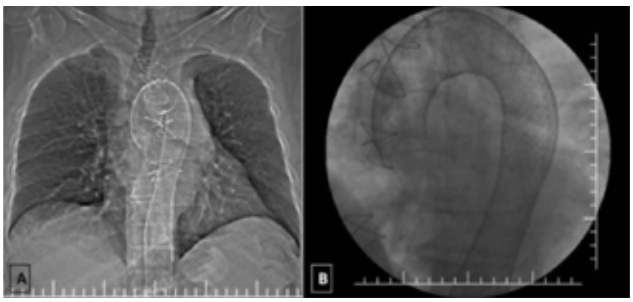

Figure 2. Postoperative computed tomography scan image **(A)** and angiography viewing **(B)**.

Kardiyak görüntüleme / Ekokardiyografi

OS-72

Mitral kapak prolapsusu ile takipli bir hastada altta yatan ender bir konjenital anomali: Çift orifisli mitral kapak

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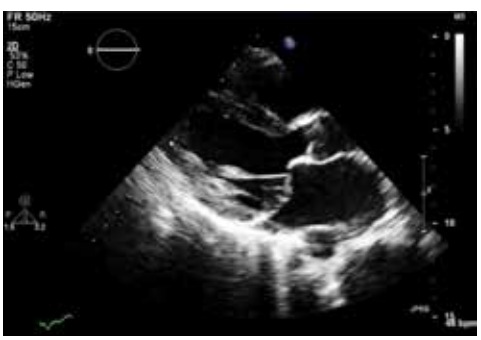
Ankara SB Dışkapı Yıldırım Beyazıt Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, Ankara

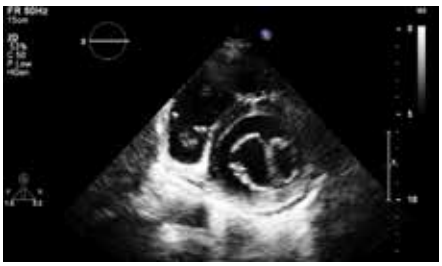
Giriş: İzole çift orifisli mitral kapak (double mitral valve orifice), subvalvüler aparatusun konjenital anomalisi sonucu gelişen nadir görülen bir kardiyak malformasyondur. Çift orifisli mitral kapak, fibröz dokudan oluşan aksesuar bir bağlantı ile ayrılmış anatomik olarak iki farklı orifisten oluşmuştur. Çift orifisli mitral kapak anomalisi önemli derecede mitral yetersizliği veya mitral darlığına neden olabilir. Çift orifisli mitral kapak hastalarının yaklaşık yarısında kapak fonksiyonu yeterlidir. Ancak geri kalan hastalarda çift orifisli mitral kapak önemli derecede mitral darlığı veya mitral yetersizliği ile sonuçlanabilir. Çift orifisli mitral kapak izole veya diğer konjenital kalp anomalileri ile birlikte görülebilir.

Olgu Sunumu: Otuz iki yaşında bayan hasta eforla gelen nefes darlığı şikayetiyle polikliniğimize başvurdu. Özgeçmişinde 6 yıldır mitral valv prolapsusuna bağlı mitral yetersizliği tanıları ile takip edildiği öğrenildi. Elektrokardiyografisi normaldi. Fizik muayenede mitral odakta 2/6 sistolik üfürüm dışında herhangi patolojik bulgu saptanmadı. Yapılan transtorasik ekokardiyografide (TTE) sol ventrikül çapları ve sistolik fonksiyonları normal, ejeksiyon fraksiyonu %65, sol atriyum hafif dilate izlendi. Sistolik pulmoner arter basıncı 40 mmHg saptandı (Şekil 1, Video 1). TTE renkli Doppler incelemede hafif-orta MY izlendi (Video 2). Parasternal kısa aks bazal mitral kapak seviyesi 2D görüntülerde dikkatlice bakıldığında çift orifisli mitral kapak ile uyumlu olabilecek görünüm izlendi (Şekil 2, Video 3). Gerçek zamanlı 3B transözofajiyal görüntülerde (TÖE) sol atriyal taraftan bakışta eşit büyüklükte olmayan medial ve lateral yerleşimli iki adet mitral orifis izlendi (Şekil 3, Video 4, 5). Renkli Doppler incelemede her iki orifisten sol atriyuma doğru hafif dereceli mitral yetersizliği ve sol atriyum boyutunda hafif genişleme mevcuttu. Mitral darlığı bulgusu izlenmedi. Bizim vakamızda, çift orifisli mitral kapağa eşlik eden ek bir kardiyak anomali tespit edilmedi. Hastaya medikal izlem kararı verildi.

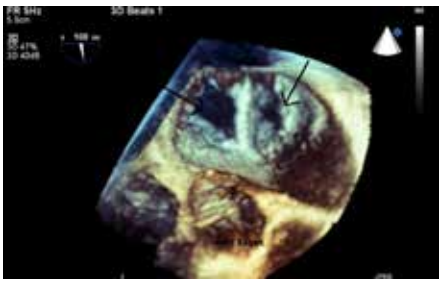
Tartışma: Çift orifisli mitral kapağı, mitral darlığı ve/veya mitral yetersizliği olan hastalarda etiyoloji araştırılırken gözden kaçırılmaması gereken nadir bir anomalidir. MY etyolojisine yönelik 3B TÖE çift orifisli mitral kapağın kesin tanısının konulmasında kullanışlı bir yöntemdir.



Şekil 1. TTE parasternal uzun aks görüntülerde mitral kapak prolapsusu.



Şekil 2. TTE parasternal kısa eksen görüntüde çift orifisli mitral kapak.



Şekil 3. 3B TÖE görüntülerde çift orifisli mitral kapak.

Kalp Kapak Hastalıkları

OS-73

An unusual cause of acute pulmonary edema: Mechanical valve fracture

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Introduction: Valve fracture is extremely rare in new generation metallic cardiac valves.

Methods: Six years after mitral and aortic valve replacement due to rheumatic valve disease, a 48-year-old man was admitted with acute pulmonary edema. He had been in good condition until the sudden onset of shortness of breath a few hours before admission. He was not using any medications other than warfarin. Physical examination demonstrated a patient with severe respiratory distress (blood pressure 80/50 mm Hg, temperature 37.2°C), and bilateral pulmonary rales were heard. Heart sounds could not be detected. The ECG demonstrated atrial fibrillation without signs of ischemia. The chest X-ray film showed bilateral pulmonary edema, sternal metallic sutures and mitral valve ring. After intubation and mechanical ventilation, a transesophageal echocardiogram was performed (Video 1, 2), which revealed massive mitral regurgitation due to an unstable mechanical valve. There also was a metallic bileaflet prosthetic valve at the aortic position. The left ventricle was hyperdynamic. The patient was urgently transported to cardiovascular surgery clinic.

Results: On emergency surgery, the mechanical mitral valve prosthesis was found to be broken. The broken valve was explanted and replaced by a 29-mm St. Jude prosthesis. However, the patient remained hypotensive and died soon after the operation.

Conclusion: In late 80s and early 90s, strut fractures were defined in Björk-Shiley convexo-concave valves. However, strut fracture and leaflet escape are extremely rare in new generation valves. The mechanism underlying this fatal complication can be wear and tear effect or manufacturing defect and should be kept in mind in patients who develop sudden pulmonary edema.

Diğer

OS-74

Ekokardiyografide parakardiyak kitle; sağ koroner arterin spontan dev psödoanevrizması

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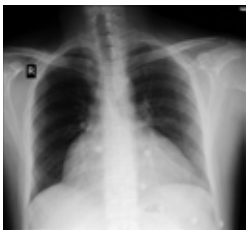
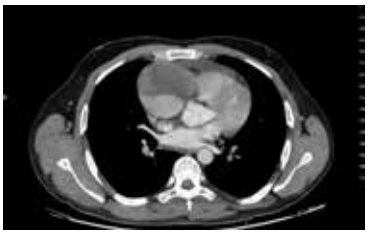
Koroner arter psödoanevrizması, nadir görülen koroner arter patolojilerindendir.. Koroner arter duvarının tüm katlarını içermeyen dilatasyonu olarak tanımlanan psödoanevrizma; çoğunlukla travma, enfeksiyon ve katater ilişkili koroner arterin diseksiyonuna bağlı gelişir. RCA da daha sık görülen koroner arter psödoanevrizması; asemptomatik olabileceği gibi ani ölümlere de yol açabilir. Özellikle büyük boyutlu olanların rüptürü sonucu perikardiyel tamponat gelişme riski mevcuttur. Bunun yanı sıra psödoanevrizmalarda tromboz, distal emboli ve psödoanevrizmaların kardiyak kavitelere fistülizasyonu görülebilir. Dev koroner arter psödoanevrizmaları çok nadir görüldüğü için tedavide belirli bir protokol olmasa da ani rüptür riski olduğu için ortak görüş cerrahi tedavidir. Burada spontan gelişen ve perikardiyel effüzyon ve parakardiyak kitle şeklinde ortaya çıkan sağ koroner arterin dev psödoanevrizması sunulmuştur. Yirmi yedi yaşında erkek hasta polikliğine son bir aydır progresif, batıcı tarzda göğüs ağrısı şikayeti ile başvurdu. Hastanın öyküsünden bir ay önce bu şikayetle başka bir merkeze başvurduğu ve akut perikardit tanısı konularak ibuprofen tedavisi başlandığı öğrenildi. Özgeçmişinde özellik yoktu. Fizik muayenesinde kan basıncı 110/80 mmHg, kalp hızı 110 atım/dk olup kardiyak oskültasyonunda patolojik ses ve üfürüm yoktu. Hastanın elektrokardiyografisinde ise sinüs taşikardisi mevcut olup, yaygın minimal ST elevasyonu izlendi (Şekil 1).

PAAG de kardiyotorasik oranın kalp lehine arttığı görüldü (Şekil 2). Laboratuvar tetkiklerinde ise kardiyak enzimleri yüksek olup; Troponin T 1112 ng/L (0-100), CKMB 97 U/L(3-25), CK 748 U/L (0-190) saptandı. Sedimentasyon hızı 61, CRP ise 75 mg/L idi. Hastanın bir ay önce perikardit tanısı konduğu dönemde yapılan transtorasik ekokardiyografisi (TTE) minimal perikardiyalefüzyon dışında normal sınırlarda idi. Hastanemizde yapılan TTE de perikardiyal efüzyonun dışında sağ atrium komşuluğunda 45x60 mm boyutlarında kaviter kitle saptandı (Video 1). Bunun üzerine hasta koroner yoğun bakım ünitesine yatırıldı. TTE de saptanan parakardiyak kitlenin daha iyi anlaşılması için toraks bilgisayar tomografisi (BT) çekildi. Toraks BT de sağ atrium lateral komşuluğunda RCA ile devamlılık gösteren, sağ atriumubasılayaniçerisinde trombüs materyalleri bulunan yaklaşık 10x7x6 cm boyutunda anevrizma kesesi izlendi (Şekil 2). Dev RCA anevrizması saptanan hastaya cerrahi tedavi düşünüldü. Cerrahi tedavi öncesi yapılan koroner anjiyografide sol koroner sistemin normal olduğu, RCA nın ise dev bir anevrizmatik keseye açıldığı görüldü (Video 2). Bu bulgular ile acil cerrahi operasyona alındı ve trombüslü anevrizmatik kese rezeke edildi. Alınan cerrahi materyalin patolojisi ise koroner psödoanevrima ile uyumlu olduğu şeklinde raporlandı. Operasyon sonrası ek sıkıntı yaşanmayan hasta taburcu edildi.



Şekil 1. Postero-anterior akciğer röntgeni.



Şekil 2. İçerisinde trombüs materyeli izlenen dev sağ koroner arter psödoanevrizması.

OS-75

Self -inserted paper clips in thoracic cavitiy

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Introduction: Self inserted sharp foreign bodies are one of the rare causes of chest traumas. Self-inserted foreign bodies in thoracic cavity may have been undiagnosed due to uncommon use of traumatic methods.

Case Report: 25 year- old male prisoner admitted the our emergency room with a complaint of chest pain which had been started in last few months. He had been smoking one pack of cigarettes a day for 10 years. In his physical examination there were multiple incision scars on the skin of chest wall and both upper extremities. Laboratory parameters and electrocardiographic findings were normal. No etiologic reason was found about chest pain. However when we detailed the history of the patient, we learned that he had self-inserted multiple straightened paper clips into chest wall. Posterior-anterior chest radiograph revealed that there were multiple sharp radiopaque foreign bodies which were located along the border of the left ventricle, in the left lung and under the diaphragm. Transthoracic echocardiography revealed a thin hyperechoic density extending to the interventricular septum from left ventricle lateral wall. Surgery was planned for the patient. However patient was following on oupatient clinic due to refusing to surgical treatment.

Discussion: Self-inserted intra-cardiac and intra thoracic sharp foreign body injuries are seen mainly in young and middle-aged adults suffering from psychiatric disorders, commonly depression, schizophrenia, and substance use disorders. In one-fourth of the cases, it is due to deliberate self-harm. The most common symptom is chest pain.About 70% use a single needle but 30% may use multiple needles. Second attempts are rare. Majority of the patients (85%) are managed by surgery and recover from the injury. The condition has a low mortality rate of 5%. The patient with chest pain who have or may have psychiatric disorder should be examined carefully or if necessary imaging methods should be sought for self-inserted foreign bodies in chest cavity.

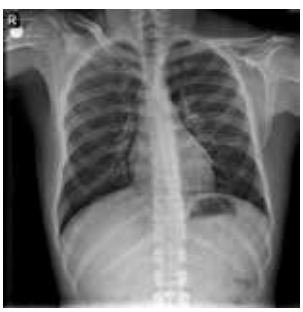

Figure 1. There were multiple sharp radiopaque foreign bodies which were located along the border of the left ventricle, in the left lung and under the diaphragm.

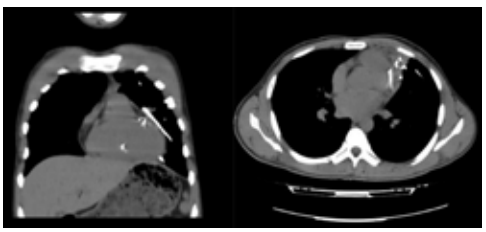

Figure 2. Computed chest tomoghraphy revaled the excact locasitaion of foreign bodies in chest cavity.

OS-76

Hiatal hernia presenting with a big left atrial mass

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Paraesophageal hiatal hernia is described as prolapsus of the stomach through the diaphragmatic esophageal hiatus. These hernias are usually remains asymptomatic and discovered incidentally, albeit a catastrophic complication may present such as bleeding or incarceration. We present a complication of paraesophageal hiatal hernia which was not reported before. A 67-year-old man was admitted with complaints of chest pain ongoing for 15 days. The character of the chest pain was progressive, retro-sternal and burning sensation. The patient was a heavy smoker and had not any other traditional risk factors for coronary artery disease. His physical examination was non-diagnostic. His blood pressure which measured from bilateral upper extremities were 113/62 and 122/61 mmHg. His electrocardiogram was normal. Chest film revealed a suspicious air space under tracheal bifurcation. A fast bed-side transthoracic echocardiography (TTE) was performed to exclude any acute aortic or coronary syndrome. TTE revealed a fixed left atrial mass in 33x34 mm dimensions adjacent to left atrial lateral wall. Although, in order to better reveal the relation between the left atrial mass and left atrium transesophageal echocardiography was used; the patient could not adapt it. Subsequently, contrast enhanced cardiac computed tomography (CT) was performed for confirmation. CT scan revealed a paraesophageal hernia which consisting air gaps and compressing the left atrium in the behind. There was not a filling a defect in the LA. After the definitive diagnosis, proton pump inhibitor treatment was administered and the patient was referred to gastroenterologic surgery department. The patient underwent a surgery of Nissen fundoplication and discharged at 13th day without a further complication.

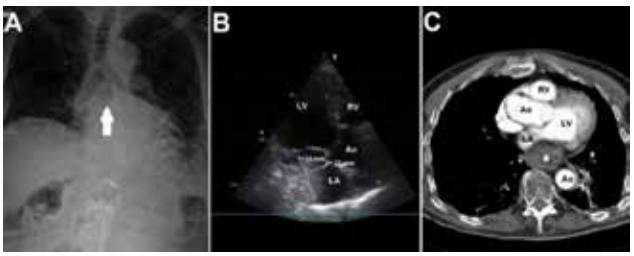

Figure 1. (A) Chest X-ray shows normal mediastinal and cardiothoracic ratios with the suspicious view of air space under tracheal bifurcation (white arrow indicates the air space). **(B)** Transthoracic echocardiogram in modified 4 chamber view shows left atrial mass in dimensions of 33x34 mm adjacent to lateral atrial wall. **(C)** Cardiac computed topographic scan shows that left atrium was compressed by the paraesophageal sliding hernia (white asterisk shows the hernia).

Kardiyak görüntüleme / Ekokardiyografi

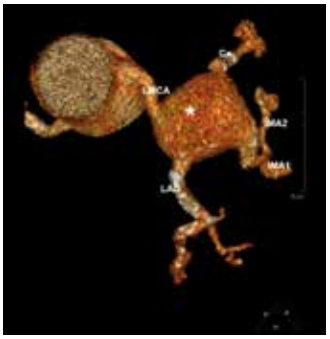

Figure 1. CT coronary angiography imaging of left coronary system after 3D reconstruction and substraction of adjacent tissues. Aneurysm (*), Circumflex artery, Cx; Intermediate artery, IMA; Left anterior descending artery, LAD; Left main coronary artery, LMCA.

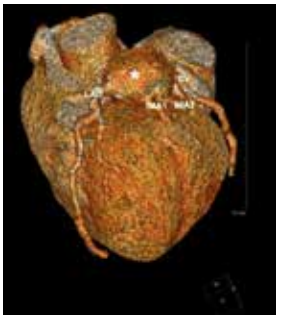

Figure 2. CT coronary angiography imaging of left coronary system with adjacent cardiac anatomic structures after 3D reconstruction. Aneurysm (*), Circumflex artery, Cx; Intermediate artery, IMA; Left anterior descending artery, LAD; Left main coronary artery, LMCA.

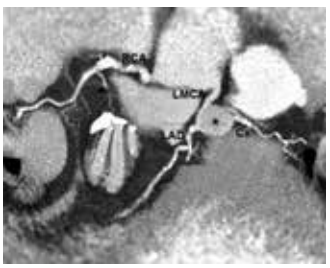

Figure 3. CT coronary angiography imaging of whole coronary system including aneurysm sac. Diffuse calcific plaque formation causing mild to moderate stenosis in all arteries are evident. Aneurysm (*), Circumflex artery, Cx; Intermediate artery, IMA; Left anterior descending artery, LAD; Left main coronary artery, LMCA, Right coronary artery, RCA.

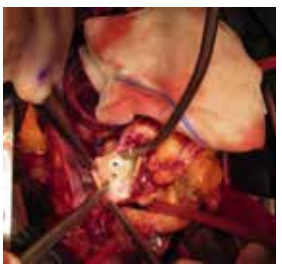

Figure 4. Peri-operative appearence of aneurysm sac. Sac was dissected and coronary ostia were exposed. Aneurysm (*).

OS-77

Huge left main coronary aneurysm evaluated with multimodality imaging

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A 44-year-old male patient on dialysis who was a candidate for renal transplantation, attended to our out-patient clinic for cardiac evaluation. He was obese and complaining about exertional dyspnea even while performing routine daily activities. Cardiac physical examination and baseline electrocardiography (ECG) was normal. Echocardiographic examination revealed normal ejection fraction, moderate left ventricular hypertrophy and mild diastolic dysfunction without significant valvular disease. Eventually, coronary angiography was scheduled due to abnormal treadmill stress test results. A giant aneurysm measured approximately 4 cm was observed at left coronary injections. The neck of the aneurysm was seemed to originate from distal left main coronary artery (LMCA), but exact anatomy could not be identified in serial injections from various angles. Moreover, left anterior descending artery (LAD) could not be clearly opacified (Video 1 and 2). Regarding the necessity of intervention to giant aneurysm which had led to ischemic symptoms and had the state of impending rupture, coronary computed tomography angiography (CCTA) was planned to evaluate the left coronary tree precisely and come to a decision about treatment strategy. ECG-gated CT scan (Step & Shoot Cardiac acquisitions on a 256-slice multi-detector CT scanner, Philips Brilliance iCT, Philips Healthcare, The Netherlands).was performed the day after and images obtained with multiplanar reformation and three-dimensional volume-rendering were assessed for definite diagnosis (Fig 1 and 2). Aneurysm sac involving scattered calcifications was measured 41x32x32 mm and no image consistent with thrombus formation was observed. It was located at distal LMCA as predicted. Aside from that LAD, circumflex artery (Cx), two intermediate arteries (IMA 1 and 2) arising from LMCA and dominant ectatic right coronary artery (RCA) were explicitly visualized, and diffuse calcific atherosclerotic plaques resulting in mild to moderate stenosis in entire coronary system were displayed (Fig 3). At the operation room, aneurysmatic segment was exposed and disected (Fig 4). Coronary ostia were seperately sealed, sac was shrinked by over and over suturing and bypass grefting to each branch was performed thereafter. Patient was discharged uneventfully at the end of first week.

OS-78

Rivaroksaban kullanan hastada nadir bir komplikasyon: Alveolar hemoraji

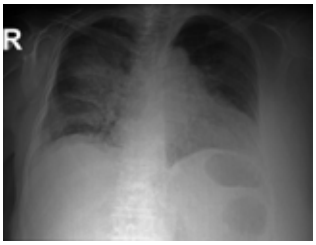
Salih Kılıç, Gülin Alkan

Nizip Devlet Hastanesi, Gaziantep

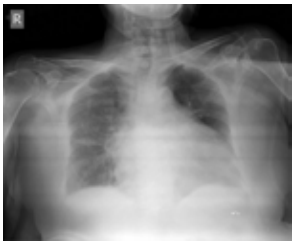
Giriş: Non valvüler atriyal fibrilasyonda (NVAF) kullanılmaya başlayan yine jenerasyon oral antikoagülanların (YOAC) kanama açısından varfarine göre daha güvenli olduğu gösterilmiş olsa da kanama komplikasyonları görülebilmektedir. Bu olgumuzda nadir bir kanama komplikasyonu olarak alveolar hemoraji (AH) gelişen bir vaka sunulmuştur.

Olgu Sunumu: 89 yaşında DM,HT ve NVAF öyküsü olan kadın hastaya 6 ay önce rivaroksaban 20 mg/gün başlandı. Hasta öksürük, nefes darlığı ve hemoptizi yakınması ile başvurdu. Genel durumu orta olan hastanın ateşi 37 derece, solunum sayısı 25/dk, tansiyon 137/77 mmHg ve kalp hızı 114 atım/dk saptandı. Sistemik bakısı, sağ akciğer alt ve orta zonda inspiratuvar ral dışında normal idi. Kan gazında pH 7.39 PCO2: 31, PO2: 47, SaO2: %78 ve laboratuvarında hemoglobin 7.9 mg/dl, trombosit 315.000, lökosit 10450, INR 1.3 kreatin 2.3 ve eGFR 28ml/dk saptandı. Akciğer grafisinde (PAAG) sağ alt orta zonda yaygın dansite artışı saptanan hasta hipoksi nedeniyle non invaziv mekanik ventilatör (NİMV) desteğine alındı (Şekil 1). Akut böbrek yetmezliği nedeniyle rivaroksaban kesildi. Uygun doz sıvı ve iki ünite eritrosit süspansiyonu verildi. AH ön tanısıyla çekilen toraks BT' de sağ perihiler bölgede buzlu cam görünümü saptandı ve AH dışlanamadı. Takipte NIMV ihtiyacı kalmadı ve satürasyonu %95'in üzerinde seyretti. Kontrol PAAG'de anlamlı düzelme saptandı (Şekil 2–5). Böbrek fonksiyonları normale döndü. Diz ağrıları nedeniyle yoğun analjezik aldığı öğrenildi. YOAC hasta kabul etmeyen hastaya INR 2-3 arasında olacak şekilde varfarin başlandı. Stabil olan hasta taburcu edildi.

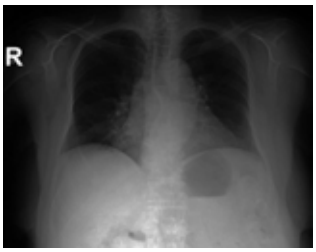
Sonuç: YOAC'ların, kullanımı hızla artmaktadır. Antikoagülan kullananlarda çeşitli kanama komplikasyonu izlenebilmektedir ancak AH çok nadir görülmektedir. AH çeşitli etiyolojik nedenlerden kaynaklı, akciğer alveolokapiller membranda hasar sonucu olan nefes darlığı, anemi ve hemoptizi ile seyreden bir hastalıktır. Daha çok immün sistem hastalıklarına sekonder olan AH' nın nadir sebepleri arasında olgumuzda olduğu gibi antikoagülan kullanımı bulunmaktadır. AH tanısında en önemli basamak klinik şüphedir ve tedavi olarak destek tedavisidir. Benzer bir şekilde YOAC alanlarda kanama durumunda destek tedavisi önerilmektedir. Her ne kadar YOAC alan hastalar gıda ve ilaç etkileşimi açısından varfarine göre daha üstün olsalar da bu ilaçlar reçete edilirken hastanın olgumuzda olduğu gibi böbrek fonksiyonlarına etki edebileceği ilaçlardan veya durumlar konusunda uyarılması önemlidir.



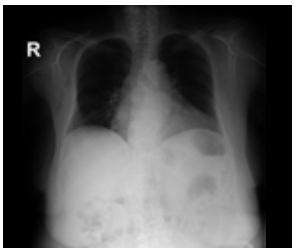
Şekil 1. Yatış.



Şekil 2. İkinci gün.



Şekil 3. Onuncu gün.



Şekil 4. Birinci ay.

OS-79

Methemoglobinemia after prilocain injection for implantation of implantable cardioverter-defibrillator

Çağın Mustafa Üreyen[1], Sibel Üreyen[2], Görkem Kuş[1], Yunus Cem Baş[1], Murat Esin[1], Emre Asiltürk[1], Özkan Kayhan[1], Gamze Korkmaz[1], Seren Ay[1], İsa Öner Yüksel[1], Göksel Çağırcı[1], Şakir Arslan[1]

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[2]*Department of Internal Medicine, Sakarya Training and Research Hospital, Sakarya*

A 42-year-old male patient with dilated cardiomyopathy was hospitalized to be implanted an ICD. Ejection fraction og LV was 25% and the patient was symptomatic (NYHA II-III) despite an optimal heart failure treatment. Prilocain was used as a local anesthetic and 20 ml was initially administered. However, the patient described severe pain during generation of the pouch for the battery, therefore, 20 ml of prilocain was again injected into the subcutaneous and muscular tissue. After implantation of the RV lead, the patient became agitated. His agitation was increased after he described visual loss. The battery of ICD was quickly placed into the preformed pouch and subcutaneous and cutenous tissue was sutured rapidly and a neurology consultation was requested. After the neurologic examination, a cranial CT was recommended. However, a slight peripheral cyanosis of the fingers was ascertained which later gradually increased and the oxygen saturation was found to be 82%. After these findings, methemoglobinemia was suspected and arterial blood gas analysis was performed. The level of methemoglobinemia was 21%. The patient was transferred to intensive care unit and methylene blue of 50 ml was administered in 5 minutes. After 1 hour, the symptoms of the patient was completely disappeared and the level of methemoglobinemia was 1.5%. Methemoglobinemia results from the presence of iron in the ferric form instead of the usual ferrous form. This leads to a shift of oxygen–hemoglobin dissociation curve to the left that results in an overall reduced ability of the red blood cell to release oxygen to tissues. Symptoms are proportional to the methemoglobin level and include skin color changes at levels up to 15%. As levels rise above 15%, neurologic and cardiac symptoms arise as a consequence of hypoxia. Levels higher than 70% are usually fatal. IV methylene blue is the first-line treatment of methemoglobinemia. Methylene blue is given in a dose of 1-2 mg/kg up to a total of 50 mg in adults. It may be repeated at 1 mg/kg every 30 minutes when necessary to control symptoms provided that the dose should not exceed 7 mg/kg. Exchange transfusion and hyperbaric oxygen treatment are second-line options for patients with intractable methemoglobinemia. In our cardiology practice, we do not need to use high doses of local anesthetics except cardiac implantable electronic device implantation. Therefore, we are not very familiar with methemoglobinemia. However, we should be aware of its complications and treatment. We deem that our case is an instructive and remarkable example of methemoglobinemia due to prilocain.

OS-80

Kritik ağır hastalarda kötü prognozu öngördüren nadir EKG bulgusu - "Spiked Helmet"

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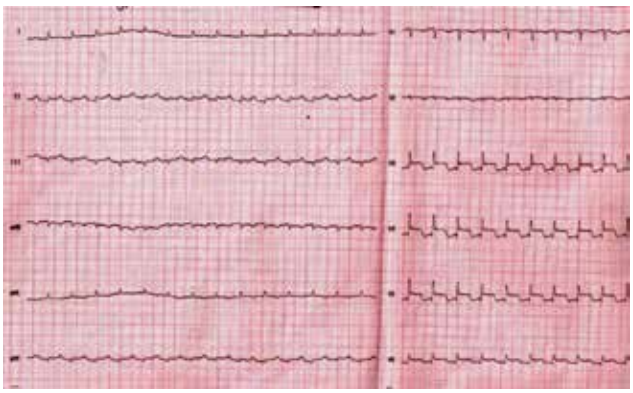
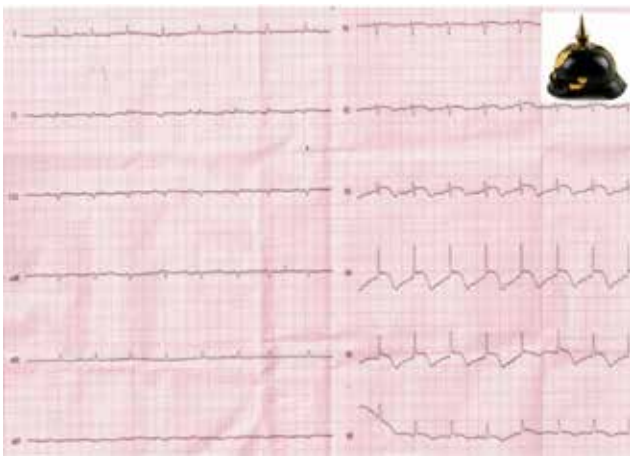
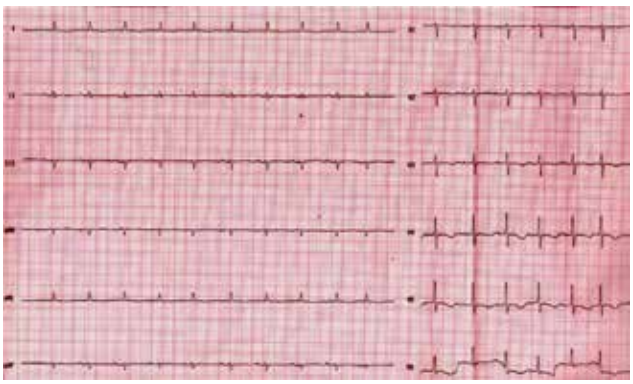
Spiked Helmet" işareti (SHİ) - nonkardiyak kritik ağır hastalarda artmış mortaliteyi gösteren, yakın zamanda tanımlanmış, ender rastlanan elektrokardiyografi (EKG) bulgusudur. Bugüne kadar literatürde yalnız kısıtlı sayıda kritik ağır hastada bildirilmiş olan bulgu, PR ve ST segmentlerinin elevasyonu ve bu ikisi arasındaki QRS kompleksi ile beraber alman askerlerinin kaskını anımsattığı için bu şekilde isimlendirilmiştir. Burada son evre böbrek yetersizliği (SEBY) olan ve sepsis sonucunda SHİ gelişimi ve rezolüsyonu dokümente edilmiş, aynı zamanda Takotsubo kardiyomiyopatisi (TK) şüphesi olan vakayı takdim edeceğiz. Sekiz yıl önce prostat kanseri tanısı konulan, SEBY ile takip edilen, gastrointestinal kanama nedeniyle yoğun bakım ünitesine yatırılan 72 yaşlı erkek hastada yatışın 2.gününde üşüme, titreme, ateş ve gastrik ağrı yakınmaları gelişmiştir. Laboratuar tetkiklerde lökositoz (19.2x10^9/L), CRP (43 mg/dL) ve prokalsitonin yüksekliği (10.4 ng/ml) saptanan hastada sepsis tanısı ile antibiyotik tedavisine başlanmıştır. Monitörde EKG değişikliği izlenen hastanın 12 derivasyonlu EKG-de yaygın ST elevasyonu, V1 ve aVR derivasyonlarında ST depresyonu ve QT uzaması tespit edilmiştir (Şekil 1). Transtorasik ekokardiyografide (TTE) sol ventrikül mid ve apikal segmentleri akinetik, bazal segmentler hiperkinetik, ejeksiyon fraksiyonu %35 izlenen hastada ayırıcı tanıda anterior miyokard enfarktüsü veya TK düşünüldü ve komorbiditeleri nedeniyle medikal tedavi kararı verildi. Troponin düzeyinde hafif yükselme saptandı. Potasyum düzeyinde sapma olmadı. 1 gün sonra çekilen EKG-de özellikle V4-V5 derivasyonlarında SHİ gelişimi farkedildi (Şekil 2). Sepsisin kontrol altına alınmasıyla beraber 3 gün sonrakı EKG-de SHİ geriledi ve prekordiyal derivasyonlarda T dalğa negatifliği izlendi (Şekil 3). Bir hafta sonraki kontrol TTE-de sol ventrikül sistolik fonksiyonlarında belirgin düzelme tespit edildi. Klinik iyileşme izlenen hasta 5 gün sonra tekrarlayan sepsis nedeniyle kaybedildi. SHİ - yeni tanımlanmış, şimdilik yeteri kadar bilinmeyen ve artmış mortalitenin prediktörü olan EKG bulgusudur. Tıbbi literatürde SHİ'li olgu sayısı 10 civarındadır. Çok az bildirilmiş olması bulgunun yeterince bilinmemesi ile ilgili olabilir. İntraabdominal veya intratorasik basınçta ani artış, sol ventrikül inferior duvarının diyaframla direk uyarılması, alkaloz durumunda diyaframın kalple senkron kontraksiyonu. diyafram solunumu bu EKG bulgusunu açıklamak için one sürülen mekanizmalardır. Her ne kadar koroner anjiyografi yapılmamış olsa da sol ventrikül sistolik disfonksiyonunun bir haftada tamamen gerilemesi TK ihtimalini akla getirmektedir. Sepsisli hastaların önemli bölümündü TK-nin gelişimi ve SHİ'nin de sadece kritik ağır hastalarda bildirilmiş olması SH'nin TK'nın doğal seyrinin bir parçası olabileceğini düşündürmektedir. Zamanında gerekli tedavi uygulanmadığında letal sonucun habercisi olması nedeniyle kardiyolog ve yoğun bakım uzmanları tarafından bu bulgunun tanınması önemlidir.



Şekil 1. V1 ve aVR derivasyonlarında ST segment depresyonu, diğer derivasyonlarda ST segment elevasyonu, QT uzaması.



Şekil 2. Özellikle V4-V5 derivasyonlarında daha belirgin olmakla "Spiked Helmet" işareti Sağ üst köşede benzerlik nedeniyle EKG işaretinin ismini aldığı kask.



Şekil 3. "Spiked Helmet" işareti gerilemiş, prekordiyal derivasyonlarda T negatifliği ve QT uzaması izlenmekte.

OS-81

The relationship between asymmetric dimethyl arginine (ADMA) and exercise parameters in type 2 diabetic patients with positive exercise test

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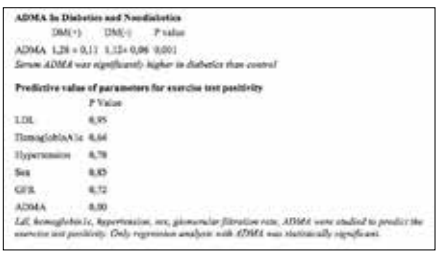
Introduction: We investigated the relationship between asymmetric dimethylarginine (ADMA), endogenous nitric oxide synthase inhibitor, and exercise parameters in type 2 diabetic patients with positive exercise test.

Patients and Methods: The study included 80 diabetic patients (mean age: 59.59 years range between 38 and 76 years). 60 patients were positive and 20 patients were negative according to exercise test. Plasma ADMA level was measured. The correlation between plasma ADMA levels and total duration of exercise, resting and maximal exercise induced heart rate, resting and maximal exercise induced systolic and diastolic blood pressure, the magnitude of ST segment depression were investigated.

Results: ADMA levels increased significantly in diabetic patients with positive exercise test (1.28+0.11 mmol/l, 1.12+0.06 mmol/l). The correlation between plasma ADMA levels and the magnitude of ST segment depression and resting heart rate were found to be statistically significant.

Conclusion: Asymetric dimethylarginine increased significantly in type 2 diabetic patients with positive exercise test. This finding suggest it can be used as a novel biomarker to show endothelial dysfunction. It correlates strongly with exercise parameters. It can be an indirect measurement of exercise response. Further investigaions needed.



ADMA In Diabetes and Nondiabetics

	DM(+)	DM(-)	P value
ADMA	1,28 ± 0,11	1,12± 0,06	0,001

Serum ADMA was significantly higher in diabetics than control

Predictive value of parameters for exercise test positivity

	P Value
LDL	0,95
Hemoglobin A1c	0,66
Hypertension	0,78
Sex	0,85
GFR	0,72
ADMA	0,00

LdL hemoglobin1c, hypertension, sex, glomerular filtration rate, ADMA were studied to predict the exercise test positivity. Only regression analysis with ADMA was statistically significant.

OS-82

El yapımı kement (Snare)

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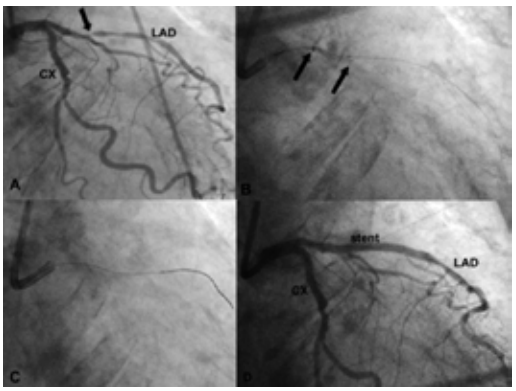
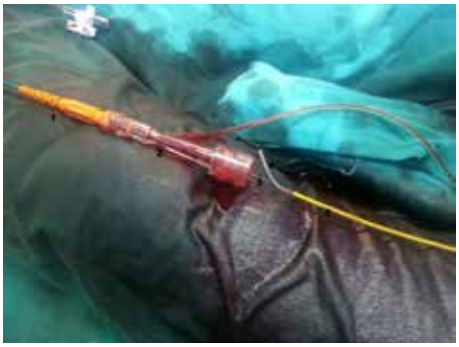
Yetmiş yaşında kadın hasta akut koroner sendrom kliniğiyle acile başvurdu. Hastaya koroner anjiyografi planlandı. Yapılan koroner anjiyografi sonucunda sol ön inen arterde ciddi lezyon saptandı (Şekil 1a). Hastanın mevcut kliniği ve lezyon ciddiyeti nedeniyle ilgili damara perkütan koroner girişim kararı verildi. Lezyon 2.0x20 balon ile genişletildi, ardından 3.0x32 ilaç kaplı stent gönderildi. Stent lezyon seviyesinden geçmedi ve kateter içine geri alınırken sıyrılarak sol ön inen arter içinde açılmamış halde kaldı (Şekil 1b). Birkaç kez elde bulunan kullanılmış kement kateteri ile stenti geri alma denendi, ancak başarısız oldu. Ardından 4 farklı telle tel sarma yöntemi denendi, yine başarısız oldu. Kement kateteri ile olan başarısızlık özellikle malzemenin kullanılmışlığına bağlı dışındaki plastik kılıfın deformasyonuna bağlandı. Elde kullanılmamış kement kateteri bulunmamaktaydı; bunun üzerine kement teli kılıftan çıkarıldı, elde bulunan 6 french guideliner içine yerleştirildi, dolayısıyla guideliner kullanılarak deforme olmuş eski kementten yeni bir kement oluşturuldu. Tek seferde başarıyla sıyrılmış stent geri alındı ve sorunsuzca dışarıya çıkarıldı (Şekil 1c, 2, 3). Mevcut lezyon guideliner desteğiyle yeni bir 3.0x32 ilaç kaplı stent kullanılarak açıldı (Şekil 1d). Hasta sorunsuzca yoğun bakım ünitesine alındı, takiplerinde herhangi bir sorun yaşanmadı ve hasta olaysız şekilde taburcu edildi. Özellikle eldeki malzemenin yetmediği ya da sorunu çözmediği durumlarda bilimsel sınırlar içinde kalınarak bu tür pratik çözümler üretilebilir.



Şekil 1. (A) Siyah ok sol ön inen arterdeki ciddi lezyonu gösteriyor. **(B)** Siyah oklar sol ön inen arterde sıyrılıp kalan açılmamış stenti gösteriyor. **(C)** El yapımı kement kateterle alındıktan sonra sol ön inen arter. **(D)** Stent takıldıktan sonraki görüntü.



Şekil 2. El yapımı kement kateterle geri alındıktan sonra stent görüntüsü. 1. 7F kılavuz kateter 2. Y konnektör 3. Kılavuz tel 4. Sıyrılmış stent 5. Guideliner.

Şekil 3. Guideliner içinden geçirilmiş kement kateter teliyle yakalanmış stent. Siyah ok kementi gösteriyor. 1. Guideliner 2. Stent.

OS-83

Akut inferior MI taklit eden özefagus rüptürü, mediastinit

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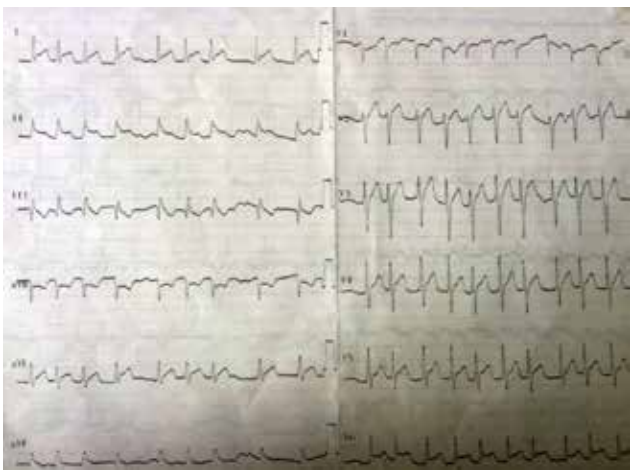
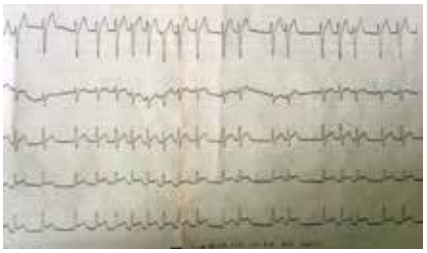
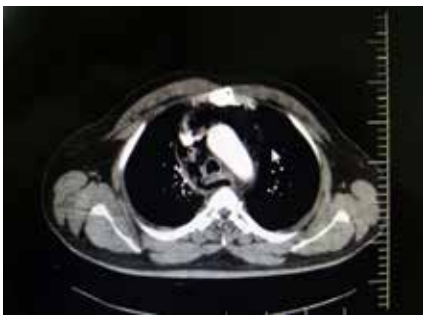
Elli altı yaşında hipertansif, sigara içicisi erkek hasta, göğüs ağrısı şikayeti ile dış merkeze başvurmuş, sorasında akut inferior miyokart enfarktüsü ön tanısıyla, primer anjiyoplasti yapılması için kliniğimize sevk edilmiş. Anemnezinde bir haftadır aralıklarla devam eden atipik bir göğüs ağrısı tarifliyordu ve yemek esnasında zorlu bir yutkunma sonrasında başladığını belirtti. Hastanın EKG si normal sinüs ritmi ve D2-3, AVf de D1 AVL ve V5-6 da 2 mm ST elevasyonu ve Paroksismal AF geçişleri izlendi. Kardiyak enzim ve markerları pozitifti. Ekokardiyografisinde inferior duvarda şüpheli bir segmenter hareket bozukluğu ve hafif düzeyde kapak hastalıkları mevcuttu. Hastaya koroner anjiyografi uygulandığında koroner arterlerin tamamen normal olduğu izlendi. Hastanın ağrısının devam etmesi ve hipotansiyon gelişmesi üzerine Aort diseksiyonu ön tanısı ile kontrastlı thoraks tomografisi çekildi. Kontrastlı tomografide özefagus rüptürü ve rüptüre bağlı mediastinal serbest hava ve sıvı izlendi. Hasta genel cerrahi bölümüne devredildi, devir sonrası hasta anestezi yoğun bakım takipleri esnasında eks oldu. Daha önce litaratürde bildirilmiş spontan özefagus rüptürüne sekonder gelişen perikardit ve göğüs ağrısı vakaları olmuştur. Özefagus rüptürü az görülen ve tanı konulmakta gecikilen bir durumdur. Tanı geçikirse hastalarda mediastinite bağlı sepsis ve septik şok tablosu ortaya çıkmaktadır. Göğüs ağrısı ayırıcı tanısının yapılamadığı durumlarda, zorlu yutkunma, kusma, öğürme gibi durumlarda anamnez ayrıntılı bir şekilde yapılmalıdır. Bu vakada göğüs ağrısı ayırıcı tanısı yapılamayan durumlarda, hastaların özefagus rüptürü açısındanda değerlendirilmesi gerekliliği vurgulanmıştır.



Şekil 1. İnferolateral St elevasyonu, AKS, perikardit.



Şekil 2. PAF atakları.



Şekil 3. Thoraks bt özefagus rüptürü.

OS-84

Asemptomatik sol ventrikül tutulumlu aritmojenik sağ ventrikül displazisi (ARVD) vakası

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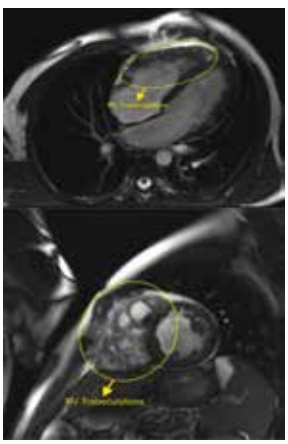
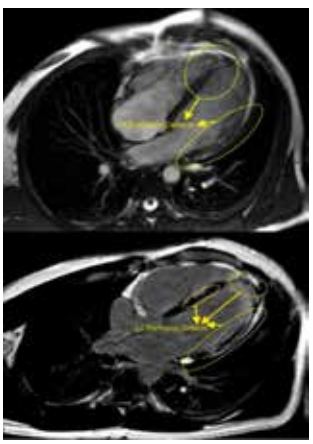
On sekiz yaşında asemptomatik erkek hasta, 17 ve 18 yaşlarında iki kardeşini ani ölüm ve 47 yaşında annesini kalp yetmezliği zemininde ani kardiyak ölüm sebebiyle kaybetmesi üzerine kontrol için polikliniğimize başvurdu. Kardiyak muayenesinde patolojik bulgu saptanmadı. Elektrokardiyografi (EKG)'sinde V1-3 de geniş QRS ve T negatifliği ile epsilon dalgası izlenmedi. Ekokardiyografide, sağ ventrikülde ileri derecede genişleme, lokalize sağ ventrikül anevrizmaları, sağ ventrikülde belirgin kontraktil disfonksiyon ve trabekülasyon saptandı. Bunun yanında sol ventrikülde miyokard kasılmasında azalma gözlendi ve sol ventrikül ejeksiyon fraksiyonu %40 hesaplandı. Kardiyak magnetik rezonans görüntülemede (MRG), sağ ventrikül dilatasyonu, lateral duvarda diskinezi ve sağ ventrikül miyokardında fibrolipomatöz değişiklikler izlendi. Ayrıca sol ventrikülinferolateral, septum ve apikal duvarda diskinezi ve fibrozis ile uyumlu geç dönem opaklanması saptandı. Multislice CT koroner anjiyografisinde koroner arter anatomisi normal olarak belirlendi. Hastaya primer korunma için ICD implantasyonu yapılarak kronik kalp yetersizliğine yönelik; metoprolol 50 mg 1x1, ramipril 2.5 mg 1x1, spirinolakton 25 mg 1x1 medikal tedavi ile taburcu edildi. Son zamanlarda sol ventrikül tutulumuyla giden veya sol ventrikül tutulumunun ön planda olduğu aritmojenik kardiyomiyopatiler daha iyi tanımlanmaya başlanmıştır. Histopatolojik çalışmalar çift ventrikül tutulumunun hastaların %75'ine yakın kısmında bulunduğunu göstermektedir (McKenna WJ, Thiene G, Nava A, et al. Diagnosis of Arrhythmogenic rightventricular displasia/ cardiomyopathy. BrHeart J 1994; 71:215-18). Bu hastalık için klasik tanımlamanın yetersiz kaldığı, yukarıda da bahsedilen tanı kriterlerinin sol taraf tutulumu olan formları tam olarak kapsamadığı görülmektedir. Bizim hastamızda da ekokardiyografik olarak sağ ventrikül yanında sol ventrikülde segmenter duvar kasılma kusuru ve sistolik fonksiyonlarda azalma saptandı. Kardiyak MR görüntülerde de sol ventrikül inferolateral, septumda ve apikal duvarda diskinezi ve fibrozis ile uyumlu bulgular saptandı.



Şekil 1. Magnetik rezonans görüntülemede sağ ventrikül artmış trabekülasyonu izlenmektedir.



Şekil 2. Magnetik rezonans görüntülemede sol ventrikül perfüzyon defektleri izlenmektedir.

OS-85

Sağ ventrikülde yaygın st elevasyonu oluşturan kitle

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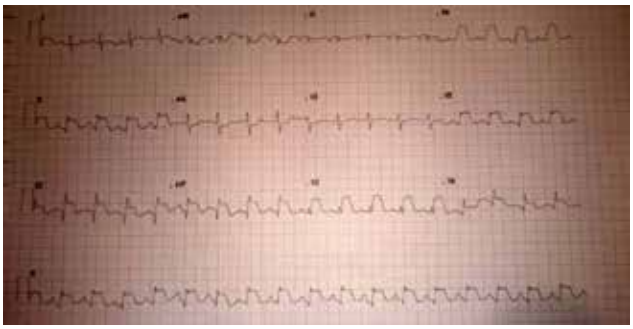
Altmış iki yaşında erkek hasta 1 yıl önce yassı hücreli paratiroid kanseri sebebiyle paratiroidektomi olmuş. kemoterapi süreci içinde kardiyoloji kontrolüne geldi. Fizik muayenede anormal bulgu izlenmedi. EKG de inferiyor ve anteriyor derivasyonlarda yaygın ST elevasyonu mevcuttu (Şekil 1). Hasta acil KAG labaratuvarına alındı. LAD orta bölgede %70 darlık izlendi RCA ve Cx normal idi. Hastaya transtorasik ekokardiyografi yapıldı. Sağ ventrikülün 3/4'ünü dolduran vantrikül duvarları ile aynı dansitede kitle imajı izlendi (Video 1, 2). Hastanın eski paratiroid kanserine yönelik araştırma yapıldı. Sağ ventrikülündeki kitle imajından biyopsi yapıldı biyopsi sonucu normal miyokard ve yağ hücreleri olarak raporlandı. Kardiyak selim tümör düşünüldü. Hastanın aktif şikayetinin olmaması üzerine takip kararı verildi. 3 ay sonraki EKG de ST elevasyonları aynı şekilde devam etmekte idi. Literatürde sekonder kardiyak tümör ve metastazların sinsü bradikardisi, atriyal fibrilasyon, ST-T değişiklikleri yaptığına dair yayınlar mevcuttur. Vakamızda hem inferiyor hem anteriyor derivasyonlarda ST elevasyonu yaparak kendini gösteren Sağ ventrikül kaynaklı bir kitle imajı (kardiyak tümör?) mevcuttur. Hem Ekokardiyografi görüntüsü hem de EKG değişiklikleri açısından ilginç vaka olduğunu düşündük.



Şekil 1. İnferiyor ve prekordiyal deviasyonlarda yaygın ST elevasyonu görülmektedir.

OS-86

Her üç koroner arterde örgü ("woven") anomalisi

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Örgü (woven) koroner arter (ÖKA), çok nadir rastlanan konjenital malformasyon olup koroner arterlerin (KA) distalde tekrar birleşmek üzere ince kanallara ayrışması ile karakterizedir. İntrakoroner trombüs veya kronik diseksiyonla karışabilen bu anomali daha çok sağ koroner arterde (RCA) bildirilmiştir. Burada, bildiğimiz kadarıyla literatürde daha önce sadece tek bir olguda bildirilmiş olan, her üç KA-da ÖKA olan olguyu sunacağız. Anamnezinde hipertansiyon ve sigara kullanımı öyküsü olan, herhangi ilaç kullanmayan 62 yaşında erkek hasta, son 1 ayda gelişen efor dispnesi şikayetiyle başvurdu. Fizik muayenesi normal olan hastanın elektrokardiyografisi (EKG) ve ekokardiyografisinde (EKO) değişiklik saptanmadı. Stres EKG testinde dispne şikayeti ve 3. evresinde lateral derivasyonlarda 2 mm ST segment depresyonu gelişti. EKG değişikliği toparlanma evresi 1. dakikada geriledi. Koroner anjiyografide RCA orta segmentinde daha belirgin olmakla (Video 1), sol anterior desendan arter (LAD) orta segmenti ve birinci ve ikinci diyagonal dallarında ve sirkumfleks arter (Cx) orta segmentinde ÖKA saptandı (Video 2, 3). Medikamentoz tedavi (beta bloker ve uzun etkili nitrat) ile takip kararı verilen hastanın şikayetlerinde belirgin gerileme izlendi. ÖKA - koroner arterin distal lümende tekrar birleşmek üzere ince kanallara ayrışması ile karakterize nadir malformasyondur. Daha çok RCA-da olsa da, diğer koroner arterlerde tekbaşına veya RCA ile birlikte ÖKA-lı vakalar bildirilmiştir. Literatürde bugüne kadar her üç KA-da ÖKA varlığı bildiğimiz kadarıyla sadece tek bir vakada bildirilmiş olup, vakamız bu yönüle özellik arz etmektedir. Genellikle insidental saptanan bu anomali iyi huylu olup, hastalar 4-5 yıllık takipleri boyunca asemptomatik oldukları bildirilmiştir. Bununla birlikte ÖKA ince kanallarının koroner tromboza yatkınlık oluşturarak, akut koroner sendroma yol açtığı ender olgular da mevcuttur. ÖKA-nın tanınması ve koroner dolum defektine neden olan trombüs ve diseksiyon gibi durumlardan ayrımı tedavi kararları bakımından önemlidir. ÖKA-nın bilinmemesi hastada komplikasyon riski yüksek gereksiz koroner işlemler uygulanmasına neden olabilir. İntravasküler ultrasonografi ve optik koherans tomografi ile daha net ayırım yapılabilse de distal lumen akımının alışılmadık derecede normal olması ÖKA lehine değerlendirilen bir bulgudur. Özellikle birden fazla damarda ÖKA varlığı ayırıcı tanıyı zorlaştırabilir. ÖKA ender rastlanan bir anomali olsa da, girişimsel kardiyologların günlük pratiklerinde karşılabilecekleri ve akılda tutulması gereken bir durumdur. Özellikle birden fazla damarda olması durumunda tanı ve tedavisi zor olabilir. Medikamentöz tedaviye yeterli yanıt alınmayan hastalarda cerrahi ve ya perkütan revaskülarizasyon düşünülebilir.

OS-87

Hipertrofik kardiyomiyopatili hastada üç farklı koroner arterde miyokardiyal köprüleşme

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Koroner arterlerin doğumsal anomalileri arasında olan miyokardiyal köprüleşme (MK);epikardiyal koroner arterin belirli bir bölümünün miyokardın içinde seyretmesi ile karakterize bir durumdur. Anjiografik görülme sıklığı %1.5 ile %16 arasında değişmektedir. Hipertrofik kardiyomiyopatili (HKM) hastalarda ise bu oran daha fazladır ve %30'lara varmaktadır. MK sıklıkla tek koroner arterde ve LAD nin gövde bölgesinde izlenmektedir. Bu olguda daha nadir görülen birden çok koroner arterde miyokardiyal köprüleşmenin olduğu genç HKM' li bir hasta sunulmuştur. Yirmi iki yaşında erkek hasta polikliniğimize başvurudan bir hafta önce efor sonrasında oluşan atipik göğüs ağrısı ve göz kararması ile dış merkeze başvurmuş. Dış merkez incelemede hastaya kalp duvarında kalınlaşma olduğu ve efor yapmaması gerektiği söylenmiş. Hastanın atipik göğüs ağrılarının aralıklı devam etmesi üzerine polikliniğimize başvurdu. Özgeçmişinde sigara dışında özellik yoktu. Fizik muayenede kan basıncı 120/70 mmHg, kalp hızı 90 atım/dk olup, kardiyak oskültasyonda sternum sol yanında 2. dereceden sistolik üfürüm mevcuttu. Elektrokardiyografisinde ise prekordiyal derivasyonlarda T dalga negatifliği ve ST depresyonu ile sol ventrikül hipertrofisi kriterleri izlendi. Sonrasında yapılan transtorasik ekokardiyografisinde (TTE) ise sol ventrikül sistolik fonksiyonları, çapları normal ve belirgin kapak patolojisi yoktu. Ancak izole olarak interventriküler septum kalınlığı abartılı artmış olup 23 mm saptandı (Video 1). TTE de ayrıca istirahatte ve provokasyon ile sol ventrikül çıkım yolunda basınç farkı izlenmedi. Nonobstrüktif HKM tanısı konan hastaya, atipik göğüs ağrılarının devamlılık göstermesi nedeniyle koroner anomaliyi dışlamak adına koroner anjiografi (KAG) planlandı. KAG de üç farklı damarda MK saptandı. PDA da daha ciddi olmak üzere LAD gövde ve İntermediate arterde miyokardiyal köprüleşme izlendi (Video 2, Video 3). Beta bloker tedavisi başlanıp genel önlemler anlatılan ve aile taraması önerilen hasta poliklinik takibine alınarak taburcu edildi. Sıklıkla benign bir durum olan MK bazen de angina, aritmi, sol ventrikül fonksiyonlarında azalma ve ani ölüme neden olabilir. Hemen her zaman LAD de görülen miyokardiyal köprüleşme; sistol sırasında koroner arterde oluşan kompresyona bağlı dinamik daralma ile sonuçlanır. HKM li hastalarda normal popülasyona göre daha sık görülür. Medikal tedaviye rağmen ciddi iskemi varlığında; perkütan girişim, miyotomi ve koroner bypass cerrahisi gibi invaziv tedaviler düşünülür. Litaratürde miyokardiyal köprüleşme ile hipertrofik kardiyomiyopatinin ilişkisi gösterilmişse de, üç büyük farklı koroner arterde miyokardiyal köprüleşmesi olan HKM li olgu çok nadir bildirilmiştir.

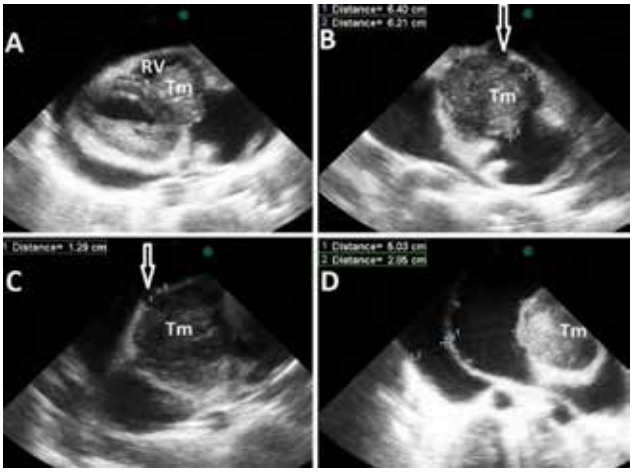

Figure 1. (A) Echocardiogram shows that the mass arising from interventricular septum and narrowing right ventricle outflow. **(B)** Giant mass in 6.4x6.22 cm dimensions and right ventricular free wall erosion. **(C)** Right ventricular free wall ruptured segment in 1.29 cm dimension. **(D)** Massive pericardial (5.03 cm) and pleural (2.75 cm) fluid.

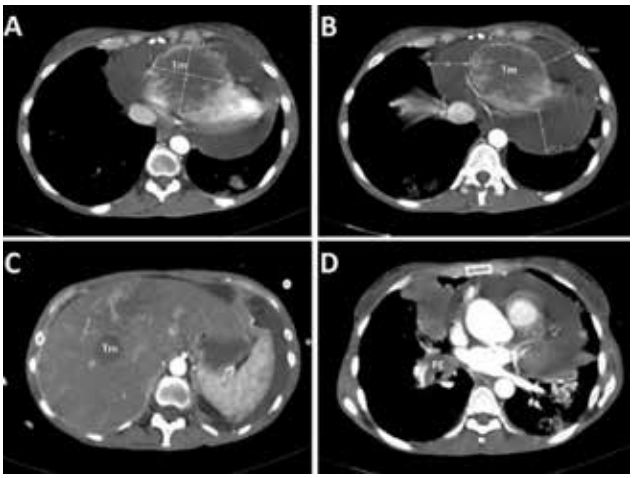

Figure 2. (A) Computed tomographic scan shows the giant mass in 6.26x6.85 dimensions filling the right ventricle. **(B)** Massive pericardial fluid. **(C)** Metastases in the liver. **(D)** Pulmonary tumor embolism.

OS-88

Cervical adenocarcinoma presenting with a giant cardiac mass, cardiac tamponade and pulmonary embolism

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Cardiac metastasis as a consequence of cervical cancer is extremely rare. We report a case of cervical adenocarcinoma presenting with giant right ventricle (RV) metastasis and pericardial tamponade concomitant with pulmonary tumor embolism. According to the literature review; we present the biggest malignant mass in the heart secondary to cervical adenocarcinoma. A 43-year-old woman was admitted to our hospital with complaints of rest dyspnea which had progressed over 2 weeks. She had a history of metastatic (pancreas and kidney) endocervical adenocarcinoma which diagnosed 11 months ago. She had conservative chemo-radiotherapies in the meanwhile. Physical examination revealed the absence of audible breath sounds over the chest and hypotension (83/57). Transthoracic echocardiography (TTE) revealed massive pericardial (max: 5.03 cm) and pleural effusion with a swinging heart and a big right ventricular mass (6.40x6.21 cm) arising from interventricular septum and narrowing the RV outflow tract. RV free wall was ruptured because of erosion by the mass and an echo free gap in 1.29 cm dimension was determined in the free wall (Figure 1a-d; Video 1, 2). Color flow did not reveal a blood flow in the wall. RV systolic function was severely depressed.CT scan showed that RV was almost filled with the septated mass(6.26x6.85cm). Additionally; massive pericardial effusion (max:4.14cm) and pulmonary artery filling defect which consistent with tumor embolism were obtained (Figure 2a-d). The patient was allowed to intensive care unit and an urgent subxiphoid incision with pericardial drainage was performed through a pericardial window, which permitted removal of 650 mL of serosanguineous fluid. The pericardial fluid was exudative and fluid immunohistochemistry was sufficiently directive. Cell block immunohistochemically staining demonstrated strongly positive for CA 125, Kit-ligand-1, Ki67, and CK7. The cell block results directed us cervical type pericardial metastasis as compatible with CT and TTE. The patient's clinical course was unfavorable. Her hemodynamic state was not improved after the drainage probably because of pulmonary embolism and right ventricular filling and emptying failure. Despite the fact that inotropic agents and fluid therapy were administered; the patient was died at 6th day of the admission due to deep hypotension and multiple organ failure.

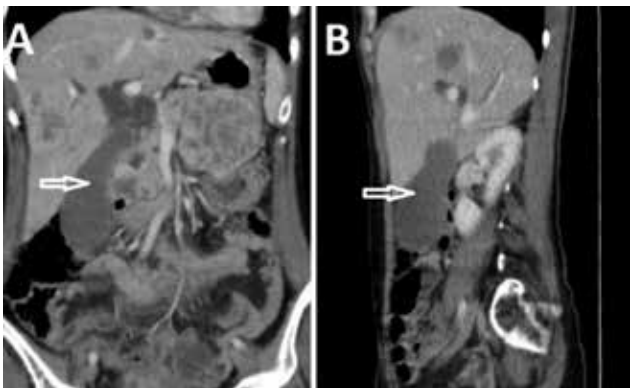

Figure 3. (A, B) Coronal and sagittal computed tomographics scans show dilated choledochus duct.

OS-89

A rare association of hypoplasia of the posterior mitral valve and bicuspid aortic valve demonstrated by three-dimensional transesophageal echocardiography

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A 35-year-old adult female with no previously known heart disease was admitted to our hospital

with dyspnea on effort. Cardiac auscultation revealed a diastolic murmur at the second right intercostal space. Transthoracic echocardiography (TTE) revealed the almost complete absence of the posterior mitral leaflet (PML) and bicuspid aortic valve (BAV). Clinical, microbiological or imaging finding of infective endocarditis was not observed. Subsequently, transesophageal echocardiography (TEE) was done, which showed an elongated, and mobile anterior mitral leaflet (AML) with severe hypoplasia of PML (Fig. 1a, Video A), and the presence of tip 1 BAV with a raphe (Fig. 1b). Color-doppler examination revealed a mild mitral regurgitation and a moderate aortic regurgitation. 3D-TEE additionally helped in the appraisal of the mitral valve abnormality (Fig. 1c, d, Video B). Ultimately, annual follow-up by TTE was suggested to the patient. Hypoplasia of PML, which is an extremely rare congenital anomaly results from the developmental arrest during conversion of the muscular chordae and leaflet to connective tissue. Dysplastic mitral valve coexisting with other cardiac abnormality is well-known, however, which accompanied by BAV is unusual. Although, Saura D, et al. have suggested that both conditions show familial aggregation, therefore, may have a genetic ground, it is not still well-known whether the association herein described is induced by a genetic abnormality or is just a coincidence. Accordingly, more investigation and experience need to answer the question about the relation between hypoplasia of PML and BAV.

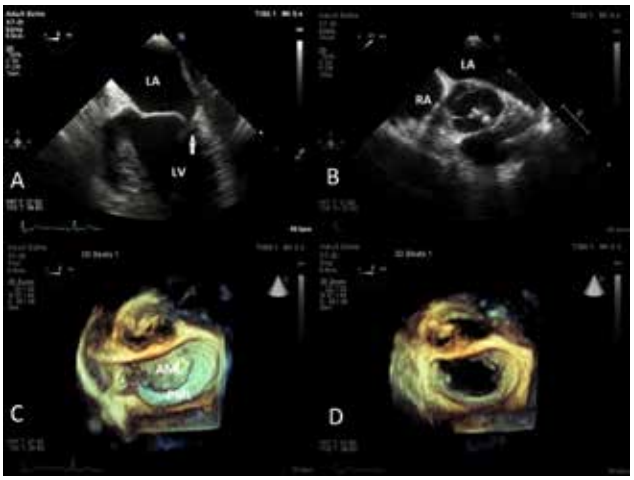

Figure 1. TEE showing an elongated and huge AML with a mild degree of prolapse, severe hypoplasia of PML (white arrow) **(A)**, and the presence of tip 1 BAV with a raphe **(B)**. 3D-TEE short axis view mid-systolic of the mitral valve **(C)**, mid-diastolic view **(D)**.

OS-90

Degeneration of aortic bio-prosthetic valve during pregnancy

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Left-sided infective endocarditis may cause severe valvular destruction and result in high rates of valve replacement. However, after valve replacement recurrence of endocarditis or valvular and paravalvular degeneration is common and pregnancy may accelerate this process. We report a case with a degenerated aortic root and aortic bioprothesis and acute heart failure at last trimester of pregnancy.

Case Report: A 26 years-old female patient at 38th week of gestation admitted our emergency department, with acute dyspnea and hypertension. She had a bioprosthetic aortic valve (St. Jude No: 21, Minnesota, USA) replacement history because of complicated infective endocarditis 16 months ago. She was in Killip class III. Transthoracic echocardiography (TTE) showed severe valvular and suspicious paravalvular aortic regurgitation, aortic annular aneurysm. After stabilisation of clinical state urgent caesarean section was performed. Transesophageal echocardiography revealed severe valvular and paravalvular regurgitation, aortic root dilatation with paravalvular aneurysm measuring 13.8x21.9 mm. Suspicious connection between aneurysm and left ventricular cavity was seen. Aortic root dilatation and pseudo- aneurysm was also confirmed with coronary CT angiography. Her blood culture results were negative for recurrence of endocarditis. After regression of heart failure symptoms the patient was referred to cardiovascular surgery department. This case highlights that cardiac surgery is not always curative after endocarditis. Structural valve deterioration was reported in 47% of patients with history of pregnancy, compared with only 14% in the non-pregnant group. Special consideration should take place during follow-up of pregnant patients with history of prosthetic valve operation.

OPS1-01

Apical hypertrophic cardiomyopathy previously misdiagnosed as recurrent inferior myocardial infarction

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Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul

A 56-year-old man was referred to our cardiology clinic with a long history of chest pain. He had diagnosed with inferior myocardial infarction for 3 times and undergone coronary angiogram recently at different hospitals. All of the angiograms demonstrated noncritical coronary artery disease. Upon physical examination, his heart rate was 54 beats/min and his blood pressure was 106/70 mmHg. No extra heart sounds or murmurs were heard, even after provocative maneuvers. His electrocardiogram showed, ST elevation in D3, aVF and aVR, left ventricular (LV) hypertrophy, and negative T waves in derivations from the precordium and D1-aVL (Fig. 1). Echocardiography revealed a preserved LV systolic function with normal LV wall motion and increased thickness at apex. There was no LV outflow or midventricular obstruction. To investigate the cause of the abnormalities on the electrocardiogram and echocardiography, we performed cardiac magnetic resonance imaging (MRI). We obtained cardiac-gated, multiplanar cine images, which demonstrated a marked concentric thickening of the LV apex that gradually decreased to normal levels at the base (Fig. 2). Comment: Apical hypertrophic cardiomyopathy (AHCM) is characterized by hypertrophy of the myocardium, predominantly in the left ventricular apex. This relatively rare variant of HCM, constituted 13% to 25% of all cases of HCM. Despite a relatively good prognosis for AHCM, long-term observations have occasionally included sudden cardiac death, severe arrhythmias, and apical infarctions with apical aneurysms. A "spade-shaped" configuration of the LV cavity at end-diastole on ventriculography and "giant" T-wave negativity in the electrocardiogram have been reported as the typical findings for this abnormality. AHCM, Echocardiography has been the 1st-line imaging method for patients with suspected HCM, but its shortcomings in evaluating the apex are well known. A cardiac MRI should be performed if the electrocardiogram raises suspicion of AHCM and if echocardiographic results are inconclusive or technically inadequate. Some echocardiographers will miss the diagnosis of apical thickening if the apex is not clearly seen, or if a thickened apex is mistaken for apical foreshortening on planar imaging. Cardiac MRI avoids this problem because it is less dependent on operators, is not subject to acoustic-window limitations, has multiplanar capability, and displays excellent soft-tissue contrast.

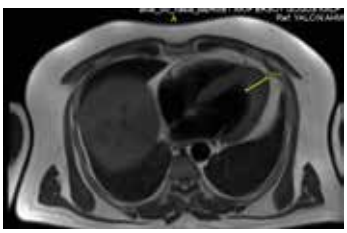

Figure 1. Cardiac MRI of the patient demonstrating apical hypertrophy.

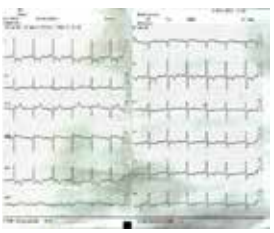

Figure 2. Electrocardiogaphy. Atypical electrocardiogram of the patient with apical hypertrophic cardiomyopathy.

OPS1-02

A rare cause of ST elevation: Aluminum phosphide intoxication

Ali Erayman, Alper Buğra Nacar, Mustafa Şahan, Mehmet Duru

Mustafa Kemal University Faculty of Medicine, Hatay

21 year old Syrian woman applied to ER with vomiting and nausea after suicidal unknown drug ingestion approximately ten hours ago. Her blood pressure was 70/40 mmHg, heart rate was 140 and confusion was present. In ECG there was inferolateral myocardial infarction pattern (Figure 1). Echocardiography showed left ventricular global systolic dysfunction with a 30% ejection fraction. Serum troponin level was high, Afterwards coronary angiography performed to rule out coronary artery occlusion. There was no occluded coronary artery nor unstable plaque formation. After angiography she was hospitalized in intensive care unit. Further investigation revealed that she had taken aluminum phosphide orally. During hospitalization period ST segment elevation resolved in time and no pathologic Q waves occurred in the ECG, but frequent ventricular extrasistols were seen (Figure 2). After one week she was discharged. Her ejection fraction was %35 at discharge. Heart failure drugs were prescribed and readmission to cardiology policlinics was advised after one month but she didn't come. Aluminum phosphide (AlP) is a highly toxic compound which is used for fumigation of grain silos as a rodenticide and insecticide. Exposure can be with inhalation of phosphine gas or can be orally for suicidal manner. It reacts with moist and produce phosphine (PH3) gas. Phosphine induces oxidative stress and boosts extra-mitochondrial release of free oxygen radicals that cause lipid peroxidation and protein denaturation of the cell membrane. Usually cardiac arrhythmias or cardiac failure is the cause of death. Increased left ventricle dimensions, decreased ejection fraction, severe hypotension and electrocardiographic (ECG) abnormalities such as dysrhythmia, ST-T wave changes and conduction defects are seen as in our case. In ECG sinus tachycardia seen in the first three to six hours of poisoning, followed by ST-T changes between six and twelve hours, and then by arrhythmias. Phosphine shows these toxic effects on myocardium with the mechanism which described above. It does not cause any thrombus formation in the epicardial arteries as shown in our case. There is no specific antidote in AlP intoxication and treatment is mainly supplementary. This case demonstrates in AlP toxicity the myocardial damage is at the cellular level. There is no thrombus formation in epicardial arteries that can explain the ECG changes or myocardial damage.

Aile hekimliği

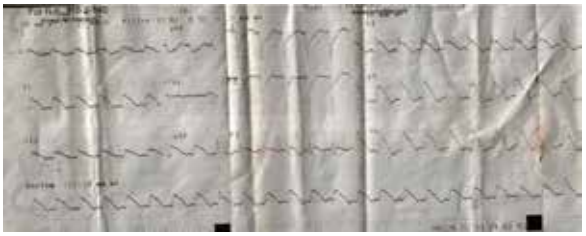

Figure 1. ECG was taken at ER after patient's admittance. ST segment elevation is prominent in inferolateral leads.

OPS1-03

Caseoma: A rare echocardiographic finding

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Finding: Chronic caseous degeneration in the internal material of mitral annular calcification is the underlying mechanism of this condition. On the echocardiographic evaluation, the calcification is around the mass with central echolucent area, typically located at the base of the posterior leaflet. This echocardiographic finding can be mistaken fo cardiac tumors, vegetations or abscesses.

Case: A 78-year-old woman with a history of hypertension, diabetes mellitus and chronic obstructive pulmonary disease was admitted for dyspnea upon minimal exertion. Her white blood cell count was normal, erythrocyte sedimentation rate and CRP levels were minimally elevatted. PA chest X-ray showed massive plevral effusion at right hemithorax and elevated cardiothorasic rate. Transthoracic echocardiography (TTE) showed a large calcified mass of 27x24 mm in the posterior mitral annulus that extended to the most basal area, the P1-P2 scallops, without causing valve stenosis (E velocity: 1.1 m/s) and with moderate mitral regurgitation. Left atrial chamber and right heart chambers were dilated, left ventricular ejection fraction was 46% (modified Simpson) on echocardiographic evaluation. The patient was offered for transesophageal echocardiography, but patient refused invasive cardiac procedures. During the follow-up period, the patient was treated with supportive medical treatment.

Discussion: Echocardiographic series report a low prevalence of caseoma, between 0.06% and 0.07%, and up to 0.6% of patients with calcification of the mitral annulus (CMA). Due to the structural deformation caseomas associated with mitral valvular dysfunction, both stenosis and regurgitation. Due to the asymptomatic course, only monitoring of the patient's progress is recommended in most cases. Surgery should be choosen for patients with significant valvular dysfunction or an uncertain diagnosis. In conclusion, caseoma is a rare variant of calcification of the mitral annulus, but should be evaluated for the differential diagnosis with other cardiac masses to prevent unnecessary surgery.

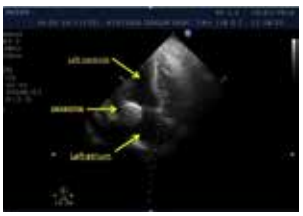

Figure 1. Caseoma on TTE with apical four chamber view. A large calcified mass in the posterior mitral annulus.

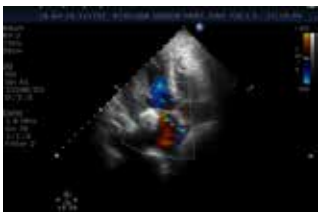

Figure 2. Eccentrical mitral regurgitation caused by caseoma.

OPS1-04

A rare congenital malformation in an elderly patient: Uhl's anomaly

Nurşen Keles[1], Serçin Özkök[1], Macit Kalçık[2], Feyza Aksu[1], Kenan Demircioğlu[1], Yusuf Yılmaz[1], Mustafa Çalışkan[1]

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A 62 years old female was admitted to our outpatient clinic with exertional dyspnea. On physical examination there was mild pretibial oedema and jugular venous distention with no cyanosis. A pansystolic murmur was present at the apex. She had been diagnosed as Ebstein's anomaly during childhood. Transthorasic echocardiography (TTE) which was performed on admission revealed distinct dilatation of right atrium and right ventricle with right ventricular systolic dysfunction and severe tricuspid valve regurgitation. The systolic function and the diameters of the left ventricle were normal. We also observed a thin endocardial and epicardial layer of right vetricle parallel to each other which was consistent with the absence of myocardium of right ventricle (Figure 1a, b). Therefore cardiac MRI was planned in order to diagnose the absence of right ventricular myocardium. Steady-state free-precession (SSFP) cardiac MRI images described a marked dilatation of the right atrium and the right ventricle with the complete absence of the right ventricular myocardial layer (white arrows) (Figure 2a, b) The left ventricle is constircted and displaced laterally on long-axis four-chamber views. The ejection fraction of right ventricle was reduced. Tricuspid valve hinged normally and was not dysplastic. The severe tricuspid valve regurgitation was considered to be due to annular dilatation. She had been diagnosed as Ebstein's anomaly during childhood and had been followed as Ebstein's anomaly till date. However, we showed that she did not have Ebstein's anomaly, actually she had UA which was demonstrated with cardiac MRI."

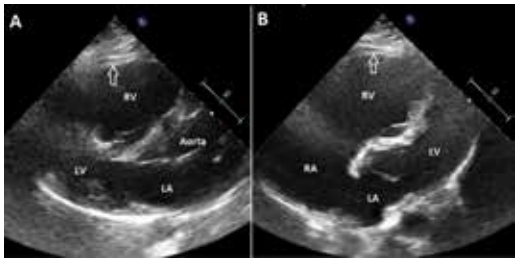

Figure 1. (A) and apical four chamber **(B)** views demonstrated dilatation of right chambers and a thin endocardial and epicardial layer of right vetricle parallel to each other (arrows) which was consistent with the absence of myocardium of right ventricle.

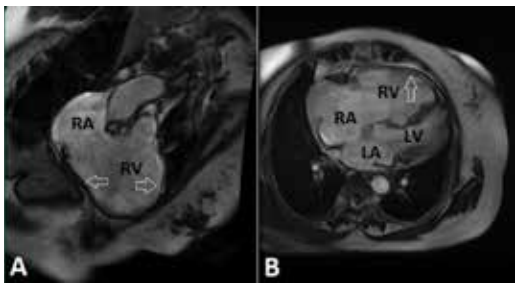

Figure 2. Cardiac magnetic resonance imaging steady-state free-precession (SSFP) images showed marked dilatation of the right atrium and the right ventricle with the complete absence of the right ventricular myocardial layer (white arrows)

OPS1-05

Anterior ST-segment yükselmeli akut miyokart enfarktüsünün sağ ventrikül tutulumlu akut miyokart enfarktüsü ile birlikteliği

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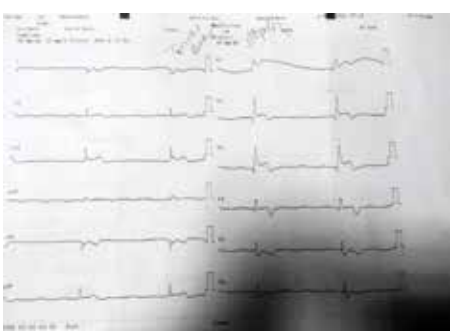
Akut koroner sendromda elektrokardiyografi (EKG) tanı konulmasının yanında, enfarktüs lokalizasyonun saptanması, riskin belirlenmesi, tedavinin yönlendirilmesi ve tedavi başarısının tespitinde ilave katkılar sağlar.(1)EKG de inferior derivasyonlarda ST yükselmesi izlendiğinde sorumlu lezyon kesin olmamakla birilke genelde sağ koroner arterdir; oklüzyon sol ön inen arterde ise ST segment yükselmesi prekordiyal derivasyonlarda tezahür eder. Ekg Sol ön inen koroner arter (LAD) ile sağ koroner arter (RCA) tıkanmalarını, sirkumfleks arter (Cx) tıkanmalarına göre daha yüksek bir duyarlılıkla ortaya koyabilmektedir. Sağ ventrikül miyokart enfarktüsü (Sğ V ME), akut inferior miyokard enfarktüslü hastaların %23-53'ünde görülmektedir. Sağ V ME'ünde hemodinamik bozukluklar erken ortaya çıktığı için olguların erken tanınıp uygun intravenöz volüm yüklenmesiyle tedavi edilmesi önemlidir. SğV Mİ tanısında V4R-V6R de 0,5-1mm üstünde ST segment elevasyonunun tanı koyduracağı, V1R-V3R nin de tanıya yardımcı olabileceği bildirilmiştir. V4R de ST segment elevasyonu bunlardan en çok kullanılanıdır.

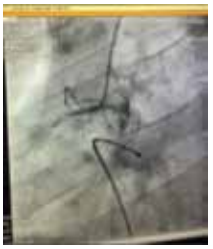
Bulgular: Öyküsünde diabetes mellitus,dislipidemi ve sigara içme öyküsü olan 45 yaşındaki erkek hasta sıkıştırıcı tarzda göğüs ağrısı,terleme ve baş dönmesi şikayeti ile başvuruyor.Hastanın çekilen EKG de anterior derivasyonlarda ve sağ derivasyonlarda ST segment yüksekliği bulunması ve ritmin atrıyoventriküler tam blok(AV tam blok) ile uyumlu olması nedeniyle primer perkutan koroner girişim (PKG) ihtiyacı açısından dış merkezden tarafımıza yönlendirildi.Hasta primer PKG planlanması amacıyla katater laboratuarına alındı.Hastanın ritminin AV tam blok ile uyumlu olması nedeniyle öncelikli olarak hastaya geçici pacemaker takıldı.Hastanın çekilen koroner anjiografisinde; RCA %100 ve LAD Diagonal 1 sonrası %99, Cx'in %70, %80 tıkalı olduğu görüldü. Hastanın kliniğinden iki lezyonun sorumlu olduğu düşünüldü.Ardından hastanın LAD %99 ve RCA %100 lezyonuna başarılı primer PKG yapıldı. İşlem sonrası çekilen EKG'de ST segment elevasyonunda ve hastanın göğüs ağrısında anlamlı azalma olduğu görüldü.Takibinde hastanın ritminin sinüs ritmine döndüğü izlendi.

Sonuç: Hastada iki sorumlu lezyon (RCA %100, LAD %99) izlendi. Hastanın kliniğinden hem RCA'nın hem de LAD'nin sorumlu olduğu düşünüldü ve her iki lezyona başarılı primer PKG uygulandı.Anterior STYME tanısı ile başvuran hastada eş zamanlı Sğ V MI ve av tam blok olması literatürde nadir rastlanan bir durum olduğu için paylaşmak istedik.

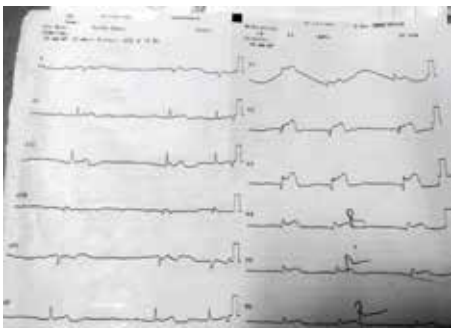


Anterior STYME+AV Tam blok.

LAD'de %99 lezyon mevcut.



RCA'da %100 lezyon mevcut.



Sağ V MI EKG bulgusu.

OPS1-06

Likenoid type cutaneous hyperpigmentation induced by nebivolol

Abdullah Nabi Aslan[1], Murat Can Güney[2], Hüseyin Ayhan[2], Telat Keleş[2], Murat Akçay[2], Tahir Durmaz[2], Engin Bozkurt[2]

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Cutaneous hyperpigmentation is a common and well defined side effect of many drugs such as nonsteroidal antiinflammatory drugs, beta-blockers and tetracyclines but to the best of our knowledge there is not any case of skin discoloration related to nebivolol. A 46 years old female patient without any other medical history diagnosed with hypertension and was prescribed 5 mg of nebivolol daily 15 months ago. The patient was not using any other medication and during her follow up blood pressure levels remained in the normal range. 3 months after the prescription, the patient was admitted to the dermatology clinic of our hospital with skin discoloration. The hyperpigmentation was extensive and involved the sun-exposed areas of the body like face, hands, and neck; the other areas were spared. Physical examination was normal except for the skin pigmentation. Laboratuary tests were normal and patient did not had a history of any other diseases besides hypertension. Clinical diagnosis was suggestive of a hypersensitivity reaction to the sunlight. However, although the patient was devoid of sunlight for at least six month of duration, the pigmentation persisted for one year. Drug habituation of the patient including herbal medicines was meticulously examined and revealed that nebivolol was the only agent used by the patient. Histopathological examination of the skin biopsy taken from the hyperpigmentated regions showed likenoid type of cutaneous hyperpigmentation in the basal cell layer, pigment incontinence within the dermis, and accumulation of pigment-laden macrophages around blood vessels and eccrine glands. Nebivolol was considered as the potential cause and following three months after the cessation of the drug skin lesions dissappeared completely.

OPS1-07

Severe gastrointestinal bleeding due to angiodysplasia after transcatheter aortic valve implantation in a patient with high frailty

Ali Osman Yildirim, Cengiz Ozturk, Turgay Celik, Mustafa Demir, Sevket Balta, Ugur Bozlar, Ahmet Ozturk, Adem Guler, Mehmet Ali Sahin, Atila İyisoy

Gülhane Military Medical Academy, Ankara

We report a case of a 76-year-old man presenting with a massive intestinal bleeding originating from intestinal angiodysplasia after percutaneous aortic valve replacement procedure due to the triple therapy. The patient was admitted to our hospital because of severe and symptomatic coronary artery left anterior descending artery (LAD) and aortic valve stenosis. There was significant and calcific stenosis on LAD. Left ventricular ejection fraction (LVEF) was 26%. Aortic valve was severely calcific and aortic valve area was 0.6 cm^2. Aortic valve maximal/mean pressure gradient: 47/30 mm. There was viable tissue in the myocardium and aortic valve mean gradient was increased to 48 mmHg on dobutamine stress echocardiography. LVEF was also increased to 50%. The patient was diagnosed as left heart failure, low flow-low gradient aortic valve stenosis and LAD stenosis, so he was referred to the surgery but he was rejected because of the high calculated Euro Score II value (65%). Because of the adequate left ventricular reserve, our heart team decided to perform the TAVI procedure because of the high cardiac surgery risk and the patient's poor clinical state. First of all, we implanted a VVI-R pacemaker and implantable cardioverter defibrillator because of the low LVEF for primary prevention. Then one week later, after adequate balloon inflation, a 4.0x18 mm drug eluting stent was implanted to calcific lesion at the mid segment of the LAD. After 6 months, the patient was still symptomatic and had low LVEF and low gradient.

Previously adjusted balloon under rapid pacing was inflated at the level of the aortic valve. After balloon-pre-dilatation, a 26 mm Edwards Sapien XT valve was implanted. The procedure was finished succesfully with no complication and the patient was discharged with acetylsalicylic acid-100 mg, clopidogrel-75 mg and apixaban-2x2.5 mg at 7 days after procedure. We have to start tripple therapy because of the risks of stroke and valve thrombosis. When the patient was using this therapy, he was admitted to the hospital because of severe gastointestinal bleeding and he suffered severe recurrent blood loss from intestinal angiodysplasia two months later the TAVI procedure. The anticoagulants and antiaggregants must be administered carefully in patients with high bleeding and thrombosis risk in order to minimize the risk of further bleeding.

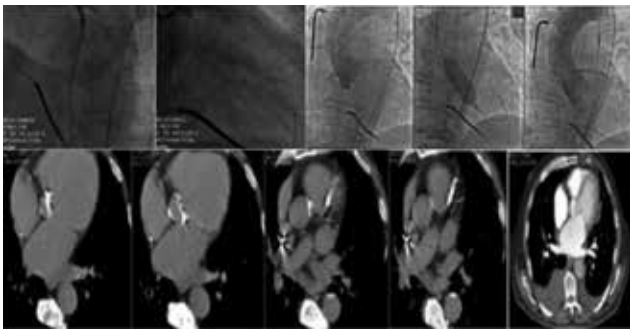

Figure 1.

OPS1-08

Successful implantation of a cardiac devices via a persistent left superior vena cava

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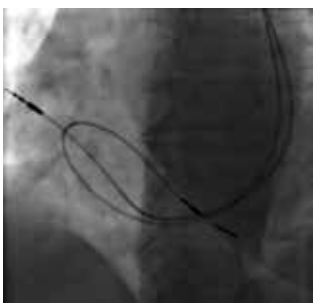
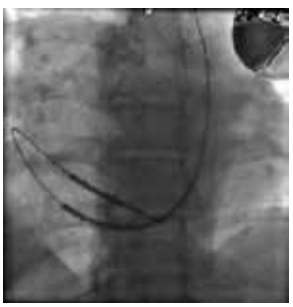
Private Koru Ankara Hospital, Ankara

Persistent left superior vena cava (PLSVC) is the most common variation in the thoracic venous system. PLSVC is found in 0.3% to 0.5% of the population and in 5% to 10% of patients with other congenital heart defects., However; Biffi et al found 0,41% prevalence with the absence of RSCV in 36% of cases in this population. This condition is typically asymptomatic, usually incidentally discovered during pacemaker implantation can complicate lead placement through the subclavian approach. We reported a case of a successful pacing and defibrillator device implantation via a persistent left superior vena cava in our two patients (DDD-R pace maker and VR-ICD).



DDD-R pacemaker. Antero-posterior (AP) view. Depicting final position of the right atrial and ventricular pacemaker leads; note how the right ventricular lead is looped in the right atrium to redirect its trajectory towards the ventricular apex. Atrial lead is screwed in the high lateral wall of the right atrium.



VR-ICD. Antero-posterior (AP) view. Depicting final position of the rigt ventricular ICD leads.

OPS1-09

Arrhythmia induced cardiomyopathy secondary to atrioventricular reentrant tachycardia in a patient with multidrug abuse

Göksel Çinier, Ahmet İlker Tekkeşin, Yasin Çakıllı, Ceyhan Türkkan, Yalçın Velibey, Nizamettin Selçuk Yelgeç, Ahmet Taha Alper

Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul

22 years old male patient presented to the emergency department with shortness of breath and swelling in both legs. His complains developed gradually during last six months but worsened recently. His medical history revealed nothing important. The patient stated that he abused illicit drugs, mostly CB's, for 5 years and last event was 15 days ago.12-lead surface electrocardiography (ECG) revealed regular rhythm, wide QRS tachycardia with left bundle branch block (LBBB) morphology and heart rate of 180 beats per minute (Figure 1a). Interestingly patient did not have sense of palpitation even with this high heart rate. Normal sinus rhythm was achieved with carotid massage; however, wide QRS tachycardia with right bundle branch block (RBBB) morphology ensued immediately after short duration of sinus rhythm (Figure 1b). Transthoracic echacardiography (TTE) was performed afterwards and revealed depressed LV ejection fraction (EF) (32%, global hypokinetic, calculated by Simpson's method)), dilated left ventricular cavity [LV end-diastolic diameter (LVEDD): 54 mm, LV end-systolic diameter (LVESD): 47 mm] (Video 1). During hospitalization in the ICU wide QRS tachycardia episodes with occasional LBBB and RBBB morphogy resistant to multiple anti-arrhythmic drugs recurred. The patient's hemodynamic status were deteriorated during tachycardia episodes and synchronized direct current cardioversion (DC/CV) was required to achieve sinus rhythm. To define the etiology of tachycardia the patient was underwent electrophysiology study (EPS) which revealed left lateral accesory pathway (Figure 2 and 3). The accesory pathway was ablated successfully without any complication. In the outpatient clinic visit at the third month, TTE revealed complete recovery of LV systolic functions (EF: 63%, calculated by Simpson's method) and normal LV cavity diameters (LVEDD:48 mm, LVESD: 32 mm). 24 hours holter monitoring revealed no tachycardia episodes or any other clinically significant arrhythmias.

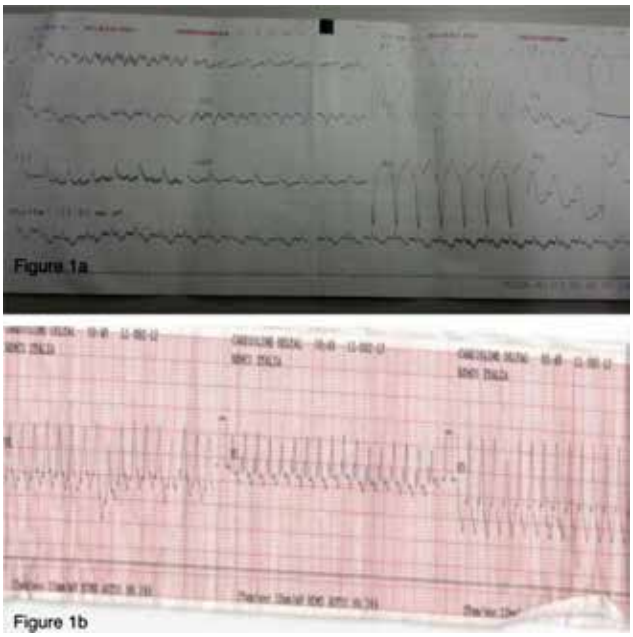


Figure 1. (A) ECG showing wide QRS tachycardia with left bundle branch block morphology. **(B)** ECG showing wide QRS tachycardia with right bundle branch block morphology.

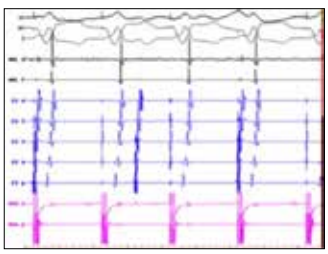


Figure 2. Electrophysiologic study revealing left accessory pathway.

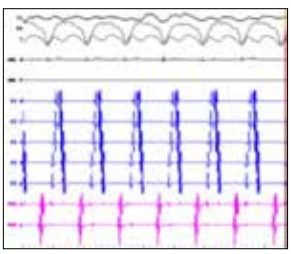


Figure 3. Electrophysiologic study revealing left accessory pathway.

OPS1-10

Primer idiyopatik şiloperikardiyum

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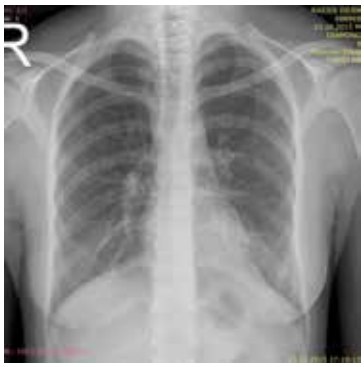
Giriş: Perikardiyal kavite içerisinde şilöz sıvı toplanması olarak bilinen şiloperikardiyum perikardiyal efüzyonun nadir görülen ancak hayatı tehtit edebilen bir formudur. Şiloperikardiyum primer (idiyopatik) olabileceği gibi, sıklıkla sekonder sebeblere bağlı oluşabilmektedir. Seconder sebelere bağlı en sık karşılaşılan durumlar ise göğüs ve ya kalp cerrahisi sonrası, travma, konjenital lenfanjiomatozis, radyoterapi öyküsü, subclavien ven trombozu, enfeksiyon (tbc), mediastinel lenfoma gibi durumlar yer almaktadır.

Olgu: 30 yaş kadın hasta dış merkez kardiyoloji polikliniğinde perikardit nedeniyle tedavi olmuş ancak nefes darlığının giderek artması üzerine tarafımıza başvurdu.Hastanın çekilen elektrokardiyografisnde (EKG) voltaj kaybı mevcuttu. Hastaya ekokardiyografi (EKO) ve akciğer grafisi planlandı. Çekilen akciğer grafisi normal olarak değerlendirildi. Hastanın yapılan EKO'sunda RV çevresinde diastolde bası bulgusuna yol açan 3,4 cm perikardiyal efüzyon saptanması üzerine hastaya perikardiyosentez planlandı.750 cc şiloz karekterde perikardiyal sıvı boşaltıldı. Alınan sıvıdan gönderilen örnekte Trigliserid (TG): 2217 mg/dl, kolesterol /TG oranı <1 olarak hesaplandı. Kültürlerde bakteriyel üreme olmadı. Hastanın tetkiklerinde kan sayımı, elektrolitler, karaciğer fonksiyon, lipid profili, serum üre, serum kreatinin, serum kalsiyum ve laktat dehidrogenaz, C-reaktif protein, toplam lökosit sayısı,eritrosit sedimantasyon hızı,antinükleer antikor ve romatoid faktör değerleri normal olarak izlendi. Çekilen göğüs tomografisinde perikardiyal efüzyon dışında bir özellik mevcut değildi. Tomografi ile mediastinel lenfoma, tüberküloz ve diğer maligniteler lehine anlamlı sonuç elde edilemedi. Perikard sıvısının kültürlerinde bakteriyel üreme saptanmadı. Mantoux testi negatif olarak sonuçlandı ve alınan perikardiyal sıvı kültürlerinde ve balgam kültüründe

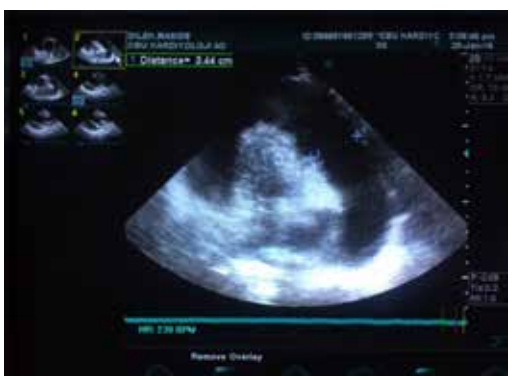
micobacterium tüberkülosis complexine rastlanmadı. Hastaya mevcut sonuçlarla idiyopatik şiloperikardiyum tanısı konuldu.Hastaya orta zincirli TG tedavisi başlandı. Hasta poliklinik kontrolüne çağırıldı.

Sonuç: Primer idiyopatik şiloperikardiyum orta zincirli TG ile zenginleştirilmiş düşük yağlı diyet içeren tedavi olabilen nadir bir durumdur. Bu yaklaşım başarısız olursa, cerrahi ligasyon ve perikardiyal pencere oluşturulması gereklidir. Bu olguyu da şilöz perikardiyumla karşılaşılması durumunda tanıya gidilmesi ve altta yatan sebeblerin araştırılması konusunda yol gösterici olacağını düşündüğümüz için paylaşmak istedik.



Akciğer grafisi.

EKG.



EKO görüntüsü.

Şilöz sıvı görüntüsü.

Aile hekimliği

OPS1-11

Farklı bir sol ana koroner arter perkütan koroner girişimi: LAD ostial aşırı ektazisi ve LMCA distal lezyonu olan bir hastada IVUS eşliğinde PKG

Sina Ali, Mehmet Bilge, Yakup Alsancak, Büşra Çolak, Muhsin Beyatlı

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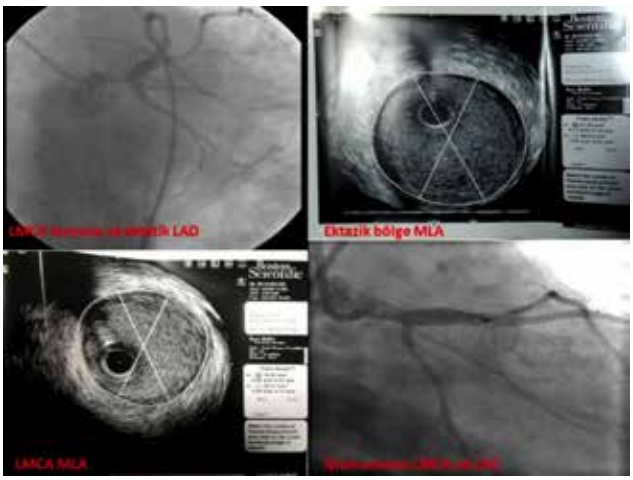
Giriş: Koroner anjiyografi yapılan hastalarda %4-6 oranında sol ana koroner lezyonu (LMCA) görülmektedir. Koroner arter ektazisinin (KAE) ise anjiyografi sonrası görülme sıklığı %0.3-5.3'tir ve bu durum akut koroner sendrom açısından bir risk faktörüdür. Ancak ciddi LMCA lezyonu ve eşlik eden koroner arter ektazi (KAE) oranı ile ilgili literatürde çok az bilgi bulunmaktadır.

Olgu: 52 yaşında 2 ay içerisinde iskemik serebrovaskuler olay sonrası sol hemipleji gelişen hastaya göğüs ağrısı nedeniyle dış merkezde yapılan koroner anjiyografi sonrasında bypass cerrahisi önerilmiş. Ancak yüksek risk (log.EuroScore %14, karotis arter hastalığı; sol taraf endarterektomi öyküsü, sağ tarafta %70 darlık) nedeniyle işlemden vazgeçilmiş. Hastanın izlenen KAG'sinde LMCA diffuz ciddi gövde lezyonu (medina 1,0,0) (Şekil 1) ile ektatik LAD osteal ve proksimal lezyonu ve ciddi gelişmiş diyagonal 1 arter lezyonu izlendi. İVUS incelemesinde LAD ostiyumunu içine alan proksimal LAD'de ektatik bölge çapı 6.8x6.2 mm olarak saptandı. Ayrıca IVUS ile Cx ostiyumunun iyi olduğu gözlendi. LMCA çapı ise 5.5 mm olarak değerlendirildi. Bunun üzerine provizyonel yan dal stentleme tekniği seçilerek öncelikle LMCA ve LAD proksimali dilate edildi. yapılmış olan çalışmalarda 4.0 mm çapında everolimus kaplı stentin (Xience) 6.0 mm çapında bir balonla 5.5 mm'ye kadar genişleyebileceği gösterilmiştir. Bu bilgiler göz önüne alınarak LMCA'ya 4.0x15 mm Xience (28 atm), LM gövdesinden ektazik LAD'yi içine alacak şekilde LAD'ye doğru uzatıldı. İmplantasyon sonrasında LMCA'da bulunan stent ise 6.5x15 mm periferik balon ile post-dilate edildi (POT). İşlem sonrası yapılan IVUS incelemesinde LMCA'dan LAD ektatik bölgeye uzanan stentin apoze olduğu görüldü ve işleme son verildi.

Sonuç: Son zamanlarda LMCA artere uygulanan PKG'lerinen az cerrahi kadar etkin olabileceği gösterilmiştir (düşük ve orta SYNTAX skorunda). Uzun dönem takiplerde major kardiyovaskuler olaylarda PKG ve CABG arasında anlamlı farklılık izlenmemiştir (%15.8'e karşın %13.7). Ancak tekrarlayan revaskularizasyon ihtiyacı (orta-yüksek SYNTAX grubu) PKG kolunda daha fazla izlenmektedir (%11.8'e karşın %6.5) (4). SYNTAX skoru yüksek olan (≥33) hastalarda cerrahi ile hedef lezyon revaskülarizasyon oranı PKG ile karşılaştırıldığında daha yüksektir. Bununla beraber, sunduğumuz vaka gibi LMCA lezyonu ve KAE'si olan, cerrahi için yüksek riskli hastalarda medikal tedaviye göre PKG uygun bir seçenek olabilir.



Şekil 1. LMCA lezyonu, ektazi IVUS görüntüsü ve işlem sonrası görünüm.

OPS1-12

Pseudo-pericardial tamponade caused by huge mediatinal metastatic mass

Ekrem Şahan[1], Suzan Şahan[2], Murat Gül[3], Murat Karamanlıoğlu[1], Omaç Tüfekçioğlu[2]

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[3]*Düzce Atatürk State Hospital, Düzce*

A 71-year-old male patient (diagnosed and followed-up with malign mesotelioma for three years) presented to our emergency department with a complaint of shortness of breath after doing normal daily activities that had persisted for two weeks. He also had fever for the last week along with night sweat, and weakness. He was examined thoroughly and significant findings were narrow pulse pressure of 13 mm of Hg (systolic blood pressure was 112 mm of Hg and diastolic blood pressure was 99 mm of Hg) and elevated jugular venous pressure of 11 cm of H2O. To evaluate the cause of clinical status and diminished heart sound, echocardiography was performed immediately. Echocardiography showed that right ventricle mid-apical free wall was compressed by a huge 6.1x4.2 cm anterior mediastinal mass and mild pericardial effusion. Computed tomography (CT) chest scan showed mediastinal mass and scan views confirmed the echocardiographic finding. After initial evaluation, supportive treatment was started and patient was referred to the surgical treatment.

Figure 1. Apical four chamber view of right ventricle compressed by mediastinal mass.

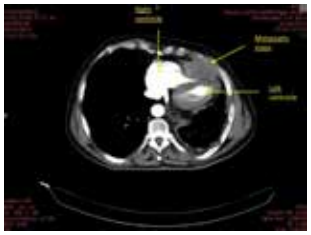

Figure 2. Computed tomography view of right ventricle compressed by mediastinal mass.

OPS1-13

A superdominant conus branch supplying both of totally occluded left anterior descending and right coronary arteries

Abdullah Nabi Aslan[1], Serdal Baştuğ[1], Hacı Ahmet Kasapkara[1], Nihal Akar Bayram[1], Hüseyin Ayhan[2], Telat Keleş[2], Tahir Durmaz[2], Engin Bozkurt[2]

[1]*Ankara Atatürk Training and Research Hospital, Ankara*
[2]*Yıldırım Beyazıt University Faculty of Medicine, Ankara*

A 44-year-old man presented to our polyclinic complaining of exertional chest pain with a walk-through phenomenon. At past medical history there was only hypertension and he was an ex-smoker. He has also a family history of coronary artery disease. Physical examination and electrocardiogram were unremarkable. Transthoracic echocardiography showed inferobasal and posterobasal hypokinesia and mildly decreased left ventricular ejection fraction (50%). Coronary angiography revealed a 100% stenosis at the proximal segment of left anterior descending artery and right coronary artery with a superdominant conus branch supplying both of them and normal circumflex artery (Figure 1a, b). We decided to follow the patient under medical therapy and patient was discharged from the hospital.

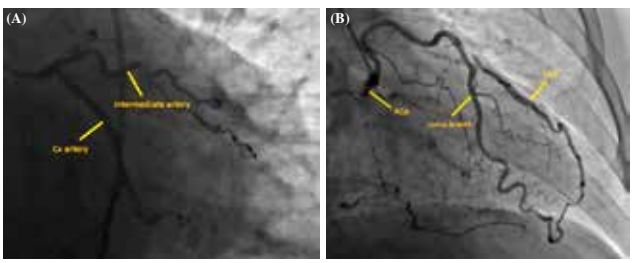

Figure 1. (A) Coronary angiography image showing a totally occluded left anterior descending artery. **(B)** Coronary angiography image showing a superdominant conus branch of totally occluded right coronary artery.

OPS1-14

Ciddi aort darlığı ve aort yetersizliği bulunan, aşırı derecede horizontal aorta ile geniş anülüse sahip bir olguda başarılı TAVI işlemi

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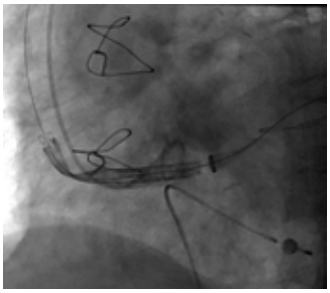
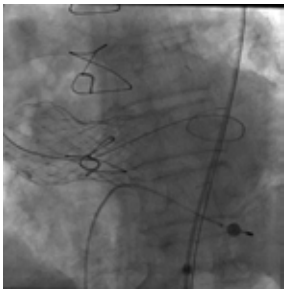
Giriş: Transkatater aort kapak replasmanı (TAVI), semptomatik ciddi aort darlığı olan ve açık kalp cerrahisi için yüksek riskli olarak kabul edilen hastalarda etkinliği kanıtlanmış alternatif bir tedavi yöntemidir. Sıklıkla daha ileri yaşlarda görülen horizontal aorta varlığı, TAVI işlemi sırasında ve sonrasında komplikasyon (cihaz embolizasyonu, yetersiz kapak açılımı, orta-ciddi paravalvuler aort yetersizliği, yüksek AV tam blok oranı ve artmış mitral kapak yetersizliği) gelişme riskini de beraberinde getirmektedir.

Olgu: Geçirilmiş koroner arter bypass cerrahisi öyküsü olan, diyabet ve hipertansiyon tanılarıyla takip edilen 85 yaşındaki erkek hasta efor dispnesi ve efor anjinası nedeniyle poliklinik şartlarında değerlendirilirken dış merkezde ciddi aort darlığı saptanması üzerine kliniğimize yönlendirilmiş. Hastanın ekokardiyografik incelemesinde ciddi aort darlığı (AVA: 0.65 cm^2, ort. gradient 52 mmHg), ciddi aort yetersizliği, orta derecede mitral ve triküspid kapak yetmezliği ile 28 mm aortik anülüs izlendi (EF: %40, PAB:65 mmHg). Aynı zamanda çıkan aort anevrizması (46 mm) tespit edildi. Yapılan bilgisayarlı tomografik incelemede aortik anülüs çapı 29 mm olarak saptanırken, hastanın horizontal aortaya sahip olduğu tespit edildi. Logistic EuroScore'u %21 olan hasta cerrahi açıdan yüksek riskli olarak değerlendirildi ve TAVI işlemine alındı.Aortik predilatasyon sırasında aortanın ileri derecede horizontal olmasından dolayı, sol ventriküle ilerletilen guidewire (Confida) birkaç kez sol ventrikülden ayrıldı. CoreValve biyoprotez kapak sisteminin ilerletilmesi sırasında da benzer sorunlarla karşılaşıldı. Birkaç denemeden sonra kapak uygun pozisyona getirilebildi. Kapağın uygun pozisyona getirilmesi sırasında sistemde ciddi bir direnç izlendi, kapak normalden daha baskılı bir biçimde aortik anülüse yerleştirilebildi. Sistemin proksimal kısmında açıya bağlı olarak kırılmalar izlendi, ancak kapak açılımı sırasında bu açılanmanın kaybolduğu görüldü. Kapağın sistemden ayrılması sırasında herhangi bir sorun izlenmedi ve kapak yüksek implantasyonla yerleştirilebildi. İşlem sonrasında hafif aort yetersizliği izlendi.

Sonuç: İleri derecede horizontal aorta nadir görülen TAVI işlemi sırasında implante edilecek biyoprotez kapağın uygun bir biçimde yerleştirilmesini zorlaştıran (kılavuz tellerin ilerletilmesi, aortik balon yerleştirilmesi, kılavuz pigtail yerleştirilmesi ve kapağın uygun pozisyona getirilmesi sırasındaki zorluk) önemli bir anatomik durumdur.



Şekil 1. Kapağın pozisyonu.



Şekil 2. İşlem sonrası kapağın görünümü.

OPS1-15

Successful transcatheter valve implantation of symetis ACURATE neo TF in a patient with severe stenotic, calcified and bicuspid aortic valve

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A 81-year-old male patient with exertional dispnea (NYHA class 2-3) and presyncope exacerbated within the last year, was admitted to the department of cardiology of our center. He had a past medical history of coronary heart disease (CHD) and benign prostat hypertrophy. Electrocardiogram showed sinus rhythm and signs of left ventricular hypertrophy. Transthoracic and transoesophageal echocardiographic assesment showed a normal left ventricular systolic function (LVEF: 65%), concentric left ventricular hypertrophy (interventricular septum/posterior wall: 1,3/1,4 cm), severe stenotic, degenerative and intensively calcified bicuspid aortic valve (aortic valve area: 0.9 cm^2, aortic maximum/mean gradient: 64/44 mmHg), moderate tricuspid regurgitation with high systolic pulmonary artery pressure (60 mmHg), and ascending aorta dilatation (AAD) (4.5 cm). The computed tomographic examination revealed a 25.3 mm sized aortic annulus (Figure 1, 2). The patient was evaluated by the Heart Team and transcatheter aortic valve implantation (TAVI) was decided (STS: 7.2%, logistic EuroSCORE: 21%). After obtaining the patient's written consent, a medium sized Symetis ACURATE neo TF (SATF) was implanted succesfully with a trivial paravalvular aort regurgitation (mean aortic gradient was 8 mmHg) (Video 1-4). Asymmetrical calcification rate is a common finding in bicuspid aortic valves. In these patients, the possibility of valve embolization is high, because of concomitant AAD incidence. SATF bioprosthesis valve is a new second generation valve with unique features of having upper crown and stabilization arches, providing lower rate of embolization via holding the valve in a more stable position. SATF may be a good choice in patients with severe bicuspid and calcified aortic stenosis.

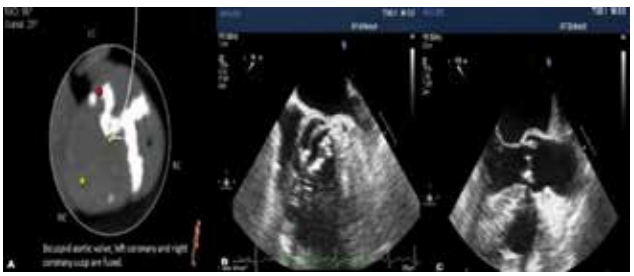

Figure 1. (A) Computed tomography is showing the bicuspid aortic valve, fusion of left coronary and right coronary cusps **(B)** Transesophageal echocardiography demonstrates the bicuspid aortic valve in short axis **(C)** Transesophageal echocardiography demonstrates the bicuspid aortic valve in long axis.

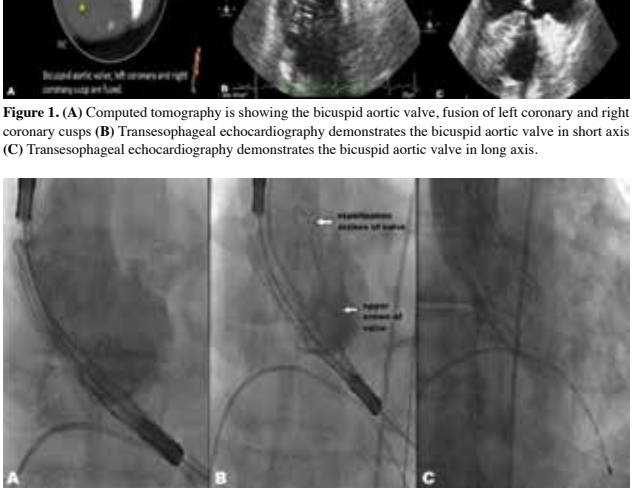

Figure 2. (A-C) Stabilization arches of valve and the position of the valve, Final aortography demonstrate no aortic regurgitation after the procedure.

OPS1-16

Anterior ST segment yükselmeli myokard infarktüsünün sağ ventrikül tutulumlu myokard infarktüsü ile birlikteliği

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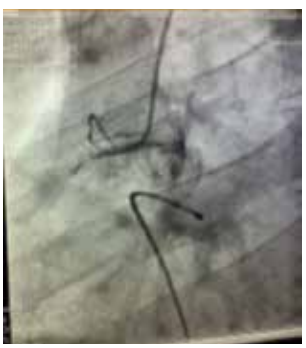
Celal Bayar Üniversitesi Tıp Fakültesi, Manisa

Öyküsünde tip 1 DM,dislipidemi ve 30 paket yılı sigara öyküsü olan 45 yaşındaki erkek hasta dış merkez acil servise saat 12:30 civarında başlayan sıkıştırıcı tarzda göğüs ağrısı, terleme ve baş dönmesi şikayeti ile başvuruyor. Hastanın dış merkez acil serviste çekilen elektrokardiyografisinde anterior derivasyonlarda ve sağ derivasyonlarda ST segment yüksekliği bulunması ve ritmin AV tam blok ile uyumlu olması nedeniyle primer perkutan koroner girişim ihtiyacı açısından tarafımıza yönlendirildi. Hastaya dış merkezde ASA 300 mg, klopidogrel 300 mg, enoksaparin 0,6 cc sc ve 1 ampul atropin uygulandığı belirtildi. Hasta merkezimize saat 15:30 civarında ulaştığında göğüs ağrısı devam etmekteydi. Hastanın tarafımıza başvurusunda yapılan fizik muayenesinde genel durumu kötü, kalp hızı 30/dk, bilinç bulanık, arteriyel tansiyon değeri 80/40 mmhg ve oksijen saturasyonu %90 olarak tespit edildi. Kardiyak fizik muayenesinde en iyi apekste duyulan 2/6 sistolik üfürüm mevcuttu. Solunum sistemi muayenesi olağan olarak değerlendirildi. Hastaya çekilen EKG de anterior ve sağ derivasyonlarda ST segment elevasyonu olduğu görülmesi üzerine hasta primer PKG planlanması amacıyla katater laboratuarına alındı. Hastanın ritminin AV tam blok ile uyumlu olması nedeniyle öncelikli olarak hastaya geçici pacemaker takıldı. Hasta katater laboratuarına alındıktan sonra çekilen koroner anjiografide; sağ koroner arterin (RCA) %100 ve sol ön inen koroner arterin (LAD) Diagonal 1 sonrası %99, sirkumflex arterin (Cx) %70 OM2 sonrası %80 tıkalı olduğu görüldü. Ardından LMCA ostiumu 7F EBU 3.5 kılavuz katater ile kanulize edildi. LAD D1 sonrası %99 lezyon 0.014 floppy guidewire ile geçildi.Hedef lezyona 2.0x15 mm balon ile predilatasyon yapıldıktan sonra 2.5x22 mm intrakoroner stent primer olarak lezyon üzerinde 16 atm'de 10 sn süre ile şişirildi ve TIMI III akım sağlandı. Daha sonra RCA ostium 6F JR-4 kılavuz katateri ile kanüle edildi. RCA ostiumundaki %100 lezyon 0,014 floppy guidewire tel ile geçildi. 3.0x18 mm intrakoroner stent primer olarak lezyon üzerinde 18 atm'de 10 sn süre ile şişirildi. Ardından %30 rezidü kalması üzerine 3.0x15 mm noncomplian balon ile postdilatasyon yapıldı ve TIMI III akım sağlandı. İşlem sonrası çekilen EKG'de ST segment elevasyonunda ve hastanın göğüs ağrısında anlamlı azalma olduğu görüldü. Takibinde hastanın ritminin sinüs ritmine döndüğü izlendi. Hastanın geçici pacemaker ihtiyacının kalmaması nedeniyle pacemaker çıkarıldı. Hasta yatışının 5. gününde şifa ile taburcu edildi.

EKG.

EKG.

KAG.



KAG.

OPS1-17

Acute anterior myocardial infarction after heavy exercise in a young sportsman; importance of intravascular ultrasonography on differential diagnosis

Hamidullah Haqmal, Cengiz Ozturk, Atila İyisoy, Turgay Celik, Mustafa Demir, Ali Osman Yildirim, Sevket Balta

Gülhane Military Medicine Academy, Ankara

We report a 29 year old sportsman with no previous cardiac history who presented with acute retrosternal chest and back pain for 3 hours after heavy exercise was admitted to our coronary care unit. His symptoms began after taking eighty five shuttle. The pain was gradual in onset, sharp and radiates to his left arm resolving with rest. The patient did not have a history of traditional cardiovascular risk factors except smoking (1 pack a day). The electrocardiography (ECG) revealed ST-segment elevation in the precordial, reciprocal changes D1-aVL leads consistent with acute anterior myocardial infarction (MI) on admission. Physical findings were not remarkable. There was significant elevation of the high sensitive troponin T level. Coronary angiography(CAG) showed diffuse thrombus formation in the proximal part of the left anterior desending artery(LAD) with no occlusion. Distal flow was good. There were no significant stenosis on the right coronary artery and circumflex artery. LVEF was 60% and mild hypokinesia of the apical wall of the left ventricle was detected on transthoracic echocardiography so we started the dual anti-platelet therapy (aspirin 300 mg +180 mg ticagrelor as for loading dosage), and unfractionated heparin plus abciximab infusion for 24 hour. Control CAG showed no change in thrombus formation 24 hour later. We decided to perform intravascular ultrasonography (IVUS) study in order to detect the spontaneous coronary artery dissection or atherosclerotic plaque and it showed soft plaque and severe thrombus in the proximal segment of LAD. A 4.0x15 mm balloon was inflated two times in the LAD. The flow was good but there was severe thrombus formation in the proximal part of the LAD with IVUS. We applied 4.0x15 mm bare metal stent to the proximal LAD but IVUS revealed residue thrombus in the lümen. The balloon was inflated with high atmospheric pressure and final imaging of the vessel was so good. The patient was discharged with no complication and segmental wall motion abnormality. In conclusion,high thrombus burden may not cause total occlusion of the coronary vessels. Aggressive antithrombotic treatment is feasible in case of low bleeding risk as in our young case. But, in case of persistant thrombus formation despite aggresive antitrombotic and anticoagulant therapy, stenting should be preferred rather than conservative approach even if the patient is very young.

Figure 1.

OPS1-18

Acute miyocardial infarction following ceftriaxone treatment

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A 39 year old man with the history of hypertension and coronary artery disease was admitted to our emergency department with fever, cough and dyspnea for a week. He was smoker and had family history of coronary artery disease. On admission; his blood pressure was 110/70 mmHg and heart rate was 100 bpm. Electrocardiogram (ECG) was normal (Panel A). The patient was diagnosed as pneumonia and intravenous ceftriaxone was given to patient. After the ceftriaxone administration, the patient developed retrosternal chest pain and redness in the face and occurred cardiopulmonary arrest. His airway was secured, and ventilation was initiated. Simultaneously, cardiopulmonary resuscitation (CPR) was started. After 5 minutes of CPR, the patient had palpable pulse. After that, ECG showed ST elevation in lead II, III and AVF and there was reciprocal ST depression in lead I, AVL (Panel B). Right-sided ECG showed ST elevation in V4, V5, V6 derivations (Panel C). He was immediately taken up coronary anjiography. Coronary anjiography showed miyocardial bridge in middle portion of left anterior descending artery (LAD) and circumflex artery was normal (CX) (Panel D) and showed 70% stenosis of proximal posterior descending artery (PDA) (Panel E). The patient's condition was thought anaphylactic reactions with acute miyocardial infarction. After the procedure, the patient was followed in coronary care unit. Intravenous hydration, antihistamines and corticosteroid was given considering antibiotic-associated allergies. After the procedure, ECG was the same as at the time of admission (Panel F). Within hours, hydration was contiuned. After 24 hours, the patient had spontaneous breathing and he was extubated. Three days after the admission, the patient was discharged as healthy.

OPS1-19

A new complication after MRI procedure in a patient with cardiovascular implanted electronic device; pace pocket infection

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Magnetic resonance imaging(MRI) is the choice of imaging modality as diagnostic tool for many clinical conditions. MRI offers better soft tissue contrast than other imaging modalities without using ionizing radiation. Although many recent publications report that MRI can be performed safely without any clinically significant interaction with cardiovascular implanted electronic devices(CIED), there is still a debate over safety concerns. The patient was 85 years old male with medical history of ischemic dilated cardiomyopathy who has been implanted cardiac pacemaker (Medtronic,VVIR) in 2009 due to symptomatic atrial fibrillation with slow ventricular rate. He presented to outpatient clinic with complaints of swelling and redness around the pace pocket with mild tenderness (Figure 1). 1 month ago he had been found unconscious at home and brain MRI (Siemens Magnetom 1.5 Tesla Scanner) had been performed to rule out cerebrovascular disease without awareness of CIED. The diagnosis of ischemic stroke was made at that time and the pacemaker functions were controlled after realizing that the patient had CIED. The pacemaker was functioning properly and the patient had been discharged with minimal sequela. At presentation, he was afebrile and his vital signs were stable. On physical examination, the pace pocket area was erythematous, tender to touch and swollen with fluctuation. Pacemaker functions were controlled and revealed all values within normal ranges. Blood tests revealed elevated C-reactive protein (CRP) level 17 mg/dl (reference range: 0.2-0.6 mg/dl). Ampiric intravenous(IV) cefazolin 2 grams for every 8 hours were initiated. Pace pocket tissue culture was positive for methicillin susceptible staphylocccus aureus (MSSA). Afterwards all pacemaker systems were removed including the generator and pace leads (Figure 2). Lead tips were also cultured and reported to be negative. The new pacemaker (Medtronic, VVIR) were implanted to the opposite side three days after the initiation of IV antibiotic. Intravenous antibiotherapy with cefazolin was continued during the hospitalization period. The patient was asymptomatic and discharged from the hospital with oral cefuroxime axetil to complete overall 14 days of antibiotherapy. The patient was seen in outpatient clinic one month later. He had no complaints and there was not any sign related to infection around the pace pocket area.

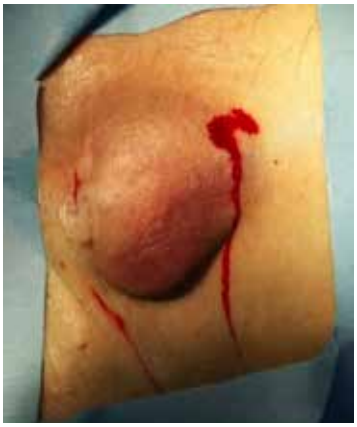

Figure 1. Pace pocket area was erythematous, swollen and tender to touch. Skin integrity was impaired beacuse of heat damage.

Figure 2. Pacemaker(Medtronic, VVIR) generator after complete system removal. Tips of the leads were melted and fused to the generator.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OPS1-20

Detection and management of lead misplacement in a intracardiac defibrillator patient

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Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul

Intracardiac defibrillators (ICD) are indicated for ventricular antitachycardia pacing and defibrillation for life threatening ventricular arrhythmias. ICDs, firstly implanted in 1980s, are with single lead or double lead in addition their half life alternate 5 to 11 years. Here we present an ICD patient admitted to our outpatient clinic for routine control who afterwards diagnosed with lead misplacement. ICD lead which was not placed in the right ventricular wall is one of the reason for preventing optimal pacing and defibrillator therapy. Here we present a 81 year-old male patient implanted VVI ICD (EnTrustD154VRC) secondary to cardiopulmonary arrest 9 years ago. R wave amplitude (ventricle sense) was demonstrated as 1,4 mV in our outpatient clinic control also elective replacement indicator (ERI) was proved. Replacement therapy was offered thus basal electrocardiogram (ECG) was obtained in order to manage replacement procedure. Despite no R wave in V1 derivation on basal ECG, R wave appears on V1 derivation when pacing starts. Misplacement was suspected following all these data and coronary sinus angiography concretized the misplacement in the middle cardiac vein. Pace rhythm of the patient was 200 msn therefore another sense-pace lead was planned to be implanted in the right ventricle. Also in order to prevent vein complications the previous lead was not drawn conversely another lead was placed in the right ventricle to upgrade VVI ICD to CRT. Our case gets attention to detection of lead misplacement which may also appear as ICD defibrillation failure. Pacing electrocardiogram guided us for misplacement fortunately our patient had no need for defibrillation in the near term. Right ventricular last pacing impedance was >3000 ohms which can easily result in defibrillation failure. Our cardiology team tried their best to cope with lead misplacement by implanting another sense-pace lead to the right ventricle.

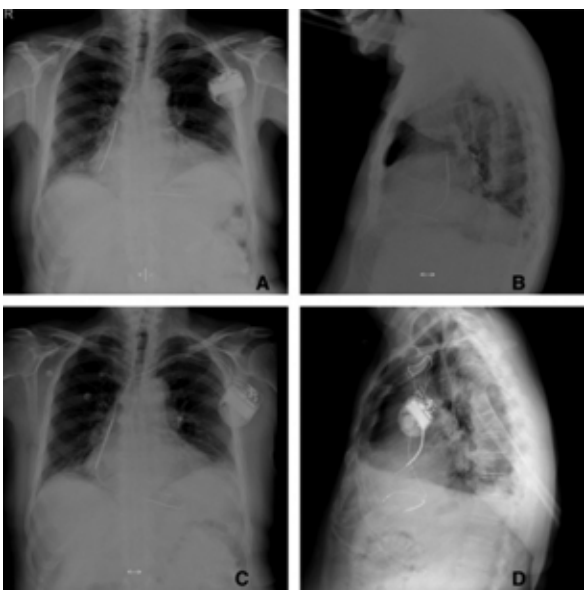

Figure 1. (A, B) Shows Chest x-ray VVI ICD in middle cardiac vein before the procedure, **(C, D)** shows implantation of pace-sense lead to the right ventricle, double leads are seen in Chest x-ray.

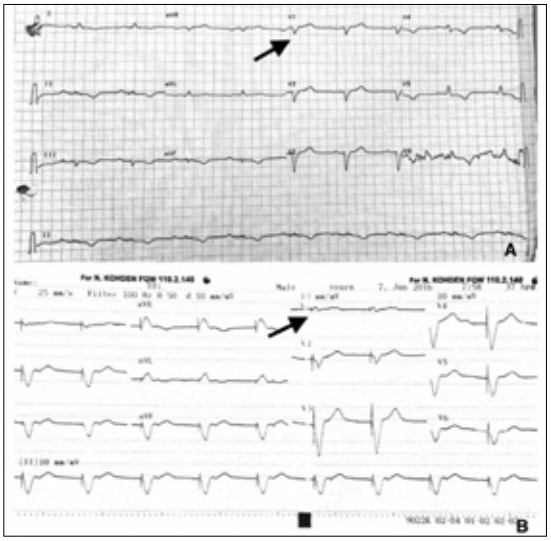

Figure 2. (A) Represents patient's own rhythm as atrial fibrillation, **(B)** represents pace rhythm regarding R wave in V1 derivation.

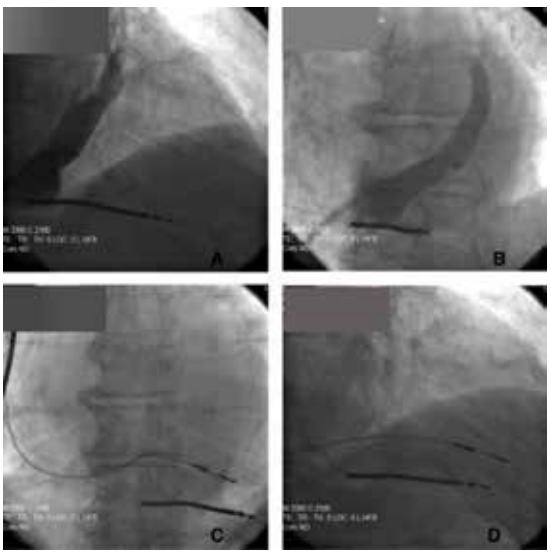

Figure 3. (A, B) Shows coronary sinus angiography showing ICD lead in middle cardiac vein, **(C, D)** showing second lead implanted to the right ventricle.

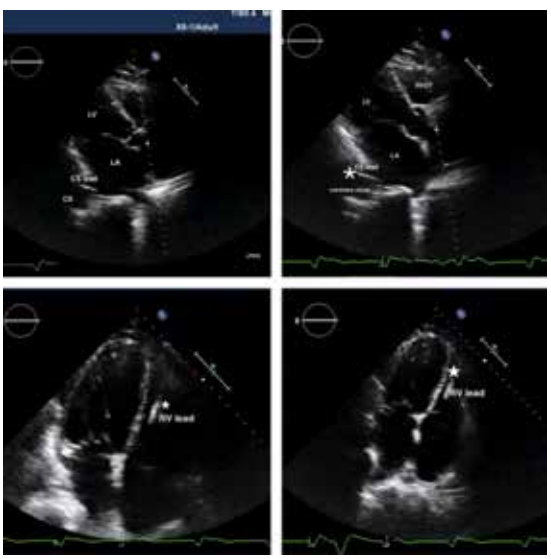

Figure 4. Shows transthoracic echocardiography images of coronary sinus lead and right ventricular lead.

OPS1-21

Hipotermik bir vakada belirgin Osborn dalgası: Unutulmuş bir elektrokardiyografi bulgusu

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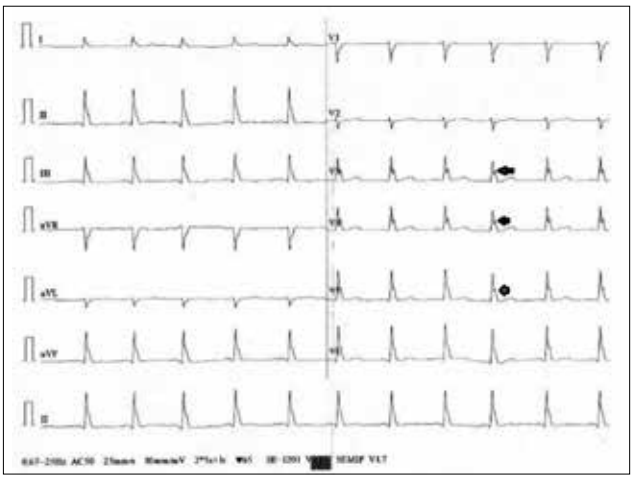
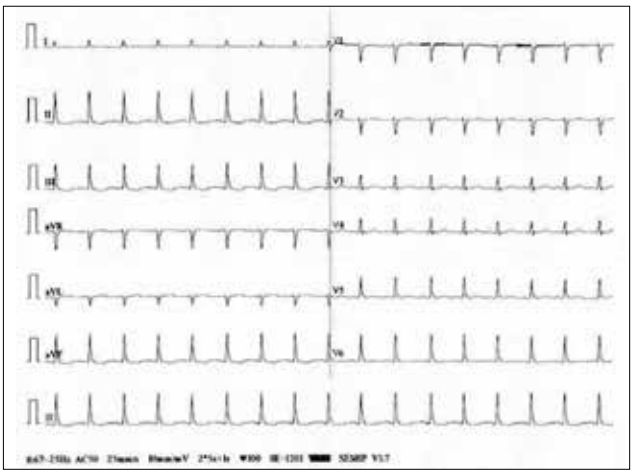
Hipotermi genellikle vücut iç ısısının 35°C nin altında olması olarak tanımlanır. Hipotermi bütün organları etkiler en önemli etkileri kardiyovasküler ve santral sinir sistemi üzerinedir. Hafif hipotermide elektrokardiyogram (EKG) (32-35°C) genellikle normaldir, nadiren Osborn (J) dalgaları saptanabilir. Orta derecede hipotermide, inferiyor ve lateral derivasyonlarda j dalgası belirginleşir, ek olarak PR ve QT aralığında uzama, QRS süresinde artış, P ve T dalgalarının amplitüdünde azalma ve sık supraventriküler aritmiler olur. Şiddetli hipotermide yukarıda belirtilen bulgulara ek olarak tüm derivasyonlarda P dalgası silikleşir, buna karşın J dalgaları ve sık ventriküler aritmiler izlenir. Osborn dalgası QRS kompleksinin sonunda ST segment bileşkesinin başlangıç kısmında iki ardışık atımda küçük pozitif defleksiyon (>1 mm) dalgası olarak tarif edilir. Biz bu olguda iskemik serebrovasküler olay nedeniyle bilinci kapalı olan, soğuk hava nedeniyle hipoterrni gelişen ve EKG'de Osborn dalgaları izlenen bir vaka sunduk. Altmış sekiz yaşında erkek hasta acil servise bilinci kapalı olarak getirildi. İlk değerlendirmede kan basıncı 90/60 mmHg, kalp tepe atımı 62/dakika, vücut ısısı 31°C, solunum sayısı 10/dk ve yüzeyeldi. Glaskow koma skoru 5/15 idi. Nörolojik muayenede Babinski refleksi plantar yanıtı lakayt ve pupiller fiks dilate idi. Bilateral ışık refleksi mevcuttu. Kardiyovasküler muayenede dinlemekle apikal bölgede 2/6 şiddetinde sistolik üfürüm saptandı. Parmak ucundan bakılan kapiller kan şekeri değeri 136 mg/dl idi. Hastanın başvuru 12 derivasyonlu EKG'sinde (Şekil 1) tüm derivasyonlarda belirgin Osborn dalgası, QRS süresinde uzama (140 msn) ve QTc değerinde artış (486 msn) izlendi. Kardiyovasküler resusitasyon, aktif ve pasif ısıtma yöntemleri sonrası yapılan kraniyal bilgisayarlı tomografide beyinde akut yaygın iskemik odaklar saptandı. Hastanın laboratuar incelemesinde beyaz küre sayısı 10800/mm^3, hemoglobin 12.6 g/dl, hemotokrit %37.5, trombosit sayısı 256000/mm^3 idi. Hastanın üre, kreatinin, karaciğer transaminazları, troponin I, potasyum, magnezyum, klor, TSH normal sınırlarda idi. Başvuruda bakılan kan gazı incelemesinde hafif respiratuvar asidoz (pH: 7,35), karbondioksit retansiyonu (PCO2: 46.4 mmHg, PO2: 52.5 mmHg) ve oksijen saturasyonu düşük (%78) olarak saptandı. Dördüncü saatteki rektal vücut ısısı 34 °C olan hastanın EKG'sinde kalp hızı arttı, Osborn dalgalarının amplitüdü ve QTc süresi (451 msn) azaldı. 12. Saatteki ölçülen vücut ısısı 36°C olarak ölçülen hastanın çekilen EKG'sinde Osborn dalgalarının ortadan kalktığı görüldü (Şekil 2). Hastanın takibi süresince nörolojik bulgularında düzelme izlenmedi ve mevcut nörolojik bulgularının kötüleşmesi nedeniyle takibinin 72. saatinde kaybedildi. EKG'de Osborn dalgaları izlenen bilinci kapalı hastalar, hipotermi yönünden araştırılmalıdır. Hekimlerin hipotermiyi ve Osborn dalgalarını erken fark edip, ayırıcı tanı yapmaları hayat kurtarıcı olabilir.



Şekil 1. Hipotermik hastada Osborn dalgaları okla gösterilmiştir.



Şekil 2. Hastanın vücut ısısının artması ile birlikte 12. saatte çekilen elektrokardiyografisinde Osborn dalgaları kaybolmuştur.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OPS1-22

Kalıcı kalp pili implante edilen ve lead revizyonu gereken subklavian ven stenozu gelişmiş nadir bir olgu: Leadin ters taraftan jeneratöre taşınması

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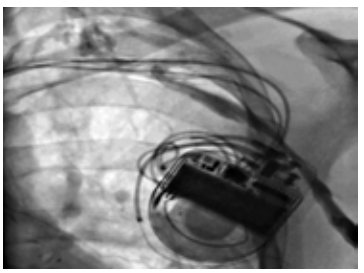
Giriş: Tedavi yöntemlerinde ilerlemelere rağmen kalp yetersizliği (KY) halen tüm dünyada yüksek prevalans, yüksek mortalite ve morbiditeye sahip bir problemdir. Kardiyak resenkronizasyon tedavisi (KRT), ileti kusuru sonucu ciddi senkronizasyon bozukluğu olan KY'li hastalar için vazgeçilmez, etkin bir tedavi yöntemi olmuştur.KRT İmplantasyonunda sağ atriyuma, sağ ventrikül apeksine ve koroner sinüs yoluyla sol ventrikül epikardiyal yüzeyine 3 adet lead yerleştirilmektedir. İmplantasyon oldukça zor olmakla beraber lead sorunu yaşayan hastalarda lead revizyonu daha çok daha güçlükle gerçekleştirilen bir problemdir. KRT lead revizyonu gereken ancak daha önceki işlemde kullanılan subklavian ven tıkanıklığı gelişmiş bir hastada diğer subklavian ven kullanımı ve leadin pace jeneratörüne ters taraftan taşınmasını vaka sunumu olarak hazırladık.

Olgu: 52 yaşında ve iskemik kalp yetersizliği ile takip edilen hasta acil servise implante edilen defibrilatör şoklaması ile başvurdu. Alınan anamnezde hastanın 15 yıl önce koroner bypass operasyonu geçirdiği ve hastaya 8 yıl önce kalp yetersizliği nedeniyle KRT uygulandığı öğrenildi. Pil kontrolü yapılan hastanın sağ ventrikül leadinde empedans artışı tespit edildi ve lead kırığı olduğu düşünülerek leadin değiştirilmesine karar verildi. Hasta lead revizyonu amaçlı kateterizasyon laboratuvarına alındı. İşlem öncesi yapılan sol sistem venografide sol subclavian venin total oklude olduğu izlendi (Video 1, Şekil 1). Sağ ventrikül leadinin plastik cerrahi desteği alınarak sağ subclavian vene yerleştirilmesine ve cilt altından sağ pectoral bölgedeki pace cebine taşınmasına karar verildi. Sağ subclavian ven ponksiyonu yapılarak sağ ventrikül leadi küçük bir insizyon yapılarak sağ pectoral bölgeye yerleştirildi (Şekil 2). Plastik cerrahi desteği ile cilt altından sol pectoral bölgeye taşındı (Şekil 3, 4). Sol pectoral bölgedeki pace jeneratörü serbestleştirildi ve lead jeneratöre implante edildi. Eski sağ ventrikül leadi geri çekilmesine rağmen geri alınamadı ve ucu kesilerek pace cebinde bırakıldı. Ölçümler yapılarak ve görüntü alınarak işleme son verildi (Şekil 5, Video 2).

Tartışma: Transvenöz pacemaker implantasyonu sonrası venöz stenoz görülme sıklığı %20-50 arasında değişmektedir. Stenoz gelişen hastaların büyük çoğunluğu kollateral venöz dolaşım gelişimi sayesinde asemptomatiktir. Venöz stenoz için en önemli risk faktörü işlemde kullanılan lead sayısıdır. Bu nedenle KRT tedavisi işlemde 3 lead kullanılması nedeniyle diğer kalp pillerinden daha risklidir. Lead revizyonu gereken hastalarda tıkalı vene anjioplasti uygulanması ya da cerrahi tedavi ile leadin yerleştirilmesi düşünülebilir. Bu yöntemlerin yanı sıra işlemin diğer subklavian venden yapılarak implnate edilen leadin diğer taraftaki pace cebine taşınması riski daha düşük ve uygulanması daha kolay bir yöntemdir.

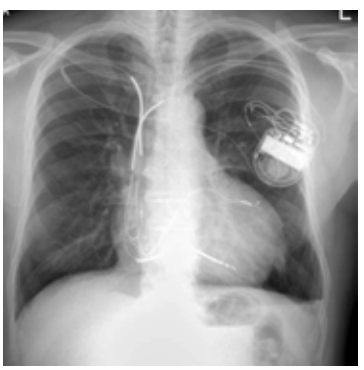


Şekil 1.

Şekil 2.

Şekil 3.

Şekil 4.



Şekil 5.

OPS1-23

Extensive Brugada syndrome

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A 34-year-old previously healthy female with and unremarkable family history was taken to our hospital with sudden cardiac death while sleeping. Clinical anamnesis included that she had nocturnal agonal respiration before sudden cardiac death. After the cardiopulmonary resuscitation, twelve-lead ECG was considered to show no significant ST-segment elevation, and PQ and QT intervals were normal (Figure 1). Transthoracic echocardiography showed a structurally normal heart and her coronary angiography was normal. Laboratory test results were unremarkable, including normal plasma levels of potassium, magnesium, calcium. When the patient had medical therapy in coronary intensive care unit, ventricular fibrillation was developed and she was successfully converted to sinus rhythm with an external defibrillator. After the defibrillation, her electrocardiography showed on V1, V2, D1, aVL and V6 coved type ST segment elevation (Figure 2). With clinical anamnesis and electrocardiographic changes, Extensive Brugada Syndrome was diagnosed.

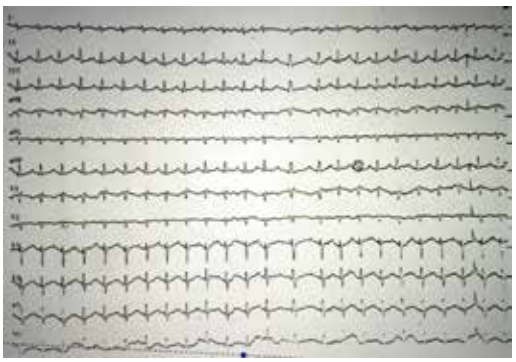

Figure 1. Twelve-lead ECG shows no significant ST-segment elevation, and PQ and QT intervals were normal.

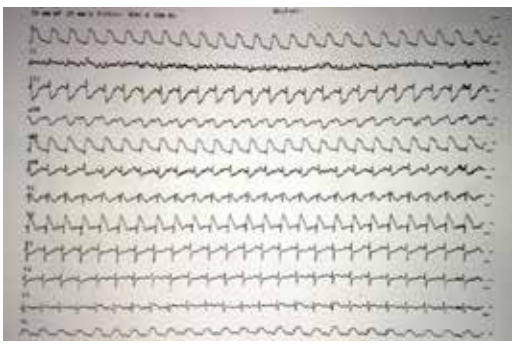

Figure 1. Twelve-lead ECG shows no significant ST-segment elevation, and PQ and QT intervals were normal.

OPS1-24

Can electrical shock provide ST-segment resolution in patients with ST-segment elevation myocardial ınfarction?

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Ventricular fibrillation (VF) occurs less frequently during STsegment elevation myocardial infarction (STEMI), but it is one of the most common causes of the mortality in patients with cardiac arrest during reperfusion therapy. VF were divided into two groups according to occuring time such as periprocedural and postprocedural VF. Underlying mechanisms of reperfusional arrhythmias is not clear. Surely, defibrillation is the definitive treatment of the VF. Pezza argued that there could be a relationship between DC shock and reperfusion in STEMI without any supporting data. In this letter, we discussed this situation with examples in three cases. The baseline characteristics of the patients were shown in Table 1. There was no previous history of coronary artery disease in all patients. First electrocardiography (ECG) samples of all patients were recorded on admission in emergency department. Before the reperfusion therapy, VF was observed in all patients within about 10 min. ECG taken immediately after successful electrical defibrillation with using 12 previous leads in all patients. Prominent ST-segment resolution was observed on the ECG samples of all patients after successful electrical defibrillation (Fig. 1). The use of DC-shock as a therapy to terminate the cardiac arrhythmia is a practice commonly used nowadays. In the event of VF, DC-shock is the only influential method of terminating the rhythm disturbance and restoring a perfusion cardiac rhythm. The relationship between the VF reperfusion discussed in some studies. In a study, reperfusion VF accounted for 22% of VF occurring during the first 48 h after STEMI treated by primary percutaneous coronary intervention. The success of DC-shock is dependent on time and the metabolic state of the myocardial cell. High-intensity fields are produced near the shock electrodes, but they can produce undesirable injury effects in the form of an increased permeability of the cell membrane and arrhythmia. External defibrillation includes delivering a sufficient electrical current flow through the heart, via electrodes implemented to the chest wall, causing simultaneous depolarization of all myocardial cells that are at that moment fully refractory. The effects of electrical shock on cardiac cells is indicated in several studies. Electrical shock can make electrocardiographic changes after cardioversion of ventricular arrhythmias. Eysmann et al. observed the ST segment changes in 60% of patients after cardioversion. In light of these observational data, electrical shock that applied with high energy can lead to fragmantate the thrombus that completely blocking the coronary artery flow. In this way, it can provide reflow of coronary blood partly, in our opinion. So, this situation may result as a ST-segment resolution in patients with STEMI. However, experimental and observational prospective studies are needed to prove this hypothesis.

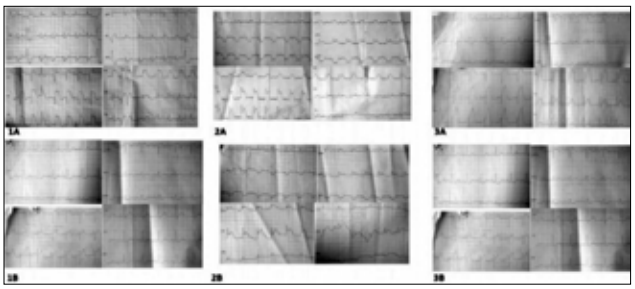

Figure 1. Electrocardiogram samples on admission **(A)** and after defibrillation **(B)** of all three cases (1, 2 and 3).

Table 1. Clinical characteristics of the patients

	Case 1	Case 2	Case 3
Age, years	43	42	53
Gender	Male	Male	Male
Type of MI	Anterior	Anterior	Anterolateral
Risk factor	Smoking	Smoking	Hypertension, hyperlipidemia
Blood pressure, mmHg	130/80	120/80	100/60
Heart rate, beat/min	102	82	80
Infarct-related artery	LAD	LAD	LAD
Duration from admission to DC-shock	5 min	8 min	6 min

DC; direct current, LAD; left anterior descending artery, MI; myocardial infarction.

OPS1-25

Epilepsi tanısıyla takip edilen ani kardiyak arrest

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Konjenital uzun QT sendromu, kalp kası hücre membranındaki iyon kanallarını kodlayan genlerdeki mutasyon sonucu ortaya çıkar ve genç popülasyonda ani kardiyak ölümle sonuçlanabilir. klinik görünüm çoğu kez epilepsi kliniği ile karışır. Biz de epilepsi tanısıyla izlenen 17 yaşındaki bayan hastayı sunuyoruz. Bilinen sistemik hastalık öyküsü olmayan ve 2 yıldır senkop atakları olan hasta, dış merkezde epilepsi ön tanısıyla takipli imiş. Hasta son zamanlarda artan çarpıntı şikayetiyle acil servise başvurdu. Acilde gözetim altında iken ventriküler taşikardi gelişti. Hasta entübe edildi ve kardiyopulmoner resüsitasyon uygulandı. Çekilen EKG'de QTC: 522 msn tespit edildi. Hastada QT uzamasına sebep olabilecek etkenler araştırıldı ve dışlandı. Kardiyoloji yoğun bakımda antiaritmik ilaçlar ile takip edildi. Takiplerinde VT tespit edilmedi. Ekstübe edilen hastaya ICD implante edildi. Beta bloker tedavisi verilen hastanın takibinde VT atağı ve ICD şoklaması izlenmedi. Konjenital uzun QT sendromlu hastalarda, spontan düzelen senkop atakları epilepsi kliniği ile karışmakta ve hastalar sıklıkla epilepsi tedavisi almaktadır. Bu yüzden, genç hastalarda epilepsi tanısı konulmadan önce uzun QT varlığına dikkat edilmelidir.

OPS1-26

Nadir bir olgu, Lyme hastalığına bağlı geçiçi atriyoventriküler tam blok: Tedaviden tanıya

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Afyon Kocatepe Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Afyon

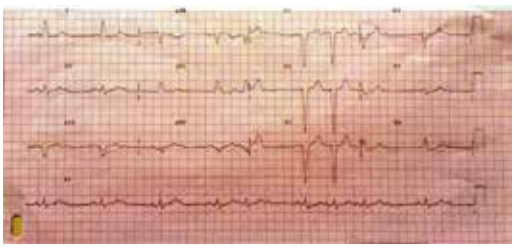
Lyme hastalığı, kene ısırığı ile vücuda giren spiroket (Borrelia Burgdorferi) enfeksiyonun dan kaynaklanmaktadır. Özellikle yaz aylarında kene ısırığı sonrasında eritema kronikum migrans (karakteristik döküntüsü), ateş, atralji ve miyalji ile karakterizedir. Haftalar veya aylar sonra eklem tutulumları, nörolojik veya kardiyak tutulum izler. Vakaların %4-10'unda kardiyak tutulum görülmektedir. Lyme hastalığına bağlı en yaygın kardiyak tutulum atriyoventriküler (AV) düğüm seviyesinde oluşan atriyoventriküler bloklardır. Bazı vakalarda akut miyokardit sonrası sol ventrikül fonksiyonlarını bozabilir ve dekompanse kalp yetersizliği gelişebilir. Akut miyokardit vakalarında diffüz ST segment çökmesi, T dalga anormallikleri ve ventriküler taşikardi atakları görülebilmektedir.

Olgu: Daha öncesinden bilinen herhangi bir kardiyak şikayeti olmayan 64 yaşında kadın hasta son 3 gündür halsizlik, efor dispnesi ve bayılma şikayetleri ile acil servise başvurdu. Hastanın EKG'sinde AV tam blok ve sol dal bloğu tespit edildi (Şekil 1). Ekokardiyografisinde; sol ventrikül sistolik fonksiyonlarında global hipokinezi, orta derece mitral yetersizliği tespit edildi. Hasta koro-

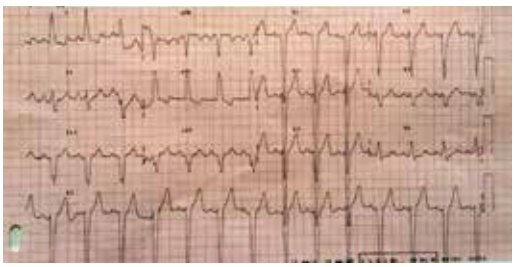
ner yoğun bakım ünitesine yatırıldı. Öyküsünde ateş, halsizlik ve eklem ağrısı şikayeti de mevcut olan hastanın laboratuvarında sedimentasyon değeri 52 mm/sa (normal değeri 1-15 mm/sa) ve high sensitiv CRP değerleri 9.84 mg/dl (normal değeri 0-0.8 mg/dl) olarak tespit edildi. Akciğer grafisinde konsolidasyon ve öksürük şikayetleri olan hasta Enfeksiyon Hastalıkları Bölümüne danışıldı. Hastaya 2 gr seftriakson IV başlanmasına karar verildi. Yatışının 1. haftasın da sedimentasyon ve CRP değerleri normal sınırlara geriledi ve 1. Derece AV blok düzeldi. Sadece sol dal bloğu devam etti. Lyme ön tanısı ile serolojik test (Eliza) için gönderilen serumda Lyme IG G'si pozitif olarak tespit edildi. Hasta 1 ay sonra tekrar baş dönmesi şikayetleri ile geldi. EKG de Mobitz Tip 2 AV Blok gözlendi. Hasta Enfeksiyon Hastalıkları ile tekrar değerlendirildi ve 4 haftalık seftriakson tedavisi başlandı. Tedavinin 1. haftasında Mobitz Tip 2 Blok düzeldi ve sinüs ritmi elde edildi. Komplet sol dal bloğu devam etti.

Tartışma: Kalbide etkileyebilen Lyme hastalığı tüm dünyada ve Türkiye de sık görülen bir hastalıktır. Ancak hastalık hakkında kardiyologların farkındalığı fazla yoktur. Dejeneratif ve iskemik olmayan AV tam bloklu hastaların tıbbi öyküsü alınır iken, herhangi bir ilaç kullanımı, deli bal yeme veya bitkisel ilaç içme öyküsü sorgulanmakta ise de Lyme hastalığı akla gelmemektedir. Lyme hastalığının tanısı, iyi bir anamnez ve ilk 4-8 hafta negatif olabilen serolojik testlere dayanmaktadır. Lyme karditli vakaların tedavisi henüz netlik kazanmamıştır. Bazı vakalarda tedavi almasa dahi hastalık kendiliğinden sınırlanabilir ve iyileşebilmektedir.

Sonuç: Özellikle öyküsünde ateş, eklem ağrısı ve halsizlik şikayeti bulunan ve AV tam blok ile başvuran hastalarda lyme karditi ön tanılar arasında yer almalıdır.



Şekil 1. Atriyoventriküler blok.



Şekil 2. Tedavi sonrası EKG'si.

OPS1-27

Succsessful implantable cardioverter defibrilator (ICD) lead insertion in a patient with occluded superior vena cava

Murat Ünlü, Ali Osman Yıldırım, Sait Demirkol, Zekeriya Arslan, Mustafa Demir, Atila İyisoy

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Introduction: Procedure of subcutaneous devices implantation includes incision of skin,insertion of sheat through subclavian vein, advanced of lead/s through sbclavian vein into right ventricle then connection of lead and device and pocked of device under the skin. We report succsessful ICD lead insertion in a patient with occluded superior vena cava.

Case: A 65 years-old female with histories of chronic hemodialysis for 12 years and central jugular vein cathater for 2 years and heart failure with reduced ejection fraction is admitted our hospital for ICD implantation. The patient is admitted to electrophysiology laborotory, skin incision and sheat insertion via left subclavian vein was performed then ICD lead was not advanced throuhg superior vena cava. Angiography of vena cava superior revealed occluded superior vena cava with little penetration of contrast media in distal of the vesssel (Video 1, Figure 1). 0.014 inch guidewire was crossed to superior vena cava (Figure 2) then coronary sinus cathater was advanced over the wire (Figure 3). ICD lead was inserted to right ventricle via coronary sinus cathater (Figure 4). There was no complication and ICD was succsessfully implanted.

Discussion: In some condition,such as occluded vena cava superior 0.014 inch guidewire and coronary sinus catheter are used for lead insertion.

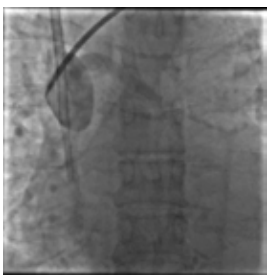

Figure 1.

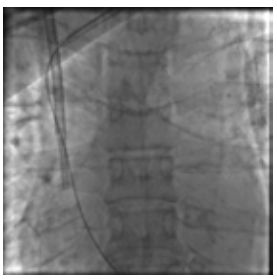

Figure 2.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

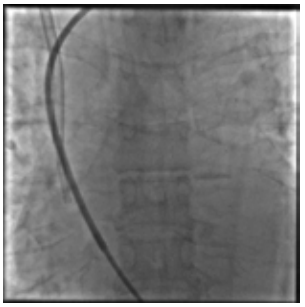

Figure 3.

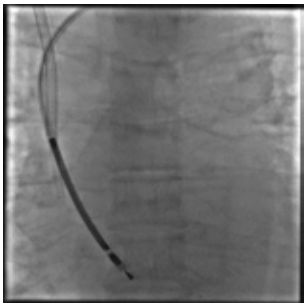

Figure 4.

OPS1-28

A case of repetitive inappropriate implantable cardioverter-defibrillator therapies due to setting wavelet algorithm to monitor

Serdar Bozyel[1], Müjdat Aktaş[2], Ayşen Ağaçdiken Ağır[2], Ahmet Vural[2], Umut Çelikyurt[2]

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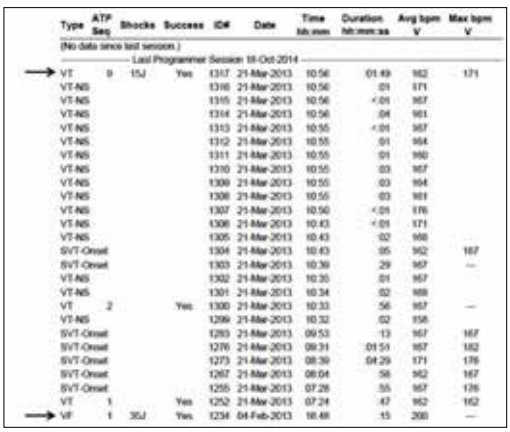
Implantable cardioverter defibrillators (ICDs) are increasingly being used for primary and secondary prevention of sudden cardiac death (SCD) in patients who are predisposed to malignant ventricular arrhythmias. Inappropriate therapy delivery remains the most frequent complication in patients with ICDs, resulting in psychological distress, proarrhythmia and battery life reduction. The WaveletTM morphology discrimination (MD) algorithm (Medtronic Inc., MN, USA) significantly reduced inappropriate detection of supraventricular tachycardia (SVT) as ventricular tachycardia (VT) in single-chamber (SC) ICDs. We present the case of a 73-year-old man with a single-chamber ICD who received an inappropriate antitachycardia pacing (ATP) and shock therapies due to setting wavelet algoritm to monitor. This case provides an example of how inappropriate ICD programming in patients with SVT could cause a problem.



Type	ATP Seq	Shocks	Success	ID#	Date	Time hh:mm	Duration hh:mm:ss	Avg bpm V	Max bpm V
(No data since last session.)									
Last Programmer Session 18-Oct-2014									
VT	9	15J	Yes	1317	21-Mar-2013	10:56	01:49	162	171
VT-NS				1316	21-Mar-2013	10:56	:01	171	
VT-NS				1315	21-Mar-2013	10:56	<:01	167	
VT-NS				1314	21-Mar-2013	10:56	:04	161	
VT-NS				1313	21-Mar-2013	10:55	<:01	167	
VT-NS				1312	21-Mar-2013	10:55	:01	164	
VT-NS				1311	21-Mar-2013	10:55	:01	160	
VT-NS				1310	21-Mar-2013	10:55	:03	167	
VT-NS				1309	21-Mar-2013	10:55	:03	164	
VT-NS				1308	21-Mar-2013	10:55	:03	161	
VT-NS				1307	21-Mar-2013	10:50	<:01	176	
VT-NS				1306	21-Mar-2013	10:43	<:01	171	
VT-NS				1305	21-Mar-2013	10:43	:02	168	
SVT-Onset				1304	21-Mar-2013	10:43	:05	162	167
SVT-Onset				1303	21-Mar-2013	10:39	:29	167	---
VT-NS				1302	21-Mar-2013	10:35	:01	167	
VT-NS				1301	21-Mar-2013	10:34	:02	169	
VT	2		Yes	1300	21-Mar-2013	10:33	:56	167	---
VT-NS				1299	21-Mar-2013	10:32	:02	156	
SVT-Onset				1283	21-Mar-2013	09:53	:13	167	167
SVT-Onset				1276	21-Mar-2013	09:31	:01:51	167	182
SVT-Onset				1273	21-Mar-2013	08:39	:04:29	171	176
SVT-Onset				1267	21-Mar-2013	08:04	:58	162	167
SVT-Onset				1255	21-Mar-2013	07:26	:55	167	176
VT	1		Yes	1252	21-Mar-2013	07:24	:47	162	162
VF	1	35J	Yes	1234	04-Feb-2013	16:48	:15	200	---

Figure 1. Device interrogation data showing a ventricular tachycardia (VT) episode. Device interrogation data showing a ventricular tachycardia (VT) episode that was treated with nine consecutive antitachycardia pacing (ATP) therapies and 15J shock (indicated by upper arrow). The lower arrow indicated ventricular fibrillation (VF) episode that was treated with ATP therapy and 35J shock.

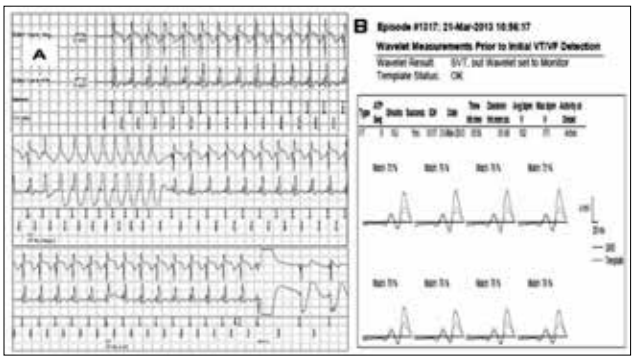


Figure 2. Stored electrograms (EGMs) and marker channel of rapid ventricular tachycardia (VT). **(A)** Stored electrograms (EGMs) and marker channel of rapid ventricular tachycardia (VT) detected in the VT zone. VT episode was initially treated unsuccessfully with consecutive antitachycardia pacing (ATP) therapies and then terminated with an 15J shock. **(B)** As the matching score was higher than the threshold (70%), the Wavelet morphology detected this tachycardia EGM as supraventricular tachycardia. But, since the Wavelet algorithm was set to only monitor, the device failed to discriminate false VT episodes and delivered ATP and shock therapies.

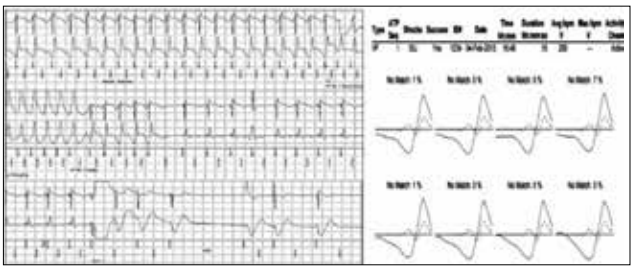


Figure 3. The intracardiac electrogram showing ventricular fibrillation (VF) episode treated by the antitachycardia pacing (ATP) and shock therapies. The intracardiac electrogram showing ventricular fibrillation (VF) episode treated by the antitachycardia pacing (ATP) and shock therapies. It was a regular tacyhcardia at a cycle length of 280-380 ms and the average ventricular rate was 200 bpm. The Wavelet morphology detected this tachycardia EGM as true VF (the matching scores was below a programmed threshold (nominal 70%).

OPS1-29

Katekolaminerjik polimorfik ventrikül taşikardisi olgusu

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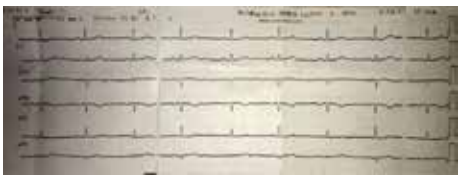
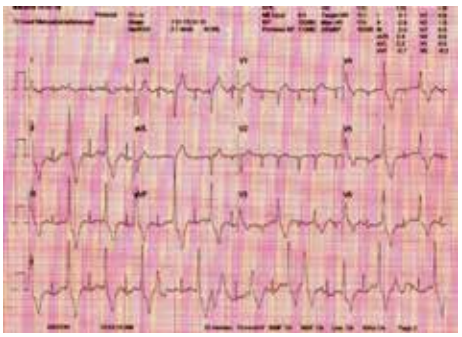
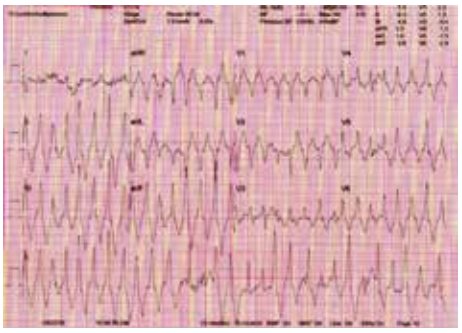
Bilinen sistemik hastalık öyküsü bulunmayan 38 yaşında bayan hastanın oğlu tekrarlayan senkop atakları olması üzerine çocuk kardiyoloji hastalıkları tarafından katekolaminerjik polimorfik ventriküler taşikardi tanısı konmuş ve ICD implantasyonu uygulanmış. Tarama için refere edilen hasta değerlendirildi. Hastanın senkop öyküsü yok belirgin çarpıntı ve angina şikayeti bulunmamaktaydı. Hastanın bazal EKG'si normal olarak izlendi (Şekil 1) (Normal sinüs ritmi f: 80vuru/dk, QTc değeri 0.34 msn). Ekokardiyografisinde sol ventrikül çap ve duvar hareketleri normal, ejeksiyon fraksiyonu %65 ve mimimal mitral yetersizlik dışında özellik saptanmadı. Holter değerlendirmesinde temel ritim sinüs ortalama kalp hızı 70 v/dak bradikardi pause yok nadir ves nadir sves izlendi. Bruce protokolü ile yapılan efor testi evre 2'nin 2. dakikasında sonlandırıldı. Hastanın efor testi başlangıcından itibaren ventriküler erken vurular gözlendi (Şekil 2). Evre 2'den itibaren polimorfik karakterde ventriküler taşikardi (VT) atağı görüldü (Şekil 3). Hastada yapılan genetik araştırmada ryanodin reseptör mutasyonu (PYR2) homozigot olarak tespit edildi. Hastada saptanan gen mutasyonu ve efor testinde görülenventriküler erken vurular, polimorfik VT atağı neticesinde hastaya katekolaminerjik polimorfik VT tanısı konuldu. Hastada tekrarlayan senkop ve çarpıntı atağı olmaması nedeni ile beta bloker ile takibe alındı.



Şekil 1. Bazal EKG.



Şekil 2. Efor testi, Evre I, VES'ler.



Şekil 3. Efor testi, Evre II, 2. dakika: Polimorfik VT.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OPS1-30

The first case of hypercalcemia mimicking NSTEMI and having long QTc interval unlike literature

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Introduction: Non-ST-elevation myocardial infarction (NSTEMI) is one of the acute coronary syndromes (ACS) requiring early diagnosis and treatment. But in fact, lots of diseases can mimic NSTEMI. Electrolyte abnormalities are one of the causes of electrocardiogram (EKG) changes imitating ACS. Particularly potassium, calcium, and magnesium abnormaities are the most common cause of these EKG changes. Hypercalcemia is an elevation of serum calcium levels above 10.5 mg/dl and above 14 mg/dl is accepted severe hypercalcemia. Review of literature suggested that hypercalcemia causes shortening of the QT interval, prolongation of PR interval, AV block, QRS widening, and changes in polarity and amplitude of T wave. It was demonstrated by case reports that hypercalcemia can cause ST segment elevation (especially in precordial leads), atrial fibrillation, Brugada like ECG changes, left bundle branch block and J wave (Osborn wave). Here we reported a case of severe hypercalcemia presenting with ST segment depression, long QTc interval and J wave in some leads on ECG with high troponin levels.

Case Report: A 61 year old woman was send to the emergency department as NSTEMI. Complaining of patient was dyspnea, epigastric pain, nausea, vomiting, and fatigue for the past week. The patient had no coronary artery disease risk factors and malignant tumor. On physical examination, the patient had a temperature of 37.1°C, with a heart rate of 90 bpm, a blood pressure of 100/60 mmHg. No rales, wheezing sound, cardiac murmurs and rubs were heard. ECG showed normal sinus rhythm with marked ST depression in leads V3 through V6 and QTc of 554 milliseconds, and J wave in limb leads (Fig. 1, 2). Serum creatinine, sodium, potassium and chloride were normal, first troponin level was 417 pg/mL and second was 795 pg/mL. Her detailed laboratory results are shown in Table 1. An transthoracic echocardiogram was done, demonstrating an ejection fraction of 60% to 65% without any segmental wall motion abnormalities, and there no significant valve disease. Then we decided to perform coronary angiography and it revealed all coronary arteries free of any stenosis but there was slow coronary flow. Afterwards detailed clinic and laboratory eveluation was done and severe hypercalcemia was found.

Discussion: Severe hypercalcemia can cause lots of ECG changes and there was a lot of different case reports about this ECG findings in literature. Hypercalcemia related QT interval shortening, PR interval prolongation, AV block, T wave changes, ST segment elevation, atrial fibrillation, Brugada like ECG findings, and J wave (Osborn wave were some of them. And there was only a case report pressenting with QTc prolongation and that case had different electrolyte abnormalities in addition to hypercalcemia which can cause QTc prolongation. To our knowledge, this is the first case report of hypercalcemia mimicking NSTEMI and had QTc prolongation, ST depression and Jwave on ECG.

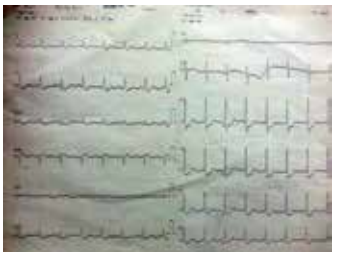

Figure 1.

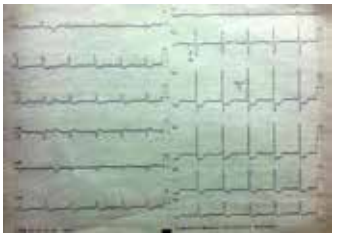

Figure 2.

Table 1. Laboratory evaluation on admission and follow up

Test	Result	Reference Range
Sodium	134 mmol/L	135-148 mmol/L
Potassium	3,8 mmol/L	3.5-5.4 mmol/L
Chloride	92,8 mmol/L	95-110 mmol/L
Phosphorus	4,2 mg/dL	3,5-4,5 mg/dL
PH	7,39	-
pCO2	44 mmHg	-
BUN	64 mg/dL	6-23 mg/dL
Creatinine	1,17 mg/dL	0,5-0,9 mg/dL
Glucose	104 mg/dL	70-110 mg/dL
Albumin	2,69 g/dL	3.4-5.5 g/dL
Calcium	21,1 mg/dL	8.4-10.5 mg/dL
Corrected Calcium	22,15 mg/dL	4-5,6 mg/dL
Parathyroid hormone	21,5 pg/mL	12-88 pg / mL
Vitamin D Total (25-OH)	3 ng/mL	30-60 ng/mL
Troponin T-1	417 pg/mL	0-100 pg/mL
Troponin T-2	795 pg/mL	0-100 pg/mL
Troponin T-3	1586 pg/mL	0-100 pg/mL
TSH	0,48 µIU	0,27-5,6 µIU
Free T4	1,1 ng/dL	0,93-1,7 ndL
CRP	258 mg/dl	0-5 mg/dL
Sedimentation	92	-
WBC	18,1 (%85 Neu) 103 /mL	4,49 – 12,68
HGB	12,5 gr/dL	11,9-14,6 gr/dL
Platelet	255 103 / µL	173-389

OPS1-31

Brucella enfeksiyonuna bağlı kalıcı kalp pili leadinin cerrahi olarak çıkarılması

Halil Fatih Aşgün[1], Burak Altun[2], Tolga Kurt[1], Bahadır Kırılmaz[2]

[1]Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Kalp ve Damar Cerrahisi Anabilim Dalı, Çanakkale
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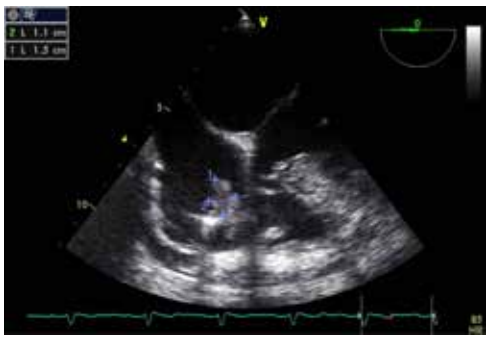
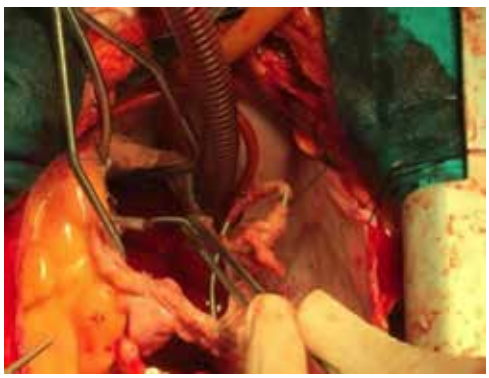
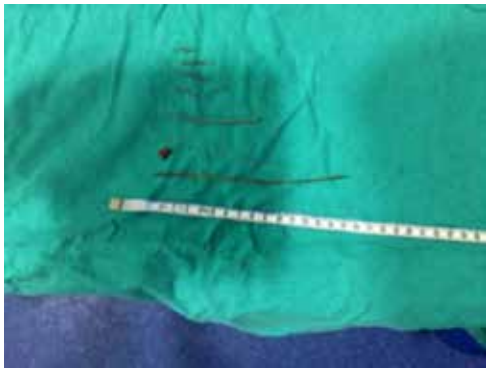
Kalıcı pacemaker (PM) ve yerleştirilebilen kardiyak defibrilatörler (ICD) lerin kullanımı son yıllarda giderek artmıştır ve bu cihazların enfeksiyonu ciddi mortalite ve morbiditeyle seyretmektedir. Kardiyak cihazlar ile ilişkili kan kültürleri hastaların %77'sinde pozitiftir. Stafilokoklar en yaygın patojenlerdir. İkinci sıradaki patojen ajanlar gram negatif basillerdir 2002 yılında DDD PM yerleştirilen yetmiş dört yaşındaki kadın hastanın PM bataryası 2010 yılında değiştirilmiş ve 2015 yılında PM cep enfeksiyonu sonrası, bataryanın ciltten protrüde olduğu görüldü.Kan kültüründe brusella enfeksiyonu saptanan hastada PM 2015 yılı Eylül ayında evolution lead ekstraksiyon cihazı ile mekanik dilatasyon denendi, fakat leadlerin 10 yıldan uzun süreli ve aşırı yapışık olmasından dolayı başarılı olunamadı. Hastanın PM bağımlı olmadığı gözlendi ve bataryası çıkarılarak takip kararı alındı, enfeksiyon hastalıklarının tedavi önerisiyle taburcu edildi. Rutin kontrollerde brusella titresi düşen hastanın yapılan son kontrol ekokardiyografisinde sağ ventrikül leadinde vegetasyon? trombüs? gözlenmesi üzerine transözofajial ekokardiyografi yapıldı ve vegetasyon düşünülerek cerrahi önerildi. Hasta Kalp damar cerrahisi tarafından işleme alındı ve başarılı olarak leadler çıkarıldı. PM cep enfeksiyonu risk faktörlerinin varlığında daha sık gelişmekte, komplike olarak uzun süre antibiyotik kullanımı ve cerrahi tedavi gerektirmektedir. PM ve ICD yerleştirme planlanan hastaların seçimi ve takibi oldukça önemlidir. Yerleştirme sonrası gelişen elektrot sorunları nedeniyle elektrot çıkarılması gereken hastalarda mekanik elektrot çıkarma sistemleri gittikçe daha sıklıkta kullanılmaktadır. Mekanik cihazlarla çıkartma yapılamadığında cerrahi olarak çıkarma işlemi yapılmalıdır. Elektrot çıkarılmasının deneyimli işlemciler tarafından, kalp damar cerrahisi bulunan, olgu sayısı ve deneyimi yeterli merkezlerde yapılması işlem başarısı ve komplikasyonların azaltılması açısından son derece önemlidir.



Şekil 1. TEE de vegetasyon.



Şekil 2. Ventriküler lead üzerinde vegetasyon.



Şekil 3. Pacemaker elektrotları.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OPS1-32

Parahisyan prematür ventriküler kompleksin kryo ablasyon yöntemi ile başarılı bir şekilde tedavi edilmesi

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Semptomatik prematür ventriküler komplekslerin (PVK) tedavisinde medikal tedavi ile birlikte kateter ablasyon yöntemleri kullanılmaktadır. Kateter ablasyon tercihlerinde ise çoğunlukla radyo frekans (RF) ablasyon kullanılırken, kryo ablasyon yöntemi nadir olarak tercih edilmektedir. His bölgesinden kaynaklanan PVK'lar nadir olsa da karşımıza çıkabilen ventriküler aritmilerdir. Bu bölgedeki PVK'ların ablate edilmesi durumunda yüksek oranda tam atriyo ventriküler (AV) blok riski bulunmaktadır. Ancak, bu bölge aritmilerinin tedavisinde daha az oranda ve geri dönebilen AV blok riski oluşturabilmesi sebebiyle RF yerine kryo ablasyonun kullanılması bize önemli bir seçenek sunabilmektedir. Biz burada medikal tedaviye dirençli olan ve günlük total atım sayısının yaklaşık %25'ini oluşturan parahisyan PVK'lı bir hastanın kryo ablasyon ile başarılı bir şekilde tedavi edilmesini sunmaktayız.

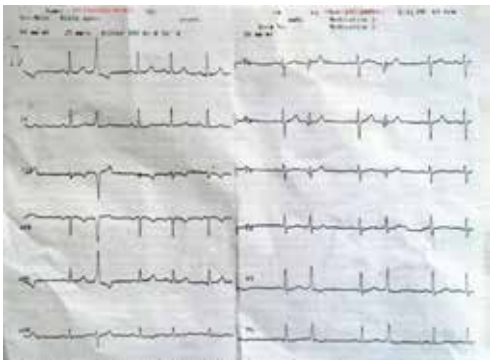

Figure 1.

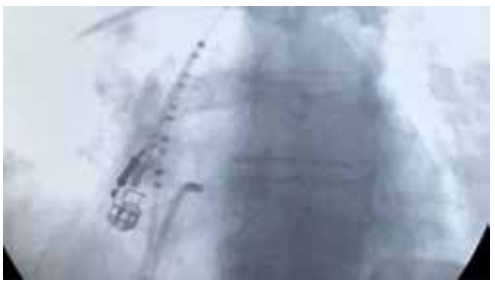

Figure 2.

OPS1-33

Markedly long QT interval with hypocalcemia without peripheral signs

Serdar Demir, Emrah Acar, Servet İzci, Mehmet Fatih Yılmaz, İsmail Balaban, Dilber Üren

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A 63-year-old female patient was admitted to emergency service with palpitation and presyncope. Her past medical history was unremarkable except than hypertension. Her medication regimen included only telmisartan 80 mg daily. On clinical examination the patient appeared anxious and in mildly impaired general condition. Her blood pressure was 80/40 mmHg. Pulse rate was regular with a rate of 75. Electrocardiogram revealed a sinus rythm with a rate 78 bpm and markedly prolonged QTc interval of 730 msn (Figure 1). Her echocardiographic evaluation showed a normal left systolic ejection fraction of 68%, mild mitral regurgitation and mild tricuspid regurgitation. Initial laboratory tests were remarkable for hypocalcemia with a calcium serum level of 3.4 mg/dL or 4.8 when corrected for albumin levels (2.6gr/dL). Serum potassium was 3.5 mg/dL, serum magnesium was 1.9 mg/dL, and parathormone levels were 11.3 pg/dL (normal values 15–65). Interestingly there were no peripheral signs of hypocalcemia such as paresthesia, muscle cramps and tetani. Also the Chvostek and Trousseau signs were negative. The patient's assessment throughout hospitalization included, among others, coronary angiography which did not reveal obstructive coronary artery disease. After endocrinologic assessment about hypoparathyroidism, it was named idiopathic hypoparathyroidism. The patient's management consisted of intravenous infusion of calcium gluconate until normalization of serum calcium levels as well as potassium and albumin repletion. The patient was discharged 7 days after admission in good condition. The pre-discharged ECG showed a sinus rythm with a QTc of 440 msn (Figure 2). Hypocalcemia prolongs the duration of the plateau phase (phase 2) of then cardiac action potential, so characteristic ECG manifestations of hypocalcemia are prolongation of the QT interval as a result of lengthening of the ST segment. Hypocalcemia may be associated with a spectrum of clinical manifestations, ranging from few if any symptoms if the hypocalcemia is mild to life-threatening seizures, refractory heart failure, or laryngospasm if it is severe. Among the symptoms of hypocalcemia, tetany, papilledema, and seizures may occur in patients who develop hypocalcemia acutely.Interestingly,in our case the patient presented with only cardiologic manifestation of hypercalcemia and that is unusual. So, it should be remembered that even if there are no clinical signs of hypocalcemia in a patient with hypocalcemia, a detailed electrocardiographic assessment can reveal dangerous situations such as QT interval prolongation.

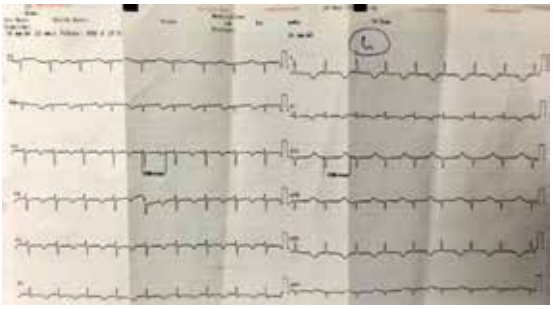

Figure 1.

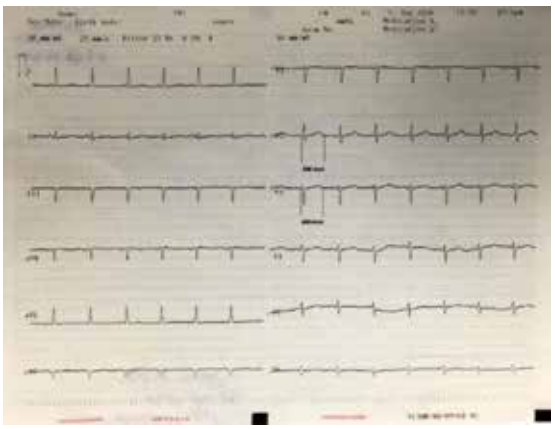

Figure 2.

OPS1-34

A case of paroksismal atrial fibrillation in a young healty man after carbon monoxide poisoning

Şahin Avşar, Yavuz Uğurlu

Department of Cardiology, Ağrı State Hospital, Ağrı

Objective: Carbon monoxide (CO) is an odorless, colorless, tasteless, non-irritating gas formed as a by-product of burning organic compounds. The most sensitive tissue to hypoxia are: the central nervous system and the cardiovascular system which require continuously large amounts of oxygen for normal function. Carbon monoxide poisoning has a variety of deleterious cardiac effects including arrhythmias, coronary spasm, ventricular dysfunction and myocardial infarction.

Case Report: A 37-year-old male patient with no preexisting disease was brought to the emergency unit for palpitation after exposure to CO. Initial ekg revealed atrial fibrillation with heart rate of 132. Echocardiography and cardiac markers were normal. 12 hours later his rhythm was spontaneously reversed sinus rhythm after hyperbaric oxygen treatment.

Conclusion: We report a CO case complicated by atrial fibrillation which was spontananeously resolved after hyperbaric oxygen tretment. This case highlights an important issue that if this patient had many risk factors for stroke should we start anticoagulation for paroksismal atrial fibrillation. Novel strategies are needed to address this important problem among patients who have repeated exposure to CO.

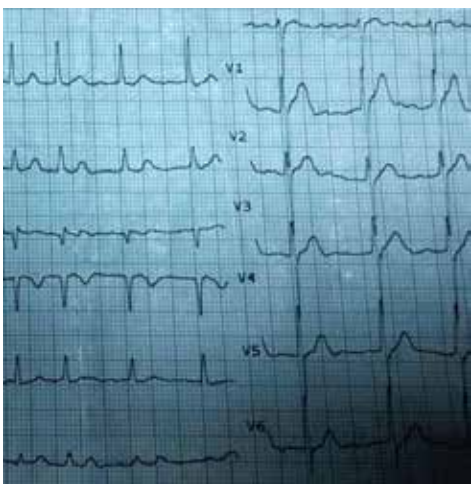

Figure 1. Initial EKG.

Aritmi / Elektrofizyoloji / Pacemaker / CRT-ICD

OPS1-35

The importance of venography during permanent pacemaker implantation

Mustafa Ahmet Huyut, Asım Enhoş, Erdem Karaçöp, Ahmet Bacaksız, Muharrem Nasifov, Aydın Nadir, Özge Özden Tok, Mehmet Ergelen, Abdülkadir Yıldız, Nuray Kahraman Ay, Ömer Göktekin, Hüseyin Uyarel

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A 65-year-old female admitted to our emergency service with complaint of new onset syncope. The patient's electrocardiography was consistent with third-degree atrioventricular block. Coronary angiography was performed and we found normal coronary arteries and implant temporary pacemaker. All laboratories values include thyroid function tests were normal. In patient's past history she did not use any drug. We decide to implante permanent pacemaker without making venous angiography we attempt to make left subclavian vein puncture but we could not be successfull. So when we made a left cubital vein venography we saw that left subclavian vein was rudimenter and had a minimal luminal area (Movie 1, Figure 1). Then we change our site to the right (Movie 2) but the worst was that we had cut the skin and opened pacemaker pocket. According to this case we advice while starting a pacemaker implantation at first to make a cubital vein venography then try to make a puncture. During the ten months follow up period, patient has no complaint.

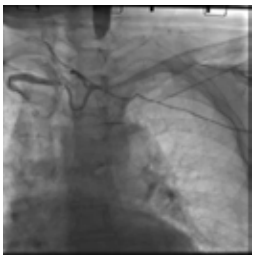

Figure 1.

Pulmoner hipertansiyon / Pulmoner vasküler hastalık

OPS1-36

Kronik myeloid lösemili hastada dasatinib kullanımına bağlı pulmoner hipertansiyon

Mustafa Yenerçağ[1], Murat Meriç[2], Metin Çoksevim[2], Korhan Soylu[2], Ömer Gedikli[2], Mahmut Şahin[2]

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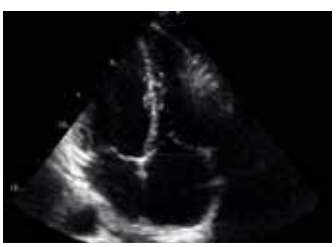
Giriş: Pulmoner hipertansiyon ortalama pulmoner arter basıncının 25 mmHg'nin üzerine çıktığı patolojik durumdur. Pulmoner arteriyel hipertansiyon (PAH) ise prekapiller pulmoner HT tipi olup, farklı etiyolojik nedenlere bağlı ortaya çıkan, pulmoner vasküler direncin yükselmesi ile karakterize bir durumdur. Dasatinib, tirozin kinaz inhibitörüdür ve kronik myelositik lösemide kullanılmaktadır. Biz kronik myelositik lösemi (KML) tedavisinde dasatinib kullanımına bağlı olarak PAH gelişen ve oral ambrisentan tedavisi verilen olguyu sunduk. Bu olgu, kliniğimizin bu konuda başarı ile tedavi ettiği ikinci vakadır.

Olgu: Kırk dokuz yaşında erkek hasta, progresif artan nefes darlığı ve bacaklarda şişlik nedeniyle kliniğimize başvurdu. Beş yıl önce KML tanısı konulan hastaya 3 yıl önce kemik iliği nakli yapılmış. Nakil sonrası 3 yıl süre ile dasatinib tedavisi almakta olan hastaya yapılan ekokardiyografide; sol kalp boyut ve fonksiyonları normal idi. İnterventriküler septumda düzleşme ve sağ kalp boşluklarında belirgin genişleme (Sağ ventrikül bazal çapı 39 mm, sağ atriyal alan 21 cm^2) saptandı (Şekil 1). TAPSE 18 mm, sistolik pulmoner arter basıncı 95 mmHg ölçüldü. Hastanın altı dakika yürüme testi 290 metre idi. Hastaya yapılan sağ kalp kateterizasyonunda ortalama pulmoner arteryel basınç 45 mmHg, pulmoner kapiller kama basıncı 5 mmHg, kardiyak debi 4.2 L/dk, pulmoner vaskuler direnç 10.5 wood ölçüldü. İloprost ile yapılan vazoreaktivite testi negatif idi. Fonksiyonel kapasitesi NYHA sınıf III olan hastanın dasatinib tedavisi kesildi, ilaca bağlı PAH tanısıyla intravenöz diüretik ve iv. iloprost tedavisi verildi. Takiplerimizde altı dakika yürüme testi 354 metreye yükselen ve ödemleri gerileyen hastaya oral ambrisentan ve diüretik tedavi ile taburcu edildi. 6 ay sonra kontrole gelen hastanın Fonsiyonel kapasitesi NYHA sınıf II, altı dakika yürüme testi 398 metre, yapılan transtorasik ekokardiyografisinde; sağ kalp genişliği azalmış (Sağ ventrikül bazal çapı 35 mm, sağ atriyal alan 20 cm^2), TAPSE 21 mm, sistolik pulmonar arter basıncı 70 mmHg ölçüldü (Şekil 2). Ambrisentan tedavisi altında takibimiz devam etmektedir.

Tartışma: KML tedavisinde tirozin kinaz inhibitörleri sık kullanılmaktadır. Dasatinib ilaçla tetiklenen PAH'un olası etkenleri arasındadır. İlaçla tetiklenen PAH da ilacın kesilmesi öncelikli tedavi olup klinik olarak iyileşmeyi hızlandırmak için endotelin reseptör blokeri kullanılabilir. Myeloproliferatif hastalıklarda dasatinib başta olmak üzere tirozin kinaz inhibitörleri kullanılırken PAH yapabileceği akılda tutulmalı, ekokardiyografi ile kontrol edilmelidir.

Şekil 1. İlk başvurudaki sağ ventrikül çapı.



Şekil 2. Altı ay sonraki sağ ventrikül çapı.

OPS1-37

Pulmoner hipertansiyon ayırıcı tanısında pulmoner darlık

Fatih Mehmet Uçar, Zehra Akmaz, Çağlar Kaya, Mustafa Adem Yılmaztepe

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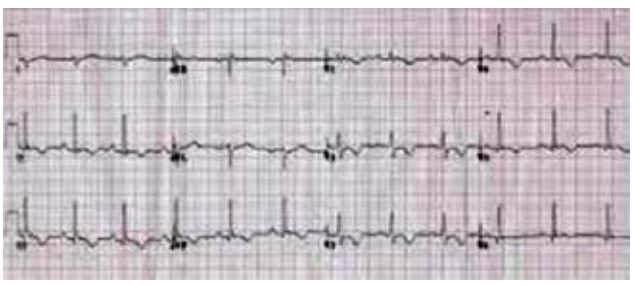
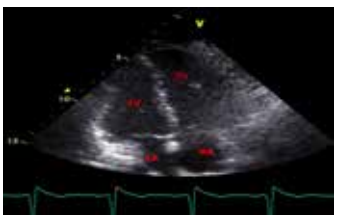
Giriş: Pulmoner arteryel hipertansiyon (PAH), pulmoner kapiller kama basıncı normal olmasına rağmen, artmış pulmoner vasküler direnç nedeniyle sağ kalp yetersizliğine yol açan ilerleyici bir grup hastalık için kullanılan genel bir terimdir. Nadir görülmekle beraber mortalitesi yüksektir. İdiyopatik PAH'de tanı konduktan sonra tedavisiz yaşam süresi ortalama 2.8 yıldır. Mortalitenin bu kadar yüksek olmasının en önemli nedenlerinden biri tanının geç konmasıdır. İdiopatik pulmoner hipertansiyon tanısı ile tarafımıza yönlendirilen, tetkikler sonrası ciddi pulmoner darlık saptanan genç bir hastayı vaka sunumu olarak hazırladık.

Olgu: Otuz dört yaşında bayan hasta son 1 aydır giderek artan nefes darlığı şikayeti ile kardiyoloji polikliniğimize başvurdu. Hastadan daha öncesinde hiçbir şikayeti yokken son 1 aydır nefes darlığı şikayetinin başladığı ve bu şikayetin giderek arttığı öğrenildi. Fizik muayenesinde bir özellik olmayan hastanın elektrokardiyografisinde sağ ventrikül hipertrofi bulguları bulunmaktaydı (Şekil 1). Hastaya yapılan transtorasik ekokardiyografide sağ ventrikülde hipertrofi ve dilatasyon öncelikle göze çarpmaktaydı (Şekil 2). İnceleme sırasında hasta idiopatik pulmoner hipertansyon tanısı ile tarafımıza yönlendirildiğinden pulmoner arter basıncı triküspit kapak yetersizliği üzerinden hesaplanarak yaklaşık 130 mmhg olarak saptandı (Şekil 3). Parasternal kısa aks görüntülerde pulmoner kapak düzeyinde artmış türbülan akım ve bu bölgeden continuous wave Doppler (CW) ile maximum 128 mmHg gradient saptandı (Şekil 4). Çekilen kontrastlı tomografide ise pulmoner kapak düzeyinden başlayan yaklaşık 1 cm' lik segmentte ciddi darlık saptanarak hasta ciddi pulmoner darlık tanısı ile takip edilmeye başlandı (Şekil 5).

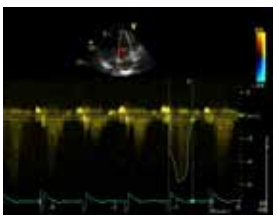
Tartışma: Pulmoner arter darlığının en sık nedeni ameliyat öncesi ve sonrasındaki Fallot tetralojisidir. Gebelikte ilk üç aylık dönemde geçirilen viral enfeksiyonlara bağlı olarak ve Alagille sendromunda da pulmoner darlık sık görülmektedir. Pulmoner hipertansiyon artmış pulmoner vasküler direnç ile karakterizedir. Ekokardiyografide pulmoner arter basıncı sıklıkla triküspit kapak yetersizliği üzerinden CW dopler ile hesaplanmaktadır. Pulmoner kapak darlığı olan hastalarda ise pulmoner vasküler yapı normaldir. Bu hastalarda triküspit kapak yetersizliği üzerinden hesaplanan basınç pulmoner arter basıncını değil pulmoner kapak düzeyindeki darlığa bağlı gradient farkını göstermektedir. Sonuç olarak pulmoner arter basıncı triküspit yetersizliği üzerinden yüksek olarak saptanılan ve pulmoner hipertansiyon düşünülen hastalarda pulmoner kapağın ve pulmoner darlığın değerlendirilmesi uygun bir yaklaşım olacaktır.



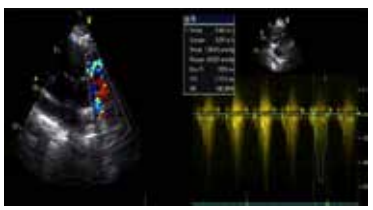
Şekil 1.



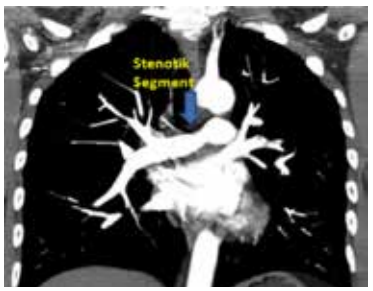
Şekil 2.



Şekil 3.



Şekil 4.



Şekil 5.

OPS1-38

Derin ven trombozu zemininde gelişen pulmoner tromboemboli ve mortal komplikasyonu: Patent foramen ovale ilişkili biatrial trombus

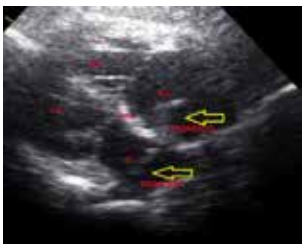
Metin Çoksevim[1], Mustafa Yenerçağ[2], Ahmet Yanık[2], Buğra Çerik[1], Murat Akçay[1], Korhan Soylu[1]

[1]Ondokuz Mayıs Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Samsun
[2]Samsun Mehmet Aydın Eğitim ve Araştırma Hastanesi, Samsun

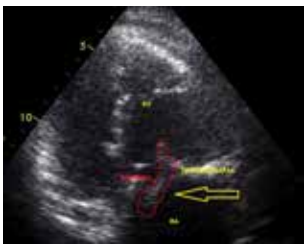
Giriş: Derin ven Trombozu (DVT) ile birlikte pulmonertromboemboli (PTE) sık görülebilirken, oluşan trombusun patent foramen ovale (PFO) aracılığı ile sol atriyuma geçişi nadir bir durumdur. Biz sizlere derin ven trombozu, pulmoner embolisi ve biatrial trombusu olan ve cerrahi olarak tedavi etmeyi denediğimiz bir vaka sunacağız.

Olgu Sunumu: 41 yaşında erkek hasta progresif olarak artan nefes darlığı yakınması ile başvurdu. EKG de sinusal taşikardi mevcuttu. Solunum sayısı 24/dk, TA:140/70 mmHg kalp hızı: 130/dk idi. Sol bacakta şişlik ve hassasiyet saptandı. Nörolojik defisit saptanmadı. Hastaya çekilen pulmoner bt anjiografide sağ ve sol pulmoner arterde subtotal darlığa neden olan dolma defekti saptandı.Alt ekstremite doppler us de sol popliteal ven lümenini boylu boyunca tıkayan ve minimal rekanilize akıma izin veren trombus saptandı. Yapılan ekokardiyografide; sol ventrikül çap ve duvar hareketleri normal, septum düzleşmiş,sağ ventrikül belirgin hipokinetik görünümde.İnter atrial septum'a yapışık hem sağ hem sol atriyum içine uzanan LA'da 19x10 mm, RA'da 27x14 mm mobil, trombus ile uyumlu görünüm saptandı (Şekil 1, 2). PAB:100 mmHg olarak ölçüldü. Hastanın hemodinamik olarak stabil olması nedeni ile heparin infüzyonu başlanarak yoğun bakıma yatışı yapıldı. Aynı gün inferior vena kava filtresi takıldı ve KVC-kardiyoloji ortak konseyinde değerlendirilerek operasyon kararı alındı. Hastaya pulmoner aterektomi, pfo onarımı, atrial trombektomi yapıldı. Operasyon sonrası 3 saat boyunca kalp-akciğer makinesinden ayrılamayan hasta exitus kabul edildi.

Sonuç: DVT olan hastaların üçte biri PTE ile presente olmaktadır. Yüzen sağ atrial trombus ise nadir olmakla birlikte hemen tamamen PTE ile ilişkilidir. Atrial trombusun PFO ile hapsolmuş hali ise çok daha nadirdir. Bu, belirgin artmış sağ atrial basınç ile ilişkildir. Bu durumlar göstermektedir ki PTE tanılı hastalarda 1. basamak tanı aracı olarak rutin ekokardiyografi yapmak gerekmektedir. Biz vakamızda; DVT, PTE, PFO ve hareketli büyük biatrial trombus saptadık. Bu aşamadan sonra 'en iyi' tedavi seçeneği halen tartışmalı olmasına rağmen güncel kılavuzlar ve çalışmalar referans alınarak hastaya cerrahi tedavi seçeneği daha uygun görülmüştür. Ancak belirgin sağ ventrikül yetmezliği gelişmesi nedeni ile kalp akciğer makinesinden ayrılamayan hasta exitus olmuştur. Yeterli tecrübe ve uygun ekipman ile bu tür olgularda cerrahi tedavi halen uygun seçenek gibi görünse de akut kalp yetersizliği gelişme ihtimali yüksek olan hastalarda diğer tedavi seçenekleri de değerlendirilmelidir.



Şekil 1. Subkostal görüntüde İAS'de 'tuzaklanmış' trombus görünümü



Şekil 2. RA içinde triküspit kapak hareketi ile mobilize olan İAS'ye yapışık trombus görünümü.

OPS1-39

A rare case: Simultaneous venous thromboembolism and peripheral arterial emboli

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A 27-year-old, white, mediterranean male was admitted to gastroenterology service of our tertiary clinic for advanced evaluation due to painful swelling in the right leg and abdominal pain. Shortly after admission has been made the patient complained paleness and weakness in the right arm. Thus, the patient was consulted for advanced cardiac evaluation. Clinical examination showed conscious, alert, cooperative and normally built male. Blood pressure was measured as 110/60 mmHg from the left arm. Oxygen saturation was 94%. There was pallor in the right arm without clubbing, cyanosis, or edema. Right brachial and radial pulses were absent. The other peripheral pulses were symmetrically palpable. Cardiac auscultation showed a 2/6 systolic murmur best heard at the third right intercostal space. Right lower extremity examination showed pretibial edema and positive Homans' sign. Electrocardiography revealed a normal axis and sinus tachycardia at 115 bpm. Trace revealed positive Q wave and negative T wave in lead DIII and negative T wave in leads V1-V3 (Figure 1). Transthoracic echocardiography revealed a normal left ventricular ejection fraction, dilated right ventricle, moderate tricuspid regurgitation, systolic pulmonary artery pressure of 50-55 mmHg, tricuspid annular plane systolic excursion (TAPSE) of 11mm, positive McConnell's and 60/60 signs and a thrombus in the right atrium and right pulmonary artery. Transthoracic echocardiography revealed an uncertain colored flow through intra-atrial septum. Agitated saline contrast echocardiography showed a shunt between right and left atrium compatible with patent foramen ovale (PFO). Arterial doppler ultrasound of the right upper extremity showed a total occlusion of the axillary artery and venous doppler ultrasound of the right lower extremity revealed a thrombus in the popliteal vein. Thorax and upper extremity computed tomography angiography demonstrated a total occluded right axillary artery and thrombus in the right and left pulmonary arteries compatible with pulmonary embolism (PTE) (Figure 2, 3). The patient was diagnosed with deep venous thrombosis (DVT), PTE and paradoxical emboli (PDE). Pulmonary Embolism Severity Index (PESI) of the patient was 57. The patient had low risk for bleeding. It was decided to administer systemic thrombolytic agent. Thus, 100mg Alteplase was infused intravenously for two hours. No complication occurred. 72 hours later transthoracic echocardiography was performed which demonstrated normal right chambers and a systolic pulmonary

artery pressure of 25 mmHg. Control right upper extremity doppler ultrasound showed a triphasic blood flow in the right axillary, brachial, ulnar and radial arteries. Anticoagulation therapy with was commenced. Thrombophilia panel test of the patient showed homozygote mutations in both PAI-1 gen and MTHFR gene. The patient was discharged on the 5th day with warfarin 5 mg qd for regular out-patient follow up.

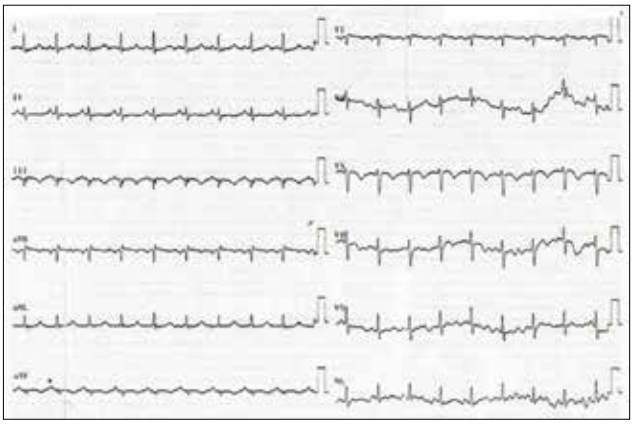

Figure 1. Electrocardiogram.

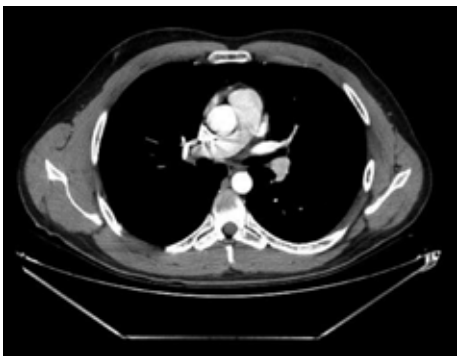

Figure 2. Thorax computed tomography angiography demonstrating thrombus in pulmonary artery.

Figure 3. Upper extremity computed tomography anjiography demonstrating totally occluded right axillary artery.

OPS1-40

Atypical presentation of pulmonary embolism with right bundle branch ventricular tachycardia

Süleyman Cagan Efe[1], Yavuz Ugurlu[1], Sahin Avsar[1], Ahmet Seyfettin Gurbuz[3], Emre Ozoran[1], Emrah Acar[2], Mehmet Fatih Yilmaz[2], Alev Kilicgedik[2], Ahmet Guler[2], Cevat Kirma[2]

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[3]*Department of Cardiology, Karaman State Hospital, Karaman*

A 72-year-old male patient admitted to the emergency department with dyspnea, palpitation, dizziness and confusion. At the admission, his blood pressure was 70/40 mm Hg and her heart rate was 180 beats per minute (bpm). He had tachypnea and respiratory distress with blur of consciousness. ECG (electrocardiography) showed a broad complex QRS in right bundle branch block morphology and superior axis deviation with typical morphologic pattern of brugada and vereckei algorithm, supporting ventricular tachycardia (Figure 1). Confusion prompted immediate cardioversion. After the cardioversion, rhythm was taken under control but confusion and hypotension persisted. He was intubated due to respiratory distress and confusion. Hypoxia and hypocarbia (SO2: 78%, pCO2: 32 mmHg) were observed in his arterial blood gas analysis despite intubation. Transthoracic echocardiography (TTE) was performed to evaluate the cardiac functions. Left ventricular ejection fraction was 60%. Dilatation and thrombus formation were seen in the right chambers (Video 1). Moderate tricuspid regurgitation was observed and the systolic pulmonary artery pressure was measured as 45 mm Hg. Immediate thrombolytic agent raphyciline was applied with 2 separate doses in the emergency department. After the application of the second dose, the patient was normotensive. Arterial hypoxia exceeded in transthoracic echocardiography thrombus ımage size decreased in right chambers. The patient was anti-coagulated with enoxiparine and warfarin to maintain an INR of 2.0–3.0, in the intensive care unit. He was extubated at the second day of hospitalization. Neither tachycardia nor respiratory distress was seen. The patient refused to undergo electrophysiological study thus medical treatment was initiated. He was discharged with warfarin prescription. The atypical presentation of pulmonary embolism with ventricular tachycardia is rare. Our case demonstrated a patient right bundle branch tachycardia with pulmonary embolism. The diagnosis of acute pulmonary embolism is one of the most challenging problems encountered in clinical practice. In patients with ventricular tachycardia if hemodynamic instability persists despite rhythm control, pulmonary embolism must be taken into consideration.

Pulmoner hipertansiyon / Pulmoner vasküler hastalık

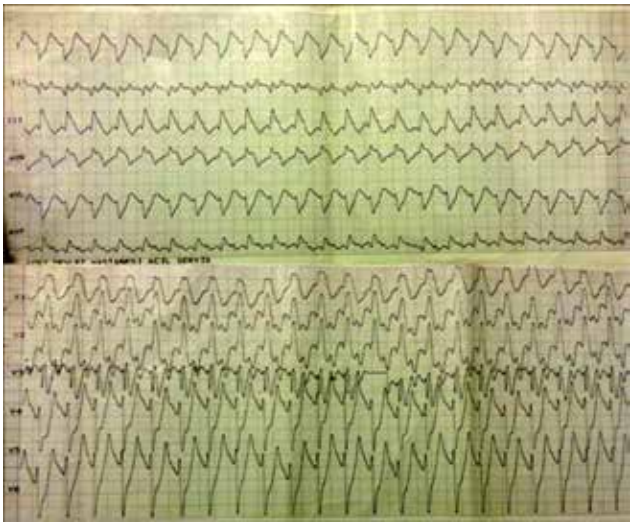

Figure 1. Right bundle brunch ventricular tachycardia.

OPS1-41

Right heart thrombus-in-transit with pulmonary embolism

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Department of Cardiology, Çukurova University Faculty of Medicine, Adana

An 76-year old female was admitted to emergency department with acute-onset dyspnea. She had hypertension for 20 years. On day 2-3 the patient suffered weakness, today her complaint was aggravation. Her electrocardiogram showed sinus tachcardia. On examination, the patient was afebrile with a blood pressure 88/54 mmHg, heart rate of 123 bpm, and oxygen saturation of 88% on ambient air, which improved to 93% on supplemental oxygen at 2 L/min via nasal cannula. Her lung, cardiac, abdominal examinations were otherwise unremarkable. Patient in the follow up was observed atrial fibrilation. The patients echocardiogram showed a normal left ventriculer ejection fraction (LVEF) and dilatation of right atrium (RA), right ventricule (RV). A large thrombus in the inferior vena cava and RA extending to the RV across the tricuspid valve was seen (Figure 1). Complete blood count and complete metabolic panel were normal. Her troponin I level was elevated at 1.26 ng/mL (0-0.04 ng/ml). Computed tomography pulmonary angiogram demostrated extensive bilateral acute pulmonary emboli, and intraluminal right atrium, intrahepatic vena cava filling defect consistent with an in-transit thrombüs (Figure 2). Thrombolysis with tPA was administered (100 mg over 2h). and thrombus was disappeared (Figure 3). Work up for evaluation of underlying malignancy was negative and a lower limb compression ultrasonography showed no deep venous thrombosis (DVT). She was discharged on oral anticoagulants and remains well.

Figure 1. Right heart thrombus-in-transit.

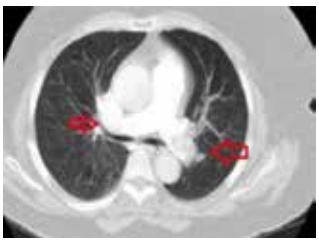

Figure 2. Bilateral pulmonary emboli.

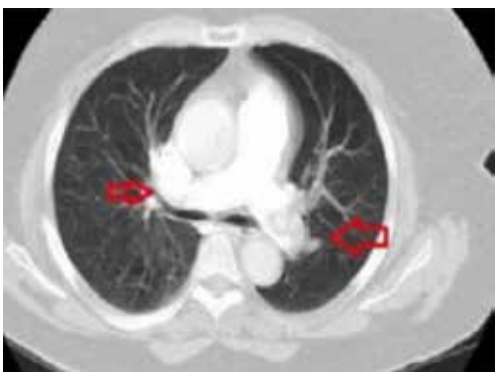

Figure 3. Thrombus was disappeared.

OPS1-42

Primer hiperoksalüri ve ASD'ye bağlı görülen nadir bir pulmoner hipertansiyon olgusu

Türkan Seda Tan Kürklü, Nil Özyüncü, İrem Müge Akbulut, Cansın Tulunay Kaya, Hüseyin Göksülük, Tamer Sayın, Deniz Balcı, Elvan Onur Kırımker

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Giriş: Glikolat metabolizması ile ilişkili enzim defektlerinden kaynaklanan primer hiperoksalüri idrarla bir günde atılan oksalat miktarının normal değerinin 3-6 katı fazla olması ile belirlenir. Bu hasta popülasyonunda çocukluk veya ergenlik döneminde sık üriner sistem taşı oluşumu görülür. Primer hiperoksalürinin iki tipi bulunmakal birlikte tip 1 primer hiperoksalüri diğer adıyla glikolik asidüri daha sık görülmektedir. Hastalığın erken tespitinde renal transplantasyon hayat kurtarıcı olmakla birlikte geç kalınması halinde vücutta biriken oksalat birçok organ hasrına sebep olmaktadır. Özellikle karaciğerde biriken oksalat karaciğer sirozuna ve buna bağlı portal ve portopulmoner hipertansiyona yolaçabilmektedir.

Olgu Sunumu: Yirmi altı yaşında kadın hasta primer hiperoksalüriye bağlı KC-S ve KBY tanıları nedeniyle KC ve Renal transplantasyon planlanarak cerrrahi kliniğine yatırılmış olup yapılan trans torasik ekokardiyografide pulmoner arter basıncı yüksek ASD tespit edilmesi sonucunda ASD operasyonu yapılmıştır. Hasta devam eden dispne nedeniyle klniğimize yatırıldı. Yapılan ekoda PAB. 70 olan hastaya hemodinami laboratuarına alınarak hemodinami yapıldı. PCWP: 14 mmHg PA: 50/21/31 mmHg PVR: 3.47wood RV:50/0/3 saptandı. Hastada mevcut pulmoner hipertansiyonun diğer nedeni olarak portopulmoner hipertansiyon tespit edildi. Pulmoner arter basıncının operasyon sınırında gelmesi üzerine hasta karaciğer ve renal nakil olamak üzere genel cerrrahiye transfer edildi. Tranfer sırasında operasyon öncesi tedavi olarak hastaya Tadalafil başlandı. Hasta karaciğer ve renal başarılı nakil operasyonu oldu.

Tartışma: Pulmoner hipertansiyon son kılavuzunda klasifikasyonda beş ana basamak belirlenmiş olup vakamıl grup beşe dahil olmaktadır. ASD ve Karaciğer Sirozuna bağlı iki sebepten gelişen pulmoner hipertansiyon olgumuz nadir görülen bir vakadır. Primer hiperoksalüri sebep olduğu karaciğer sirozuna bağlı pulmoner hipertansiyona yol açtığı düşünülen bir hastalık olmakla birlikte. Çok az olguda kardiyak oksalat birikimine bağlı kardiyak disfonksiyon buna bağlı sekonder pulmoner hipertansiyon gösterilmiştir.kardiyak tutulumun olmadığı nadir olguda ise pulmoner vasküler yatakta biriken oksalat kristallerinin pulmoner hipertansiyona sebep olacağı belirtilmiştir. Olgumuzda kardiyak disfonksiyon gösterilmemiştir.

OPS2-01

İlginç bir kardiyak kist hidatik olgusu

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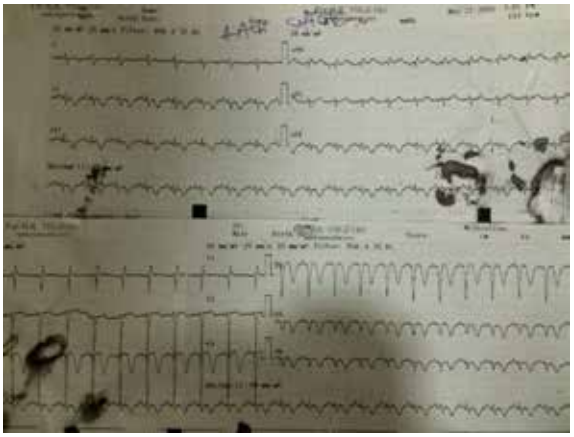
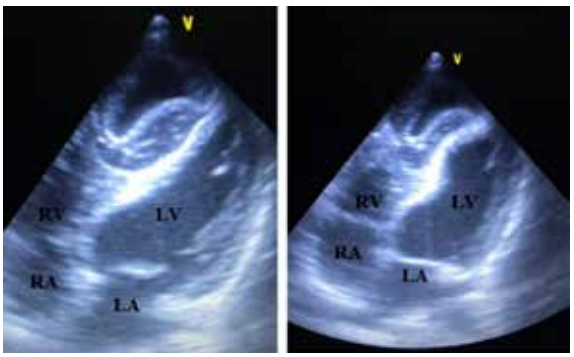
Giriş: Kist hidatik gelişmekte olan ülkelerde görülen önemli bir hastalıktır. Genellikle karaciğer (KC) ve akciğer'de yerleşim gösterir; kardiyak yerleşim daha nadirdir (%0.5-2). Kardiyak kistler genellikle uzun süre asemptomatik seyreder, ancak kistin yerleşimine göre atriyoventriküler blok, perikardit ve ani ölüm gibi ciddi komplikasyonlar görülebilir. Bu olgumuzda ürtikeryal semptomlarla başvuran ilginç bir kist hidatik olgusu sunulacaktır.

Olgu Sunumu: On yedi yaşında erkek hasta acil servise hipotansiyon, göğüs ağrısı ve ürtikeryal semptomlarla başvurdu. Kan basıncı 80/50 mmHg ve tüm vücutta yaygın ürtikeryal plakları mevcuttu. Çekilen EKG'de D2-3, aVF, V3-6'da simetrik derin negatif T dalgaları mevcuttu (Şekil 1). KC ve böbrek fonksiyon testleri normal aralıklarda idi, ancak Troponin T [0.024 (0.003-0.014)] hafif yükselmişti. Hemoglobin 17.4 g/dl, hematokrit %51.5, beyaz küre 7800/mm^3 ve eozinofil %1.5 (%0-7) idi. Yapılan EKO'da sol ventrikül apeks önünde, interventriküler septuma doğru uzanım gösteren kistik bir lezyon izlendi (Şekil 2). Ön planda kist hidatik düşünülen hastada, çekilen torakoabdominal BT'de KC'de düzgün kontürlü ve kalsifikasyonlar içeren grade 5 kalpte ise interventriküler septum düzeyinden başlayarak apekse doğru uzanım gösteren 63x51 mm boyutunda grade 2 kist hidatik izlendi. Kardiyak bölgedeki kistin germinatif membranından ayrıştığı, ancak rüptür bulunmadığı tespit edildi (Şekil 3). Kist rüptürü bulunmayan hastanın semptomatik tedavisi yapıldıktan sonra tedavi seçeneklerinin değerlendirilmesi amacıyla ileri bir merkeze sevk edildi.

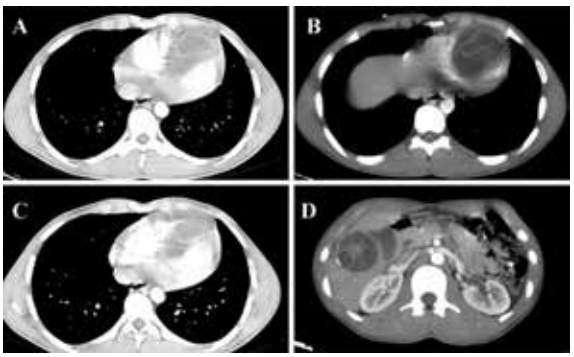
Tartışma: Kardiyak kist hidatik nadir görülen, ancak lokalizasyon itibariyle önemli sorunlara yol açabilen bir hastalıktır. Kistler genellikle koroner dolaşımla kalbe ulaşır ve asemtomatik seyreder. Kist sıvısının antijenik yapısından dolayı bizim vakamızda da olduğu gibi anaflaktik reaksiyon gelişebilir. Bunun dışında, yerleşim yerine göre AV blok, perikardit, koroner arterlere bası nedeniyle miyokard iskemisi ve sol ventrikül boşluklarına açılarak sistemik embolizasyona yol açabilirler. Olgumuzda muhtemelen kistin yarattığı miyokard iskemisine bağlı olarak EKG değişiklikleri ve kardiyak Troponin T yüksekliği mevcuttu. Kardiyak kist hidatik EKO'da tipik görünümü ile kolaylıkla tanınabilir. Boyut ve yerleşimini göstermede BT kullanılmaktadır. Bu sırada diğer organlarda tespit edilen başka kistlerin varlığı tanıyı kuvvetlendirir. Olgumuzda TTE ile şüphelenen vakanın tanısı BT ile doğrulandı. Kesin tedavisi cerrahidir. Medikal tedavinin cerrahiye eklenmesi tedavi başarısını yükseltir. Ancak intramiyokardiyal kritik yerleşimli kistlerde cerrahi girişimin mortalitesi yüksektir. Bizim olgumuz, yerleşim itibarı ile yüksek riskli grupta değerlendirildi. Bu nedenle cerrahi dışı diğer tedavi seçeneklerinin araştırılması için ileri bir merkeze sevk edildi. Bu hasta grubunda, mortaliteyi önlemek için perkütan boşaltma gibi daha az invaziv yöntemlerin geliştirilmesi gerekir.



Şekil 1. EKG'de muhtemelen kistin yarattığı miyokard iskemisine bağlı olarak D2-3, aVF, V3-6'da ST depresyonu görülmektedir. (Bu hastalarda kistin vasküler yapılarla ilişkisini değerlendirmek için koroner anjiyografi yapılabilir).



Şekil 2. EKO'da sol ventrikül apeks önünden, interventriküler septuma doğru uzanım gösteren, 6x5 cm boyutlarında, düzgün sınırlı kistik görünümlü lezyon. Renkli Doppler görüntülerinde, kistik alan ile kardiyak boşluklar arasında herhangi bir şant akımı izlenmedi.



Şekil 3. Tanıyı doğrulamak ve diğer organ tutulumlarını incelemek amacıyla çekilen Torakoabdominal BT'de; kalpte interventriküler septum düzeyinden başlayarak sol ventrikül anterioruna ve apekse doğru uzanım gösteren en geniş yerinde 63x51 mm ölçülen, düzgün konturlu, germinatif membranı ayrışmış olan grade 2 kist hidatik ile uyumlu lezyon izlendi. Lezyon sol ventriküle kısmen bası yapıyordu. Karaciğer segment V düzeyinde 49 mm çapında, düzgün konturlu, kalsifikasyonlar içeren grade 5 kist hidatik ile uyumlu lezyon izlendi.

OPS2-02

A rare complication: Diffuse alveolar hemorrhage following acute coronary syndrome diffuse alveolar hemorrhage

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48 year-old male patient with past medical history of essential hypertension and smoking was admitted to our hospital with anterior ST segment myocardial infarction(STEMI). After treatment with 300 mg acetyl-salicylic acid (ASA), 180 mg ticagrelor and 7500 IU unfractioned heparin iv bolus, the patient underwent coronary angiography showing total occlusion of the left anterior coronary artery. Angioplasty was performed but slow flow phenomenon occured after drug eluting stent (DES) implantation. Accordingly, tirofiban bolus were administered over 3 minutes, followed by a 0.15-μg/kg/min tirofiban infusion. The patient trasferred to coronary intensive care unit and his pain

was disappeared. Transthoracic echocardiogram (TTE) revealed anterior septal wall hypokinesis with slighty decreased systolic dysfunction (ejection fraction of 50%) and mild mitral regurgitation. Then, the patient developed severe dyspnea, tachypnea and hemoptysis of bright red blood. On physical examination, he had diffuse rales over both lung fields. The pulse oximetry revealed a severe oxygen desaturation of 80% despite 100% O2 support. The chest X-ray film showed that bilateral diffuse opacities (Figure A). He had a chest computed tomography (CT) scan and it demonstrated widespread alveolar filling (Figure B and C). The laboratory tests showed hemoglobin level declined from 15 g/ dL to 12.3 g/d and platelets count, prothrombin time, and partial thromboplastin time were normal. On the basis of this findings, new onset shortness of breath and hemoptysis after administration of antiplatelet agents, diffuse alveolar hemorrhage was diagnosed.

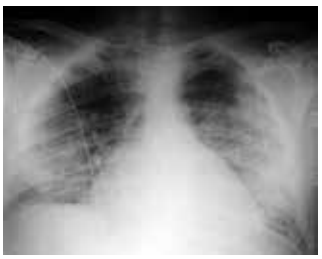

Figure 1. Chest X-ray revealing bilateral diffuse lung opacities.

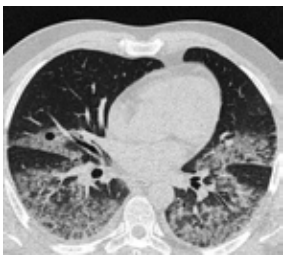

Figure 2. Chest computed tomography scan in axial plane showing widespread alveolar filling.

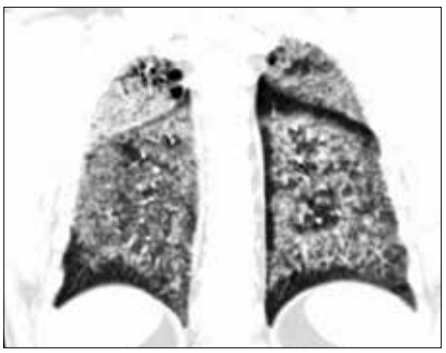

Figure 3. Chest computed tomography scan in coronal plane showing wide spread alveolar fillling.

OPS2-03

Spontaneous hemorrhagic pleural effusion under apixaban treatment

Mehmet Onur Omaygenç, Ersin İbişoğlu, İbrahim Oğuz Karaca, Hacı Murat Güneş, Beytullah Çakal, Ekrem Güler, Filiz Kızılırmak, Ümeyir Savur, Filiz Çelebi, İrfan Barutçu

Department of Cardiology, İstanbul Medipol University Faculty of Medicine, İstanbul

A 72-year-old male patient attended to our-patient clinic with heart failure (HF) decompensation symptoms like fatigue, gradual increase in exertional dyspnea and orthopnea. He had already diagnosed as Heart Failure with Preserved Ejection Fraction and permanent non-valvular atrial fibrillation. He was evaluated for right sided massive recurrent pleural effusion previously and laboratory work-up did not yield a certain diagnosis other than HF at that time. Eventually, he was treated with thoracentesis and pleurodesis. He had been taking a proper drug regimen for these medical conditions including low dose furosemide and apixaban (5 mg, b.i.d) since then. CHA2DS2 VASC score was 3. Baseline physical examination revealed arrhythmic heart sounds, mild diastolic murmur on left parasternal area. Respiratory sounds were decreased on left base and right base to mid pulmonary auscultation zones. Lab tests were close to normal for his age group. Creatinine level was 1.2 mg/dl with an eGFR value of 58.4 ml/min. Initial chest x-ray revealed interstitial edema, right-sided pleural effusion at basal one-third of lung field, a phantom tumor and increased cardio-thoracic ratio (Figure 1). In echocardiographic examination, mild to moderate aortic regurgitation, normal LVEF and biatrial dilatation were observed. Pulmonary artery systolic pressure was estimated as 35 mmHg. Regarding these findings, he was hospitalized and in addition to his existing medical treatment intravenous furosemide with close monitorization of urine output was initiated. Symptomatic improvement was achieved in first two days without a deterioration in renal function tests. On the third day of treatment a sudden onset shortness of breath occured. There was no respiratory sound at the right side and control chest x-ray demonstrated an outrageous increase in pleural effusion (Figure 2a). Computed tomograhy verified the diagnosis (Figure 2b) and emergent thoracic tube drainage was performed. Fluid was in hemorrhagic appearance (Figure 3a). Abundant drainage was observed in first two hours and then output gradually decreased, thus, reversal of anticoagulation was not administered. Further biochemical, pathological and microbiological investigation of the material did not offer an alternative diagnosis rather than hemorrhagic conversion. Clinical recovery was achieved dramatically and control chest x-ray was obtained the day after. Effusion was minimal and no additional pathology was observed in lung parenchyma except mild edema (Figure 3b). Drainage tube was withdrawn and medical treatment was proceeded. Ultimately, patient did well after this event and could be discharged at the end of the week. Long term anticoagulation strategy was discussed and reducing the dose (2.5 mg b.i.d) seemed reasonable. Patient was symptom-free at sixth month visit and no bleeding event was observed henceforward.

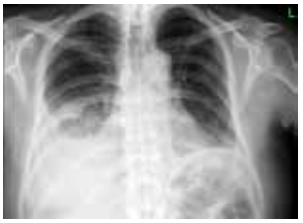

Figure 1. Baseline x-ray of the patient. Interstitial edema, right-sided pleural effusion at basal one-third of lung field, a phantom tumor and increased cardio-thoracic ratio is evident.

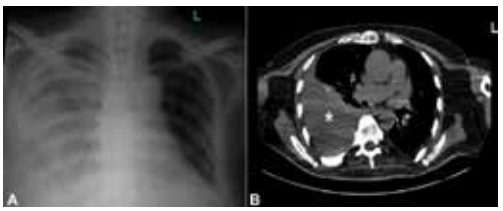

Figure 2. Third day x-ray of same patient. Massive pleural effusion on the right side. **(A)** Concurrent thorax computed tomography image confirming effusion. (*). **(B)**.

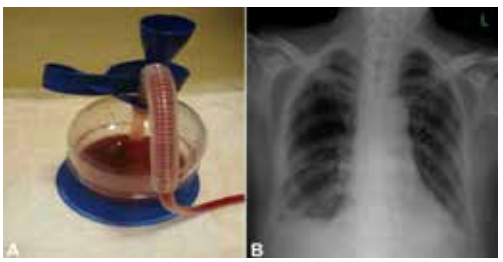

Figure 3. Drainage material in hemorrhagic appearance. **(A)** Chext x-ray obtained the day after tube drainage. Effusion is almost completely resolved **(B)**.

OPS2-04

Coughing responsible for a.... heart attack

Nassime Zaoui

EHS Draa Ben Khedda Tizi-Ouzou

The acute coronary syndrome is a high-risk situation, its association with respiratory emergency as pneumothorax is rare and life-threatening, only a few cases have been described in the literature. We describe the case of a 77 years old male, smoker, with chronic bronchitis, admitted for management of chest pain with severe cough and hemodynamic instability, including ECG revealed ST segment elevation in AVR lead with an anterior ST-segment depression Coronary angiography done urgently reveals atherothrombotic tight lesion of the ostial left main treated with emergency angioplasty with implantation of an active stent providing hemodynamic stabilization, angiography also revealed a lucency of the right hemithorax whose X-ray and CT scan confirmed a total right compressing pneumothorax with mediastinal deviation treated successfully by surgical drainage. The combination of an acute coronary syndrome with a pneumothorax is rare but dangerous, it can be either simple electrical changes during pneumothorax or a true partner coronary syndrome, in which case the coronary compression is criminalized or preexisting plaque rupture due to adrenergic discharges because of the pleural pain, either the mechanical distortion caused by the pneumothorax.

OPS2-05

Atrial fibrillation assosiated with high dose corticosteroid

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18 years old male patient with no known organic disease was admitted ear,nose,throat clinic with sudden hearing loss in the left ear. The patient's blood pressure was 130/80 mmHg and body temparature was 36.5 degrees celcius. ECG was normal sinus rhythm. The patient's blood count, kidney and liver function tests, thyroid function tests, erythrocyte sedimentation rate, C-reaktif protein values were within normal limits. Brain computer tomography is normal. 1mg/kg (patient's weight 70 kg) methyl prednisolone intramuscular, vitamin B oral and proton pump inhibitor was started for treatment. Palpitations occured in the first 24 hours of the treatment. ECG showed atrial fibrillation taken after palpitation. Echocardiographic examination was normal left atrial size was 3.5 cm. Steroid treatment caused sudden hearing loss was decreased and patient was monitored. About 24 hours of monitoring showed normal sinus rhythm. Atrial fibrillatin wasn't seen again. Sudden hearing loss is partiallly reduced. He was discharged after healed by ear,nose,throat clinic after 3 days. Patients with atrial fibrillation after 1 month follow up didn't occur in the cardiology clinic made. In patients treated with high doses of steroid need to be careful in terms of atrial fibrillation and other cardiac side effects.

OPS2-06

Diagnosis of hyperpotasemia on eletrocardiography in a young woman with adrenal insufficiency

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Introduction: Severe hyperkalemia is defined as a serum potassium level (K^+) of 6.0mmol/L or greater with electrocardiographic (ECG) changes or a serum potassium level of 6.5 mmol/L or greater without ECG changes.

Case Report: A 21-year old female patient with no previous disease presented to the ED with complaints of fatigue, dizziness. The patient reported having lost 15kg of weight within the last 6months. There was increased pigmentation in oral mucosa and various areas of the body. Wide QRS complexes were recorded on ECG. Even though the rhythm was sinus, the P waves were flattened and were not clearly discernible in some leads and the PR interval was lengthened (250 msn). Peaked T waves were noted and QRS complexes had merged with the S wave (Figure 1). Serum K^+ level was 8.3 mmol/L and Na was 118 mmol/L. The patient was hospitalized with initial diagnoses of adrenal insufficiency. The patient was given 225 mg of calcium gluconate and 20% dextrose buffered with insulin was infused. Further examination of the patient revealed decreased cortisol (1.96 μg/mL) and increased ACTH (60 pg/ml).The synacthen test showed low cortisol level (7 μg/mL).The patient was given 20% dextrose buffered with insulin as well as 50 mg of sodium hydrogen carbonate and 20 mg of methylprednisolone for 4 days. In addition, fludrocortisone was administered in a dose of 0.4 mg. There was no change in sodium level on day 4 (122 mmol/L) but K^+ level was 5.7 mmol/L. At the 4 day follow up, an ECG showed normal sinus rhythm (Figure 2).

Discussion: Hyperpotassemia is usually associated with various ECG changes and these changes can be used to follow the effects of K^+ on the heart. As serum level of K^+ increases, so does transmembrane permeability of cardiac cells, which results in influx of K^+ flow into the cells. As a result, resting membrane potential is less negative, the action potential is shortened and conduction between myocytes is suppressed. As K^+ levels rise above normal, the earliest ECG change is usually peaked, symmetric, narrow-based T waves. A further increase in potassium levels results in declined conduction velocity, widened and flattened P wave, lengthened PR segment and wide QRS complex with slurring of the S wave into a rapidly ascending limb of the T wave. As a result, P wave disappears and the QRS complex resembles "sine wave" that may result in ventricular fibrillation or asystole. In the case presented here, the K^+ level was 8.3 mmol/L.An ECG showed flattened P and peaked T waves. The PR interval was lengthened and there were wide QRS complexes reaching 0.16 sec in some leads and the S wave of the QRS complex had merged with the ascending limb of the T wave. The findings of physical examination and ECG were of guidance in establishing etiological diagnosis. In conclusion, hyperpotassemia is an important clinical condition that requires urgent diagnosis and treatment. In these patients, ECG findings play an important role in diagnosis and treatment.

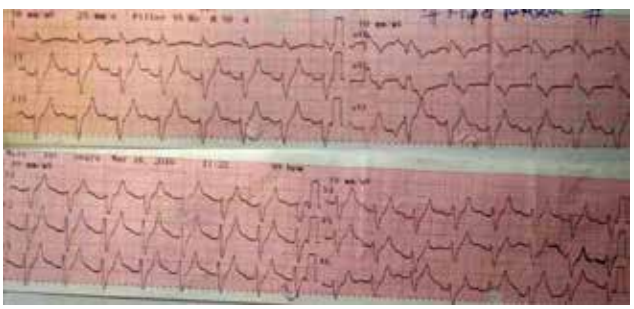

Figure 1. Electrocardiography in a 21-year-old woman with a 8.3 mmol/L potassium level.

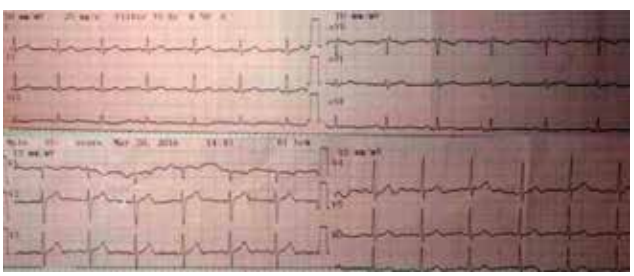

Figure 2. Electrocardiography after the treatment (potassium level 5.7 mmol/L).

OPS2-07

Cardiopulmonary arrest after a giant diaphragmatic hernia

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Introduction: Diaphragm, in addition to separate intraabdominal and intrathoracic spaces as a barrier, provides the 70% of the force for the respiratory functions. Diaphragmatic hernia is a rare clinical entity that is easily detected on the X-ray obtained for any other reasons. Severe elevation of the diaphragm or giant hernias may cause life-threatening lung and heart conditions and the early diagnosis may preserve the life.

Case Report: A 63 year-old male patient referred to our clinic for increasing epigastric pain and distention for three days on the sixth day of the hernia surgery. Blood pressure was 120/74 mmHg, heart rate 80 bpm on admission examination in the morning. Heart and lung sounds were normal in auscultation. Intestinal sounds were decreased and there was dullness in percussion of the abdomen. ECG showed normal sinus rhythm. Transthoracic echocardiography (TTE) showed normal left ventricular systolic functions. Biochemical analysis revealed moderate anemia (Hb: 9.1 g/dl), leukocytosis (WBC: 12000) and creatinine of 1,9 mg/dl with normal electrolyte levels. According to his previous history, the patient was administered rectal enema and only flatulence was achieved following the surgery for days ago. Although the fecal excretion had not been observed, the patient continued to take oral feeding; besides he ate 4-5 boxes of soda drink and 900 g banana at the same day before admission. Severe gastric and intestinal distention occurred at the patient who was unpretended from oral taking. The patient had the complaints of epigastric discomfort and distention, progressive dyspnea was followed by palpitation. He admitted to the emergency service. A giant diaphragmatic hernia was seen on the X-ray, compressing the heart mildly to leftward in time (Fig 1a, b). By means of nasogastric tube, approximately 4 liter liquid was aspirated from the gut (Fig 1c). After a while the patient was performed CPR and intubated due to cardiopulmonary arrest. Metabolic acidosis, pulmonary edema and severe bradicardia are suspected to be responsible for the cardiogenic shock and arrest. The patient died after ineffective supportive treatment for 6 hours.

Conclusion: Relatively big diaphragmatic hernias may cause severe distention and result in cardiopullmonary arrest even if treated with gastric lavage. Early diagnosis and emergent treatment is of vital importance for saving life.

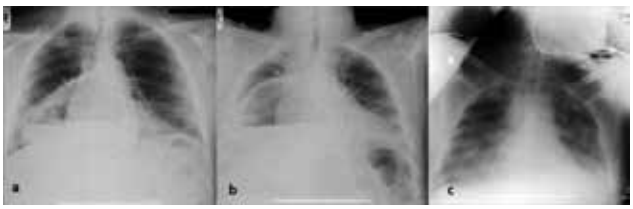

Figure 1. (A) X-ray shows diaphragmatic hernia on admission of the patient. **(B)** Severe distention shifted the heart mildly to the left. **(C)** Pulmonary edema is seen after emtying of the gut by nasogastric lavage.

OPS2-08

A different and rare cause of cardic tamponade: Ankylosing spondylitis

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A 28-year-old man admitted to our emergency department with compliants of severe dyspnea and pleuritic chest pain which had began 1month ago and had gradually increased. His medical history was unremarkable other than back pain in the mornings resolving during day time for five years. On physical examination, blood pressure was 80/60 mmHg, pulse rate was 118/min and respiratory rate was 24/min. Jugular venous distension was very apperent. Lung auscultation showed bilateral decreased breath sounds at the bases. An electrocardiogram denoted low voltage and diffuse ST segment elevation in all leads. The echocardiogram demonstrated a large pericardial effusion with collapse of right atrium suggestive of cardiac tamponade. Emergency pericardiocentesis via xiphoid approach was then performed and 750 ml of pericardial fluid was drained. Pericardial fluid cytology showed cells of an inflammatory response with no microorganism identified by Gram stain or Ziehl-Neelsen stain. PCR for tuberculosis was negative. Labaratory investigation demonstrated that hemoglobin was 11 g/dl with white cell count was 17.400/mm^3. Erythrocyte sedimentation rate was 88 mm/hr, C-reactive protein was markedly elevated, 107 mg/dl. Chest and sacroiliac X-ray was taken. Chest x-ray was compatible with bilateral pleural effusion at the base of lungs. Moreover, total ankylosing was observed on pelvic and sacroiliac X-ray. Since, the patient had inflammatory back pain and ankylosis of sacroiliac joints was ascertained, the patient was consulted to rheumatology deparment, thereafter. Patient's HLA B27 was positive and antinuclear antibody as well as anti-double strand DNA were negative. Ankylosing spondylitis (AS) was diagnosed and high dose steroid therapy along with sulfasalazine was administered. On the follow-up, not only pericardial effusion, but also pleural effusion was disappeared. Pericardial effusion has been rarely reported in AS patients. To the best of our knowledge, there is no previous report regarding cardiac tamponad associated with AS. We consider that our paper is the first case report in the literature demonstrating ankylosing spondilitis presenting with cardiac tamponade as an initial presentation. Moreover, our case emphasises that AS could also be a very rare etiologic factor of cardiac tamponade and rheumatologic diseases should be kept in mind as a differential diagnosis.

OPS2-09

Spontaneous intramuscular hematoma due to subcutaneous enoxaparin

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Objective: Low molecular weight heparins (LMWH) are generally used for acute coronary syndrome treatment. Although bleeding is the most common complication associated with the use of LMWH, it that can lead to hemodynamic disorder is rare. Intramuscular hematoma occurring after use of LMWH is a rare bleeding complications. Here we present a case of rectus abdominis intramuscular hematoma after use of subcutaneous LMWH.

Case Report: A 73 -year-old female patient admitted to the emergency room with palpitations and dyspnea was hospitalized with a diagnosis of acute coronary syndrome and pneumonia. Due to acute coronary syndrome, LMWH was started as anticoagulant therapy. When follow up in the service, the patient developed sudden abdominal pain. Immediately, abdominal ultrasonography

and computed tomography were performed and left rectus abdominis intramuscular hematoma was detected. Emergency surgery was not intended and supportive therapy (fluid, erythrocyte suspension, analgesics) was given to the patient. Hemodynamic parameters of the patient were followed up closely. Hematoma declined in follow up and reducing the patient's symptoms. Coronary angiography was performed when patient stabilizied hemodynamically and stent was placed to obtuse marginal artery which is a branch of the circumflex artery. After the procedure, the patient without symptoms was discharged with the recommendations.

Conclusion: Patients who LMWH administered subcutaneously and was performed suddenly abdominal pain with a palpable mass in physical examination, rectus abdominis intramuscular hematoma should be considered in the differential diagnosis. In patients with predisposing factors to reduce the risk of fatal rectus abdominis intramuscular hematoma, lower dose of LMWH should be used and care should be taken to make the injection under the skin. When rectus abdominis intramuscular hematoma develops, early diagnosis and a multidisciplinary approach and not be delayed treatment are the most important factor in combating these complications.

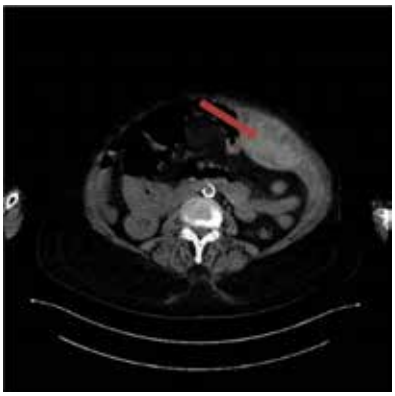

Figure 1. Rectus abdominis intramuscular hematoma.

OPS2-10

Ülseratif kolitde abdominal aorta trombozu

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[1]Muğla Sıtkı Koçman Üniversitesi Eğitim ve Araştırma Hastanesi, Kardiyoloji Bölümü, Muğla
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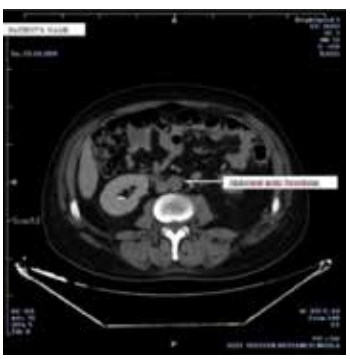
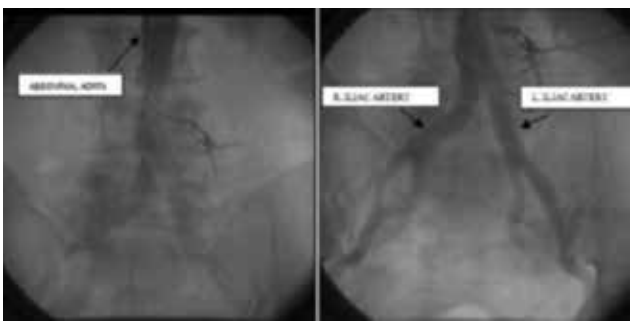
Ülseratif kolit birçok komplikasyonu olan kronik bir barsak hastalığıdır. Tromboembolik komplikasyonları nadir olmakla birlikte özellikle arteriyal trombozlar çok daha seyrektir. Bu komplikasyonlar özellikle genç hastalarda ve post-operatif dönemde görülür. Burada sunulan vakada aktif ülseratif kolitli 44 yaşında erkek hastada abdominal aorta ve iliak arter trombozu gelişmesi nedeni ile acil trombektomi yapıldı. Hastaya sonrasında yapılan periferik anjiyografide abdominal aorta ve dalları tamamen normal olarak izlendi.



Şekill 1. Kontrastlı abdominal tomografi.



Şekill 2. Periferik anjiyografi.

OPS2-11

Carotid Doppler indication for coronary angiography

Nassime Zaoui, Tarik Mokrab, Malika Sadour, Nadjib Laassakeur

EHS Draa Ben Khedda Tizi-Ouzou

The carotid IMT, carotid plaques and coronary stenosis are two locations of the same disease, atherosclerosis. Many studies have suggested the existence of a good correlation between carotid IMT and the presence of coronary lesions. Our work took place over two phases, during the first part we have confirmed the existence of a good correlation between carotid IMT and plaque score one hand and the existence, extent and severity coronary lesions (expressed by the angiographic Syntax Score) on the other in 500 patients admitted to our clinic for coronary angiography regardless of the indication and thus have all benefitted for this work from Doppler ultrasound of the supra-aortic trunks This phase allowed us to identify threshold values for these parameters and to check their sensitivity and specificity for the detection of the existence, extent and severity of angiographic coronary lesions Then on the basis of positive and negative likelihood ratios we proposed a new algorithm completing that proposed by the ESC for the indication of an ischemic test or coronary angiography facing a chest pain syndrome. The second phase of our work was to classify 189 patients undergoing elective coronary angiography for exertional angina according to the standard ESC guidelines and sort them according to our new algorithm including the carotid IMT and plaque score. Our new algorithm was able to correctly reclassify 92% of patients only 8% were classified as high risk while their coronary angiograms were normal, this algorithm could have saved us 40% of the stress tests and 63% of normal coronary angiographies (without significant stenosis) Our monocentric results, should inspire other centers in Algeria and in all countries where access to angiography remains delicate in order to reach a new consensus based on this inexpensive and non-invasive test.

Epidemiyoloji

OPS2-12

The routine use of unique left amplatz technique for coronary sinus lead placement with high success rate

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Kazakh National Medical University

The positive effect of left ventricular or biventricular pacing on cardiac heamodinamy was first described by Gibson et al. In patients with heart failure, the implantation of coronary sinus (CS) lead is considired as a complex process. There is an important learning curve for the successful implantation of biventricular pace. By the experiences, the implantation time, fluorocsopy duration and complication rates are decreased considerably. The main point of implantation procedure is intubation of CS ostium. The CS ostium is localized at posterior interatrial septum, behind the tricuspid valve, in right atrium. However, in patients with heart failure, because of the dilatation, the CS ostium is dislocated to the downwards and posteriorly. Preshaped guiding delivery sheaths, deflectable electrophysiology catheter and various angiographic catheters are used for intubation of CS. However, the success rates of left ventricular lead placement were reported between 53% to 98. Rutinely, we have been preferred to use left amplatz (AL2) catheter for the canulation of CS in biventricular pace implantation. Between 2009 and 2016 we performed 348 biventricular pace implantation, and in all patients the canulation of CS was succeeded by this technique. Shorter procedure and fluoroscopy times, lower amount of contrast, lower dissection risks, selective canulation of CS branches are the advantages of AL2 technique. By this technique, AL2 replaces into the outer sheath that is placed in right atrium. Y-connector is connected to AL2 catheter, and wash out. The operator passes to the right side of the patient. In the left anterior caudal oblique position, first the catheter is pushed forward to tricuspid valve and then the catheter is pulled on from interatrial septum towards the CS by counter clockwise rotation, a second operator gives low amount of contrast, when the CS ostium is engaged, the catheter is pushed slightly by clockwise rotation. Then, angiographic image is taken to analize CS anatomy. The 0.014 mm guidewire is placed to suitable vein of CS, then the catheter is pushed till the bifurcation of the vein and CS. After that, outer sheath is brought closer to the target vein over the AL2. When the outer sheath is brought to the ostium of target vein, 0.014 mm guidewire is fixed, and AL2 is pushed out slightly. After all, CS lead is replaced through the 0.014 mm guidewire. Because of these advantages and high success rate, we recommend using the left amplatz tecnique for CS canulation as a first choice and routinely.

Hipertansiyon

OPS2-13

Flush pulmonary edema: a rare cause and possible mechanisms

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Introduction: Hypertensive flash pulmonary edema (FPE) results from rapid elevation of the left ventricular end-diastolic pressure. Absence of underlying valve disease or cardiomyopathy, it is usually caused by renal vascular disease. It may frequently develop in case of bilateral renal artery stenosis (RAS), unilateral RAS and accompanying functional solitary kidney. However, unilateral RAS with bilaterally functional kidneys may also lead to a clinical picture of pulmonary edema. This paper presents a case of FPE caused by accessory RAS in a hypertensive patient with bilaterally functional kidneys.

Case Report: A 59-year-old male patient presented to the emergency department with rapidly developing shortness of breath and impaired consciousness after a 12-hour long road trip. The patient was hypertensive for 4 years but not taking any medication. He was a current smoker (46 packyears). On initial presentation, blood pressure was 255/133 mmHg, heart rate was 110 beats/min, and peripheral oxygen saturation measured with pulse oximeter was 50%. Initial blood tests

showed slight elevation in blood glucose (243 mg/dL), blood urea (51 mg/dL), and creatinine (1.28 U/L) levels. Blood gas analysis revealed severe hypoxemia (pO2: 42.5 mmHg, sO2: 50%), hypercarbia (pCO2: 73.6 mmHg), and mixed acidosis (pH: 7.007). Troponin level was within the reference range. NT-proBNP was elevated (3340 ng/L). D-dimer (2160 ng/mL, reference: 0-243) and fibrinogen (678 mg/dL, reference: 200-393) levels were increased. The telecardiogram showed patchy alveolar infiltrates indicative of pulmonary edema (Figure 1a, b). In transthoracic echocardiography, left ventricular enddiastolic diameter was 5.0 cm and ejection fraction was 55%. Right chambers of the heart were not dilated. Minimal mitral regurgitation was observed. Left ventricular hypertrophy (septum: 1.4 cm, posterior wall: 1.1 cm) and ascending aortic dilatation (4.5 cm) were other co-existing abnormalities. The following day, heart failure symptoms was completely resolved and patient was hospitalized. The follow-up echocardiography revealed grade 2 diastolic dysfunction. In renal ultrasonography, parenchymal disease and collecting system dilatation were absent. In Doppler sonography examination, the left renal artery flow rates were normal while the right renal artery could not be assessed due to rotational anomaly. The patient underwent coronary and renal angiography. No occlusive lesion was present in the coronary arteries. Double renal arteries were supplying the right kidney and the upper accessory renal artery had an ostial stenosis of 95% (Figure 2a-c). Percutaneous renal artery revascularization was planned. Intrarenal nitroglycerin was administered and following a 3.0x17-mm balloon predilatation, a 4.0x12-mm coronary stent was successfully implanted (Figure 2d). Post-procedural one-month follow-up demonstrated that the arterial pressure was under control with antihypertensive monotherapy.

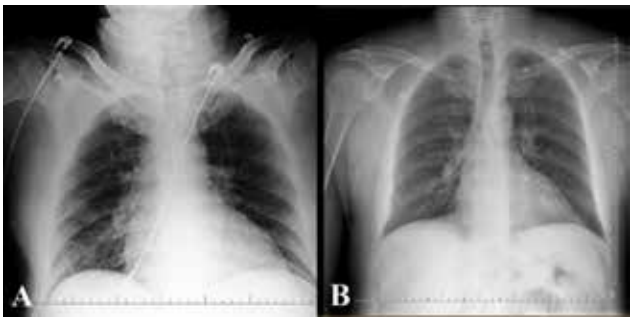

Figure 1.

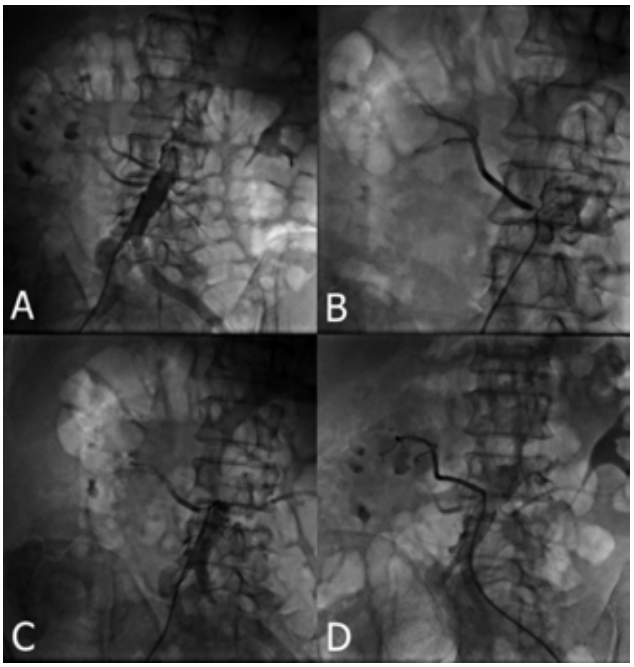

Figure 2.

OPS2-14

Amlodipine bağlı hepatotoksisite

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Giriş: Amlodipin hipertansiyon(HT) tedavisinde sıkça kullanılan bir ajandır. Kalsiyum kanal blokörleri, vasküler düz kas hücrelerine depolarizasyon sırasında kalsiyum(Ca) iyonu girişini engelleyerek etki gösterirler. Periferik ödem, çarpıntı, seksüel disfonksiyon amlodipin ile sık görülebilen yan etkilerdir. Hepatit ise amlodipin kullanımı ile nadir olarak ortaya çıkan bir yan etkidir.

Olgu Sunumu: Altmış bir yaşında erkek hasta HT mevcut. Altı yıldır sağ bacakta şişlik şikayeti olan hasta yaklaşık 6 ay önce derin ven trombozu(DVT) tanısı almış. Hasta bemiparin ve zofenopril tedavisi almaktaydı. Hasta kronik DVT etiyoloji amacı ile kliniğe yatırıldı. Klinikte çalışılan kan tetkiklerinde kreatinin değeri normal sınırın hafif üzerinde gelince zofenopril kesilerek amlodipine geçildi.Hastanın amlodipin başlandıktan sonraki kan tetkiklerinde karaciğer fonksiyon testlerinde yükselme saptandı. Beş gün sonra ALT 220 U/L (referans değer 0-49 U/L), AST 137 U/L (referans değer 0-34 U/L), alkalen fosfataz 203 U/L (referans değer 73 U/L), GGT 482 U/L (referans değer 45-129 U/L) olarak sonuçlandı. Hastanın çalışılan viral serolojik testleri negatif ve hepatik ultrasonu normaldi. Antimitokondriyal antikor ve antinükleer antikor dahil otoantikorları negatif geldi. Tetkik sonuçları göz önünde bulundurularak ön planda amlodipine bağlı hepatit düşünüldü ve amlodipin kesildi. Amlodipin kesildikten bir hafta sonra karaciğer fonksiyon testleri normale döndü.

Tartışma: Amlodipin sık kullanılan bir antihipertansif ilaçtır ve genel olarak hastalar tarafından iyi tolere edilmektedir.Amlodipine bağlı hepatit nadir görülen bir yan etki olmakla birlikte genel olarak karaciğer fonksiyon testlerinde ılımlı bir yükseliş ile birliktedir.Klinik aşikar hepatit çok çok nadir görülmektedir ve literatürde sadece birkaç adet vaka bildirilmiştir.Serum enzim yüksekliği paterni genel olarak miks ve kolestatiktir. Hepatotoksisitenin mekanizması tam olarak bilinmemektedir fakat muhtemel sebep amlodipin metabolizması sırasında oluşan ara ürünlerdir. Biz bu yazımızda kliniğimizde yatarken amlodipine bağlı hepatit gelişen bir olguyu sunduk. Hastanın amlodipin kesildikten sonra serum enzim düzeylerinde normale dönüş olması ve hastanın enzim yüksekliğini açıklayacak başka etiyolojik neden bulunmaması tanımızı desteklemektedir.

OPS2-15

Dirençli hipertansiyon tedavisinde santral etkili antihipertansif kullanımı

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Selçuk Üniversitesi Selçuklu Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Konya

Otuz üç yaşında dirençli hipertansiyon nedeniyle dış merkezde 3 yıl önce sağ adrenokortikal adenom nedeniyle opere olan 2 yıl sonrasında hipertansiyon şikayetinin geçmemesi üzerine feokromasitoma araştırılan ve feokromasitoma saptanmayan hasta beşli antihipertansif tedavi almasına rağmen dirençli hipertansiyon nedeniyle kliniğimize başvurdu. Hasta valsartan+hidroklortıazıd 320/25 mg 1x1 nifedipin 120mg 1x1 spironolakton 100 mg 1x1 doksazosın 8 mg 2x1 metoprolol 100 mg 1x1 tedavisi almasına rağmen takılan tansiyon holter kaydında 24 saatlik ortalama kan basınıcı 169/114 mmhg olarak saptandı. Yapılan ekokardiyografisinde sol ventrikül ejeksiyon fraksiyonu: %60 sol ventrikül fonksiyonları normal ve hipertansiyona neden olacak sekonder patoloji saptanmadı. Renal renkli doppler USG de böbrek parankimleri toplayıcı sistem sürrenal lojlar normal ve her iki renal arter akımı normal olarak saptandı. Hastaya dirençli hipertansiyon tedavisinde santral etkili antihipertansif rezerpin 0,25 mg tb 1x1 başlandı. Kan basıncı takibinde tansiyonları 90/60 mmhg ya kadar düşen hastada kullandığı diğer antihipertansiflerin dozu azaltıldı ve kontrollü olarak rezerpin 2x1 olarak devam edildi. Valsartan+hidroklortiazid 320/25 mg takiplerde valsartan 80 mg a kadar düşülülerek kesildi. Nifedipin tedavisi 30 mg a düşürüldü. Doksazosın tedavisi 4 mg 1x1 devam edildi. Metoprolol tedavisi 25 mg a düşürülerek kesildi. Hastaya bu tedavi altında tansiyon holter takıldı. 24 saatlik ortalama kan basıncı 126/77 mghg olarak saptandı. Kliniğimizde tansiyon takipleri 130/80 mmHg altında olan hasta bu medikal tedavi ile taburcu edildi. Rezerpin adrenerjik nöron blokörüdür. Periferik sinir uçlarında katekolamın depolarını boşaltarak ya da katekolaminlerin burdan salınımını inhibe ederek etki eder. Rezerpin kullanımında ciddi ortostatik hipotansiyon bulantı kabızlık eklem ağrısı depresyon gelişebilir. Bizim hastamızda da depresif semptomlar gelişti. Psikiyatri kliniğine konsulte edilerek tedavisi düzenlendi. Biz bu vakamızda santral etkili antihipertansiflerin kombine tedaviye rağmen dirençli hipertansiyon tedavisinde son seçenek olarak kullanılabileceği belirtmek istedik.

OPS2-16

Using 0.014 inches guiding wire and coronary balloon to pass unopened left leg of endovascular stent during EVAR procedure

Cengiz Öztürk[1], Atila İyisoy[1], Mehmet Ali Sahin[2], Turgay Celik[1], Hamidullah Haqmal[1], Murat Tavlasoglu[2], Sevket Balta[1], Mustafa Demir[1], Faruk Cingoz[2], Siddik Erdogan[1]

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We report a 77 year-old woman patient was admited to our clinic with abdominal pain for a few days. The pain was tearing in nature and radiated to her epigastric region and back resolving with rest. Her medical history included coronary artery bypass and chronic obstructive pulmonary disease. No other cormobidities were present. His medication included acetylsalicylic acid and bronchodilator therapy. Her familial history was otherwise unremarkable. Pulse rate was 68 beat/minute and regular, arterial blood pressure was 120/80 mmHg, lungs showed diffuse chronic disease, breath rate was 24/pm, and no murmur on physical examination. WBC was 6300 e3/μL, HGB was 14.3 g/dL, kreatinin was 1.2 mg/dL and other electrolytes tests were normal. HSCRP was 30.76 mg/dL, sedimentation rate was 21 mm/hour. The electrocardiography revealed sinus rhythm with 80 beat/minut, ST-segment depresion in the inferior and posterior leads. Transthoracic echocardiography showed minimaly depressed left ventricle ejection fraction 54% and hypokinesia of inferior and posterior wall and grade I diastolic dysfunction. Abdominal aortic aneurysm was diagnosed with abdominal ultrasonography and was admited our department. Ultrasonography showed aneurysm with 60 mm and 45 mm focal intimal flep. For forther invastigation comuted were performed. There was an aortic aneurysm at the level of infrarenal level of abdominal aorta with a maximum diameter 47x50 mm with no extravasation below 2 mm of right renal artery on tomography angiography. The patient was referred to open surgery treatment, but surgery was presumed to it high risk. We decided to perform EVAR for further treatment. Under local anesthesia right femoral artery was used for delivery system and left femoral artery was used for aortography. Firstly an endograft was implanted to the infrarenal level of abdominal aorta via the right femoral artery (Medtronic Endurant II Stent Graft System) but the left leg was not opened. When we want implant the left leg of graft, the floopy wire and pigtail catheter was not forwarded through the left leg. We decide to perform use the 0.014" coronary wire and inflate a 3.0x20 mm coronary balloon to pass the unopened segment. Thanks to the 0.014 inches wire and coronary balloon, it was passed successfully and implanted the left leg of the grafted stent. The procedure was finished without any endoleak or vascular complication. The patient was transferred to the coronary care unit. The patient was discharged after four day with no complication. In conclusion, using of 0.014" coronary wire and coronary balloon is feasible and rational for this unwanted event as in our case.

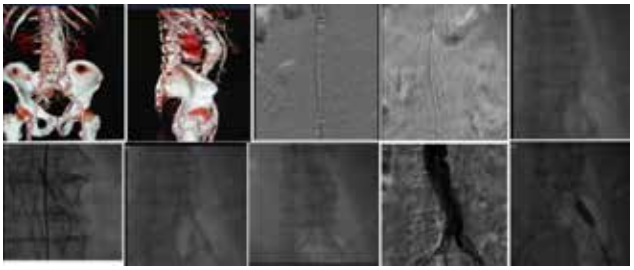

Figure 1.

OPS2-17

Surgical treatment of a coronary cameral fistula and coronary artery disease in a patient with non-compacted myocardium

Belma Kalaycı, Elif Coşkun, Ziyaeddin Aktop, Süleyman Kalaycı, Mustafa Büyükateş

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A 77 year old men admitted to the emergency department with acute chest pain. His past medical history included diabetes mellitus, hypertension, hyperlipidemia and smoking. Physical examination was normal. Electrocardiography was unremarkable on admission except ST segment depression in V5 and V6 leads. Troponin T level was measured 0,5 ng/dl (0-0.03 ng/dl). We planned to perform coronary angiogram in the patient with non-ST elevation myocardial infarction. Coronary angiogram showed totally occlussion in left anterior descending (LAD) artery and collaterals to LAD territory in right anterior oblique projection (Figure 1). Besides the angiogram revealed a large coronary camerial fistula between LAD artery and pulmonary artery (Figure 1). On angiogram left circumflex artery had significant stenosis in the middle segment and right coronary artery is nondominat and free-stenosis (Figure 2, 3). Left ventriculography revealed non-compacte myocardium as increased deep trabeculation communicated with the ventricular cavity (Figure 4). We decided to perform coronary artery bypass grafting and surgical ligation of the coronary artery fistula. Coronary artery bypass graft had done left internal mammary artery to left anterior descending artery and the saphenous venous graft to left circumflex artery. At the same session the coronary fistula was ligated (Figure 5). Coronary cameral fistula (CCF) is a rare congenital or acquired entity. They drain into the right-sided chamber or great vessel in approximately 90% cases. It is usually asymptomatic in majority of patients. In general CCFs are incidentally detected in coronary angiogram incidentally. Echocardiographic examination is usually sufficient for diagnosis of non-compacted myocardium. However he has no sufficient echocardiographic images due to his echogenicity was poor. Left ventriculography could be used for diagnosis as an alternative image method when the echocardiographic images are insufficient for diagnosis. We reported a case had severe coronary artery disease and CCF who had no-compacte myocardium. According to our literature research the coexistence of coronary cameral fistula, coronary artery disease and non-compacted myocardium was not reported previously. We reported a patient had coronary cameral fistula and severe coronary artery disease with non-compacted myocardium and the surgical treatment.

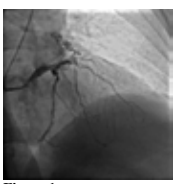

Figure 1.

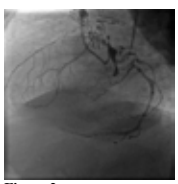

Figure 2.

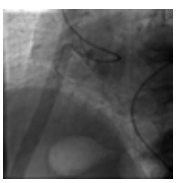

Figure 3.

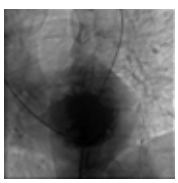

Figure 4.

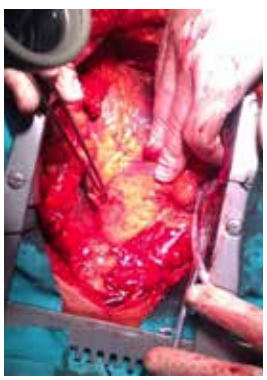

Figure 5.

OPS2-18

The rupture of ascending aorta with severe aortic regurgitation and pericardial effusion due to blunt trauma: A silent presentation

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Ascending aortic aneurysm is a serious illness which often undiagnosed due to being asymptomatic and if it ruptures death occurs in a very short time. The risk of rupture closely correlates with the diameter of aorta. We present a case of ruptured ascending aorta with severe aortic regurgitation after a blunt trauma with light intensity. A 66-year-old male patient with a complaint of vague pain on the chest after blunt trauma came to our clinics. The patient's chest was hit by steering wheel during car accident which did not cause any apparent injury to the patient. He was treated with a combination of angiotensin converting enzyme inhibitor and beta-blocker with the diagnosis of hypertension and ascending aortic aneurysm. He was diagnosed as ascending aortic aneurysm 4 years ago and his ascending aorta was calculated as 5.3 cm via transthoracic echocardiography (TTE) 2 years ago. He did not come to regular controls in spite of advice for routine echocardiographic examinations. Physical examination was nondiagnostic except deep heart sounds and weakly heard diastolic murmur at left sternal border. His blood pressure which measured from bilateral upper extremities were 127/72 and 131/81 mmHg. A fast bed-side TTE revealed severe aortic regurgitation, pericardial effusion and normal left ventricular systolic functions. The ascending aorta was measured as 5.6 cm via TTE. Contrast enhanced computed tomography showed the rupture of ascending aorta and also pericardial effusion. After an urgent consultation with cardiovascular surgeons, an urgent surgery decision was made. The patient underwent a successfull Benthall procedure which involves the replacements of aortic valve, aortic root and ascending aorta. Rupture of the ascending aorta caused by blunt trauma rarely has been diagnosed and treated. Careful examination and clinical suspicion are very important for the diagnosis. Close echocardiographic examinations are important and surgery should be performed when the upper limit was reached.

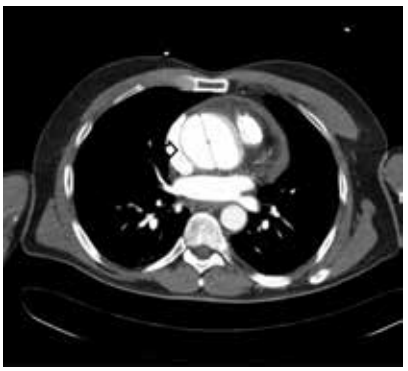

Figure 1. Contrast enhanced computed tomography shows the rupture of ascending aorta. Arrow indicates pericardial effusion secondary to the rupture of ascending aorta.

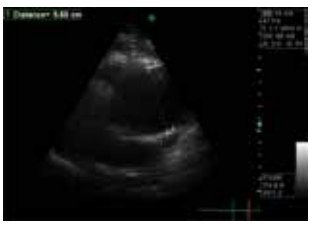

Figure 2. Transthoracic echocardiogram shows ascending aortic aneurysm.

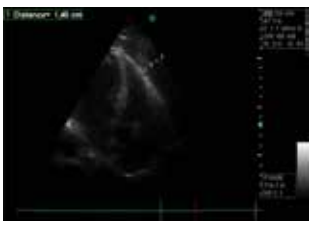

Figure 3. Transthoracic echocardiogram shows pericardial effusion due to the rupture of ascending aorta.

OPS2-19

Right sided endocarditis with multiple septic pulmonary embolisms in a injection drug user

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Introduction: Infective endocarditis have been described approximately 2 to 4 cases per 1000 of injection drug users. We report a case of right sided endocarditis with multple septic pulmonary embolisms in a injection drug user.

Case Report: A 20 years-old male drug user presented with hip and knee pain, fever,hemoptysis and dispnea for 2 week.In his physical findings, blood pressure is 110/60 mmHg pulse is 120 pbm,body temperature is 39 celcius degree. Lung sounds reveal bilaterally diffuse cracles. Heart sounds are rhtymic, no any murmurs was heard.There are petechial rash in dorsum of foot (Figure 1). RIE graphy shows diffuse infiltration in both lungs (Figure 2), Thorax computed tomography indicates multple septic embolic focuses in fields of both lungs (Figure 3). Transthoracic echocardiography shows mobile vejetation on tricuspide valve (Figure 4 and Video 1) No any microorganisms are isolated blood culture. The patient emprical administered vancomycine and imipenem intraveneously.

Discussion: Infective endocarditis have been described approximately 2 to 4 cases per 1000 of injection drug users. Many factors may role for pathogenesis of infective endocarditis in injection drug users. Particulate matter of ilicit drugs may damage to tricuspit valve. Repetetive use of drugs may cause cumulative endothelial damage to tricuspit valve. During drug injection,users may inject microroganisms present on the surface of the skin, in the drug itself, or in diluents. S.aureus is the most common agent and it is responsible for half of cases. Right sided endocarditis is less common than left sided in general population. Most of cases are injection drug users. Right sided endocarditis relatively have a good prognosis than left sided endocarditis. Large vegetation size and fungal etiology are poor prognostic indices.

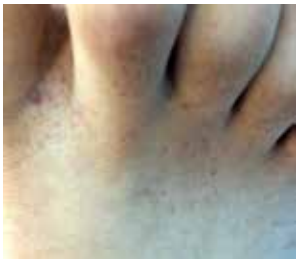

Figure 1. Shows petechial rash on dorsum of foot.

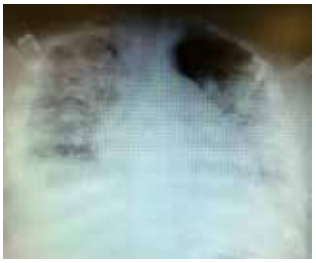

Figure 2. RIE graph shows diffuse bilaterally pulmonary infiltration.

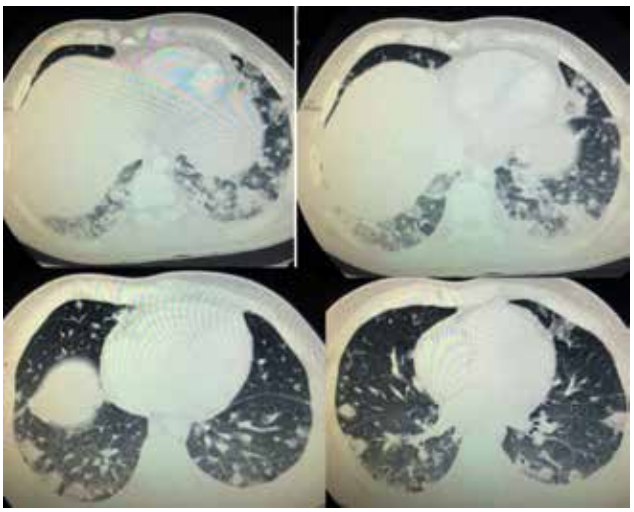

Figure 3. Thorax computed tomography shows septic embolic focuses in both lungs.

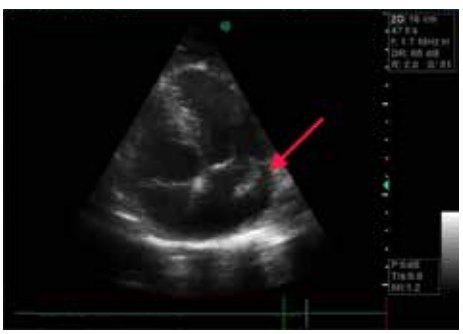

Figure 4. Transthoracic apical four chamber view; arrow indicates vegetation on tricuspid valve.

OPS2-20

Multipl septik pulmoner emboli ile tanı alan triküspit kapak endokarditi

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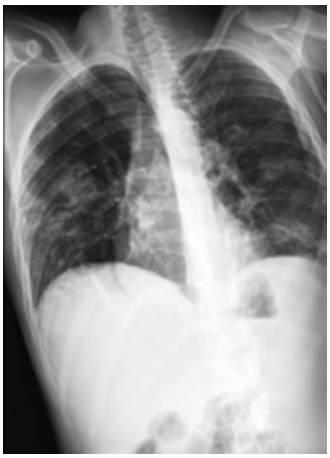
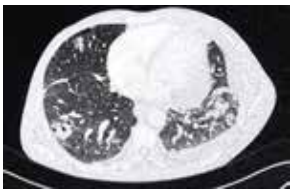
Giriş: Yasa dışı ilaç kullanımı serebral iskemi,beyin absesi,enfektif endokardit (EE) gibi fatal nörovasküler ve kardiyovasküler komplikasyonlara sebep olmaktadır. Fatal komplikasyonlar genel olarak bu hastalardaki immunyetmezlik ve enfektif hastalıklar ile ilişkilidir. EE kardiyovasküler yapıların veya intrakardiyak cihazların mikrobiyal enfeksiyonudur. EE yasa dışı ilaç (YDİ) kullanan hastalarda sık görülen bir komplikasyondur. Biz de bu yazımızda YDİ kullanımı ve multipl septik pulmoner embolisi (PE) olan bir EE olgusunu sunduk.

Olgu Sunumu: Otuz üç yaşında erkek hasta, acil servise ateş, nefes darlığı ve balgam şikayeti ile başvurmuş. Özgeçmişinde iv eroin kullanımı, sigara içiciliği ve dört yıl önce araç içi trafik kazası sonrası gelişen pelvik fraktür öyküsü mevcut. Hasta ara ara buprenorfin ve naloksan kullanmakta. Solunum sistemi muayenesinde sağda orta zonda kaba ral ve her iki akciğerde yaygın ronküs saptanmış. Hastanın ateşi 39.5 derece ve kalp hızı 120/dk idi. Kardiyovasküler muayenede belirgin özellik yokmuş. Akciğer grafisinde sağda infiltrasyon olması üzerine hasta göğüs hastalıklarına pnömoni ön tanısı ile yatırılmış (Şekil 1). Ampirik antibiyotik tedavisi olarak ampisilin/sülbaktam ve klaritromisin başlanmış. Kontrol akciğer grafisinde sol akciğerde lokule plevral sıvı saptanması üzerine torasentez yapılmış ve geleni olmamış. Bunun üzerine göğüs cerrahisi ile tüp torakostomi için görüşülmüş ve torakal tomografik anjiyo önerilmiş. Tomografi sonucunda her iki pulmoner arter, ana dalları ve segmenter dallarında PE ile uyumlu dolum defekti saptanmamış. Periferik yerleşimli kavitasyon içeren konsolide alanların varlığı septik emboli olarak yorumlanmış (Şekil 2 ve 3).Bunun üzerine hastaya ekokardiyografi planlanmış ve tarafımızdan görüş istenmiş. Yaptığımız transtorasik ekokardiyografide triküspit kapakta 15x14 mm boyutunda vejetasyon olduğu görüldü (Video 1 ve 2). Hastanın kan kültüründe Staphylococcus Aureus üremesi oldu ve hastaya EE tedavisi başlandı.

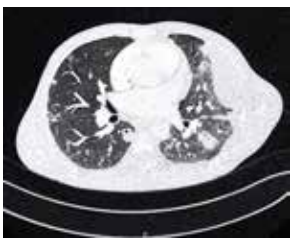
Tartışma: iv YDİ kullanan kişilerde enjeksiyon sırasında virulan mikroorganizmaların kan akımına direkt olarak enjeksiyonu olmaktadır. Bu kişilerde EE nedenli ani ölüm riski madde kullanımı olmayan kişilere göre 300 kat artmıştır. Hastaların yarısında etken S. Aureus'tur ve sıklıkla triküspid kapak tutulur. Klinik prezentasyon tutulan kapağa göre değişmektedir. Sol taraf kapak tutulumlarında multipl sistemik embolizm, organ abse ve enfarktları meydana gelebilir. Sağ taraf kapak tutulumunda ise PE ve parodoksal embolizm olabilmektedir. Multipl PE veya triküspid kapak destrüksiyonu sonucunda sağ boşluklarda dilatasyon görülebilmektedir. Bu hastalarda antibiyotik tedavisi sonrasında kür sağlanabilse de zor eredike edilen mikroorganizmaların varlığı veya 7 günden fazla bakteriyemi olması, rekürren PE sonrasında 20 mm üzerinde vejetasyon olması, diüretik tedaviye cevap vermeyen ciddi triküspit yetmezliği nedenli sağ kalp yetmezliği bulunması, rekürren PE ve paradoks emboli olması durumunda cerrahi tedavi gerekmektedir.



Şekil 1. Akciğer grafisinde sağda infiltrasyon görülmekte.



Şekil 2. Kavitasyon içeren konsolide alanlar.



Şekil 3. Konsolide alanlar ve solda plevral mayi.

OPS2-21

Mitral mekanik anüler trombüslü hastada multimodalite görüntülemeler kılavuzluğunda çok yavaş düşük doz doku tipi plazminojen aktivator infüzyon ile brakiyal arter tromboembolizmin başarılı tedavisi

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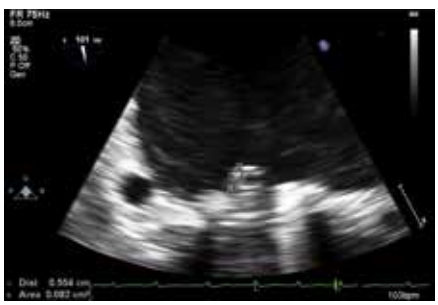
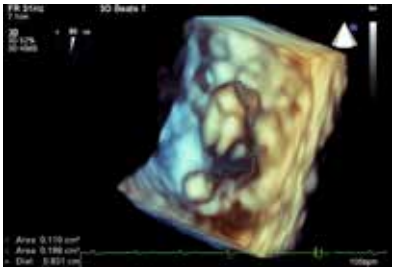
Sağ kol ve elde ağrı ve solukluk şikayeti ile 55 yaşında kadın hasta polikliniğimize başvurdu. Özgeçmişinde yaklaşık 15 yıl önce mitral mekanik protez kapak replasmanı, 9 yıl önce geçici iskemik atak, 4 yıl önce sağ-sol alt ekstremite arteryel embolektomi, renal tromboembolizm, hipertansiyon ve diabetes mellitus öyküsü var. Fizik muayenesinde sağ brakiyal, ulnar ve radiyal arterlerde nabızlar zayıf hissedildi. Labaratuar değerleri normal saptanıp, INR 1.8 idi. Üst ekstremite Doppler ultrasonografide sağ brakiyal arter distalinde fokal, hiperekoik trombüs materyali izlendi. Brakiyal arter seviyesinde minimal akım görüldü. Sağ üst ekstremite arteryel anjiyografisinde brakiyal arterde ve ulnar artere uzanan trombüsle uyumlu görünüm izlendi (Video 1). Transtorasik ekokardiyografide (TTE) mitral mekanik kapak gradienti 12/5 mm Hg kapak alanı (pht) 2.8 cm^2 ölçüldü. Transözafageal ekokardiyografide (TEE) posterior menteşe arkasında iki boyutlu (2D) TEE ile 5 mm uzunluğunda 0.1 cm^2 alanında, üç boyutlu (3D) TEE ile 9 mm uzunluğunda 0.2 cm^2 alanında 3 mm mobil parçası olan anüler trombüs izlendi (Şekil 1, 2, Video 2). Hastaya 25 mg/25 saatte çok yavaş düşük dozda doku tipi plazminojen aktivator (t-pa) infüzyonu uygulandı. Tedavi sonrası hastanın sağ kol ve elinde olan ağrı, solukluk ve uyuşma şikayetleri geriledi ve brakiyal, ulnar ve radiyal arter nabızları dolgun palpe edildi. Kontrol TEE de mekanik mitral kapak üzerinde trombüs izlenmedi (Video 3, 4). Çok kesitli bt anjiyografide brakiyal arterde akımı kısıtlamayan trombüs izlendi (Şekil 3). Etkin varfarin tedavisi sonrası hasta taburcu edildi. Mitral mekanik protez kapak trombozuna bağlı gelişen brakiyal arter tromboembolisimi üst ekstremite Doppler ultrasonografi, çok kesitli bt anjiyografi, üst ekstremite arteryel anjiyografi, 2D ve gerçek zamanlı 3D TEE içeren multimodalite görüntülemeler kılavuzluğunda çok yavaş düşük doz t-pa infüzyonu ile tedavi edilebilir.



Şekil 1. İki boyutlu TEE de mitral mekanik protez kapakta trombüs. Transözafageal ekokardiyografide (TEE) posterior menteşe arkasında iki boyutlu (2D) TEE ile 5 mm uzunluğunda 0.1 cm^2 alanında trombüs izlendi.

Kalp kapak hastalıkları



Şekil 2. Üç boyutlu TEE de mitral mekanik protez kapakta trombüs. Üç boyutlu (3D) TEE ile 9 mm uzunluğunda 0.2 cm^2 alanında 3 mm mobil parçası olan olan anüler trombüs izlendi.

Şekil 3. Trombolitik sonrası rezidüel brakiyal trombus (ok). Çok kesitli bt anjiyografide brakiyal arterde akımı kısıtlamayan trombüs izlendi.

OPS2-22

Erken dejenere TAVİ kapağı içine tekrar TAVİ işlemi

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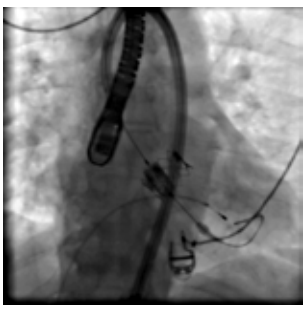
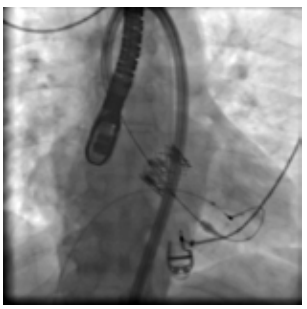
Yetmiş altı yaşında bayan hasta nefes darlığı şikayeti ile başvurdu. 22 ay önce ciddi romatizmal aort darlığı sebebiyle tarafımızca hastaya perkütan femoral yolla 23 numara biyoprotez aort kapağı (edwars saphien) implante edilmişti. Diyebetes mellitus için oral antidiyabetik kullanan hasta atriyal fibrilasyonu için yeni oral antikoagulan kullanıyor. Hastanın fizik muayenesinde mezokardiyak odakta 3/6 diyastolik üfürüm mevcuttu. Hastaya transtorasik ekokardiyografi yapıldı. Valvuler ciddi aort yetersizliği izlendi. Etyoloji için yapılan kan tahlillerinde enfeksiyon lehine bulgu izlenmedi. Kapak anatomisini değerlendirmek için transözofageal ekokardiyografi yapıldı. paravalvuler kaçak izlenmedi ancak valvuler ciddi aort yetersizliği görüldü (Video 1). kapakta trobüs pannus lehine bulgu izlenmedi. Hastanın semptomatik olması ve sık sık dekompanse olması üzerine hastaya cerrahi AVR önerildi. Hastanın işlem ve kapak ücretini karşılayacağının beyan etmesi ve cerrahi AVR işlemini kabul etmemesi üzerine hastaya tekrar perkütan yolla eski biyoprotez (TAVİ) kapağının içine femoral yolla biyoprotez kapak takılması planlandı. Daha önceki 23 numara kapağın için yine femoral yolla 23 numara (edwars saphien) kapak implante edildi (Video 2, 3). işlem sonrası yapılan kontrol eko da aort yetersizliği izlenmedi. Hastanın şikayetleri geriledi hasta taburcu edildi. Ciddi aort darlığında STS, Euroscore veya anatomik özelliklerine göre yüksek riskli hastalara TAVİ işlemi yıllardır başarı ile uygulanmaktadır. Hastamıza da daha önce ciddi aort darlığı ve yüksek riskli cerrahi olması üzerine TAVİ işlemi uygulanmıştı. Hastanın 1 yıl önce bakılan ekosunda kapakta hafif kaçak varken toplam 22 ay sonra endokardit, tromboz, düzensiz tedavi gibi anamnezler olmaksızın ciddi Aort yetersizliği gelişmesi literatürde sık rastlanmayan bir durumdur. Biyoprotez kapakta erken dejenerasyon olarak vakanın bildirilmesi gerektiğini düşündük. Etyolojik olarak bir sebep bulunmamaktadır. Hasta tedavi olarak (metformin 2x1000 mg rivaroksaban 20 mg, bisoprolol 5 mg, rosuvastatin 20 mg alıyor hgba1c düzeyi %7). Ayrıca aort yetersizliği tanısıyla biyoprotez kapak içine tekrar TAVİ işlemi ile biyoprotez kapak takılarak başarılı tedavi hasta için yüksek konfor ve ciddi klinik rahatlama sağlamıştır. Hasta 4 aydır takip edilmekte ve klinikte bozulma izlenmemiştir.



Şekil 1. Eski tavi protezi için yeni kapak yerleştirilmesi.



Şekil 2.

OPS2-23

Acquired warfarin resistance in a patient with mitral valve replacement

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Warfarin is an oral anticoagulant, that is widely used for the prevention of thromboembolic complications. But, there is large inter-individual differences in the dose required for its anticoagulation effect. Additionally some patients are warfarin resistant, as a result of genetic variability, drug interactions, dietary habits, malabsorbtion or also idiopathic etiology. We describe a case of acquired warfarin resistance. A 37-year-old woman with mitral valve replacement(MVR) was accepted to our hospital to adjust the dose of warfarin. Since 2005 she had had a prosthetic mitral valve and had taken warfarin. Until 2 years ago, her INR value had been effective, while she twas taking 5 mg warfarin on one day and 2.5 mg warfarin on the other day. However, her INR values suddenly decreased to ineffective levels two years ago. Therefore daily warfarin dose had been increased up to 20 mg per day. In spite of this, INR values had not become effective. For this reason, tinzaparin had been added to the therapy. Despite receiving 20 mg warfarin per day her INR value was 1,14 at the admission. Complete blood count, renal function tests, TSH level and lipid profile were normal. She said that she had avoided food rich in vitamin K, had taken warfarin tablets every morning at the same time, had not used any drug, including herbal or dietary supplements and alcohol, other than ASA and Tinzaparin. Also, she had not had any illnes previously diagnosed. It was suspected that this patient may fit into the group of genetic resistance to warfarin. Genetic test was performed in the blood. But, variant alleles detected in our patient were not sufficient to explain high-dose warfarin requirement. Her INR values were assessed every day with the increase in the daily dose of warfarin (Table 1). As it was seen in the table, warfarin resistance was broken after a certain point and we were able to reach the targeted INR level with usual doses. After the discharge we learned by phone that INR measurements of our patient's were in therapeutic range while taking 5 mg warfarin on one day and 2.5 mg on the other day. This case summarize an acquired form of warfarin resistance, which appeared independent of medication, diet and comorbid disease. This case also emphasize the possibility of breaking warfarin resistance with increasing dose. After this breaking point, the need of warfarin can decrease to usual dose and the patient can be protected against adverse effects of high amount of warfarin.

Table 1. (A, B) The table shows daily warfarin dose and the corresponding INR value.

(A) Date	Dose(mg/day)	INR
04.07.2011	25	1,14
05.07.2011	30	1,26
06.07.2011	40	1,3
07.07.2011	50	1,48
08.07.2011	60	1,54
09.07.2011	80	1,56
10.07.2011	90	1,99
11.07.2011	75	3,12
12.07.2011	-	6,35
13.07.2011	-	6,08
14.07.2011	-	3,72
15.07.2011	75	1,84
16.07.2011	75	1,62
17.07.2011	75	2,78
18.07.2011	-	4,8
19.07.2011	-	4,73
20.07.2011	50	2,92
21.07.2011	50	2,66
22.07.2011	-	3,99

(B) Date	Dose(mg/day)	INR
23.07.2011	-	5,23
24.07.2011	30	3,08
25.07.2011	40	2,34
26.07.2011	-	3,78
27.07.2011	-	4,47
28.07.2011	20	2,86
29.07.2011	20	2,05
30.07.2011	20	2,89
31.07.2011	10	3,75
01.08.2011	-	6,27
02.08.2011	-	4,54
03.08.2011	-	4,07
04.08.2011	5	2,61
05.08.2011	5	1,86
06.08.2011	2,5	2,91
07.08.2011	5	2,69
08.08.2011	2,5	3,12
09.08.2011	5	3,2
10.08.2011	2,5	2,86
11.08.2011	5	2,9

OPS2-24

Native valve endocarditis by pseudomonas aeruginosa in a non-drug user patient: A rare presentation

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Infective endocarditis (IE) is an inflammation of the endocardium, the inner lining of the heart and heart valves. IE is known highly morbid and mortal infection. Gram positive microorganisms such as: staphylococci, streptococci and enterococci are the most common causative agents for IE. Gram negative bacteria rarely cause IE. Pseudomonas aeruginosa is a typical microorganism in the hospital settings and it is a frequent cause of nosocomial infections. Endocarditis caused by this pathogen is very serious with a high mortality rate. Treatment of pseudomonas aeruginosa endocarditis usually necessitates the combination of antibiotics and surgery to achieve effective eradication of the culprit. More than 200 cases of pseudomonas aeruginosa endocarditis have been reported. In the most of these patients, intravenous drug usages have been reported. We present a rare case of pseudomonas aeruginosa endocarditis in a non-drug user patient. A 62 year-old female patient was presented to our emergency department with complaints of progressive fever up to 39 C, shortness of breath, disorientation and generalized weakness for last five days. Her medical history was significant for diabetes mellitus type 2 and chronic kidney failure. In her physical examination; blood pressure was 94/46 mmHg, heart rate was regular and 102 bpm. In auscultation; 4/6 systolic murmur at left parasternal side and bilateral fine crackles in lungs was heard. A fast bedside transthoracic echocardiography revealed mobile vegetation above native mitral valve of 4.0x1.0 cm in size. After blood samples were taken, an empiric treatment of ampicilin+sulbactam, gentamicin and vancomycin was started. Transesophageal echocardiography was also done, demonstrating severe mitral valve insuffiency with suspected perforation of the anterior mitral leaflet as well as vegetation consistent with the findings in the previous transthoracic echocardiography. Laboratory tests were remarkable for a white blood cells count of 11.90 K/mm^3 (normal range 4.6-10.2 K/mm^3), C-Reactive Protein of 134 mg/dl (normal range 0-0.5mg/dl), platelet count of 39.600 K/mm^3 (normal range 150.000-400.000 K/mm^3). Splenic infarction was evidenced in abdominal US. Blood cultures yielded growth of Pseudomanas aeruginosa. Antibiotic treatment with cefepim + amikacin was adjusted according to the microbiological testing. 8 days after admission, the patient was taken to surgery for mitral valve replacement, although the patient continued to be thrombocytopenic despite platelets apheresis. Unfortunately, at 13th post-operative day, the patient died due to septic shock.

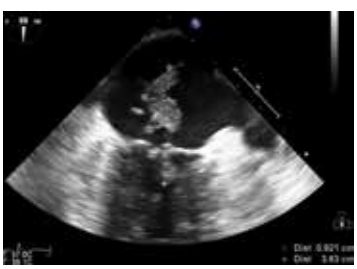

Figure 1. Two chamber transesophegeal echocardiogram shows the vegetation in 36x9 mm dimensions originating from native mitral valve.

OPS2-25

Double-valve infective endocarditis complicated by a ruptured sinus of valsalva aneurysm

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An 82-year-old man undergoing haemodialysis was referred to outpatient clinic with a history of high fever (39°C) and thrombocytopenia. High serum C- reactive protein level (251 mg/dl) and white blood cell count (17300/μl) were also accompanied. The patient had a previous suspicious infective endocarditis diagnosis. A mid-diastolic murmur of a grade 3/6 was found near the aortic foci in his cardiac auscultation. The blood pressure was 100/50 mmHg and heart rate was 95 beat/min in admission. These findings suggested an initial diagnosis of infective endocarditis. All blood cultures were positive for Staphylococcus haemolyticus. To confirm the diagnosis, transthoracic echocardiography was performed. Transthoracic echocardiography revealed multiple vegetations of the aortic (maximum dimension 18 x 11 mm) and tricuspid valves (maximum dimension 11 x 9 mm) (Figure 1, Video 1). These findings were confirmed by transesophageal echocardiography, which also disclosed a ruptured sinus of Valsalva aneurysm and a shunt from aorta to the right atrium (Figure 2, Video 2). The patient was referred for cardiac surgery. The infected aortic and tricuspid valves replaced with mechanical valves and ruptured sinus of Valsalva aneurysm was repaired. After the completion of antimicrobial therapy, patient was discharged from hospital. At follow-up visits, his clinical situation is going well after the procedure. Infective endocarditis is a challenging clinical situation that causes a lot of complications. Some of the complications treated medically but most of them treated surgically. One of the complications is ruptured sinus of Valsalva aneurysm and its prevalence is less than 1%. Most of sinus Valsalva aneurysms arise from right coronary sinus and rupture into the right atrium as in our clinical case. The manifestations appear depending on the site where the sinus has ruptured. Main diagnostic method is echocardiography in these situations. When necessary, additional tools like magnetic resonance imaging, computed tomography and cardiac catheterization can be used. After confirming the diagnosis, treatment has to be started immediately. Medical management usually involves stabilization (eg, optimizing medications for heart failure), perioperative assessment and management. Appropriate antibiotics have to be continued as a result of blood cultures sensitivity test. Immediate surgical procedures should be considered when anatomical complications accompanied by clinical deterioration are seen.

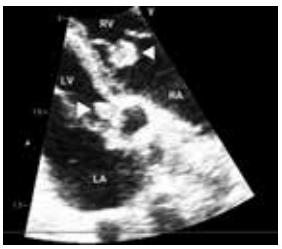

Figure 1. Two-dimensional subcostal transthoracic echocardiography view showing simultaneous aortic and tricuspid valve endocarditis (white arrowheads). RA=Right atrium; LA=Left atrium; RV=Right ventricle; LV=Left ventricle.

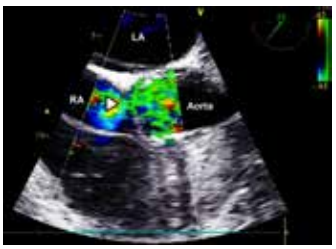

Figure 2. Two-dimensional transesophageal color Doppler echocardiography view showing the turbulent flow from the aorta to the right atrium through the perforation in sinus of Valsalva (white arrowhead). RA=Right atrium; LA=Left atrium.

OPS2-26

Primary cardiac lymphoma

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A previously healthy 32-year-old woman was admitted shortness of breath and palpitation.She had initially developed chest discomfort and increasing shortness of breath 4 weeks before admission. On presentation, the patient was afebrile, had an oxygen saturation of 92% on room air, and was tachycardic with a blood pressure of 115/67 mmHg and was notable for jugular venous distention. Her heart rhythm was regular with normal S1 and S2 and 3/6 continuous murmur on mesocardiac focus.A chest radıography showed no an enlarged heart. Her ECG was notable for sinus rhythm at 83bpm,incomplete right bundle branch block. An initial bedside transthoracic echocardiography showed ejection fraction (EF) 65%, no regional wall motion abnormalities left ventriculer hypertrophy, mild-to-moderate tricuspid regurgitation, subaortic ventricular septal defect and 45x 49 mm mass in the right atrium (Figure 2). A computed tomography scan revealed a large mass in the right atrium (Figure 1). Patients were consulted on cardiovascular surgery. Median sternotomy was performed. After heparinisation an arterial canula was inserted into the ascending aorta and a venous cannula was inserted into the superior vena cava.A cannula could inserted femoral vena because the mass was extended vena cava inferio (IVC). Cardiopulmonary bypass (CPB) was started, under normothermıc CPB a vertical atriotomy was performed and tumour was found in the right atrıum. The tumour was attached to the right atrıum wall and was extending IVC. The tumour was pulled out right atrium and primum atrial septal defect (ASD) was seen. Primum ASD closed with pericardial petch. Mezensimal tumour was detected in the frozen study (Figure 3). The atrial incision was quickly closed after surgery control echocardiography revealed no visible tumour. İmmunohistochemical analysis showed the neoplastic cells were positive for CD30 and CD 4 anaplastic large cell non-hodgkin lymphoma (Figure 4). Stains were negative for CD43, Pax5, CD117, CD31, CD34, ALK, desmin and keratin. The patient uneventful postoperatıve course and was discharged 7 day later. Positron emission tomography confirmed cardiac isolation.There were no evidence of extra-cardiac lymphoma present, thus the final diagnosis was primary cardiac lymphoma. When the patient recovered from the surgery, she decided to continue her chemotherapy treatment at a foreign oncology clinic.

Figure 1. Large mass in the right atrium.

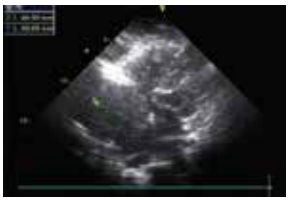

Figure 2. Subaortic ventricular septal defect and 45x49 mm mass in the right atrium.

Figure 3. Macroscopic assesment.

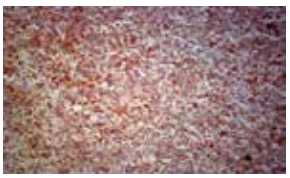

Figure 4. CD30 (+) anoplastic large cell non-hodgkin lymphoma.

OPS2-27

Replase aort kapak infekif endokarditi zemininde gelişen psödoanevrizma

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Yedi sene önce prostetik aort kapak replasmanı olan 38 yaşında erkek hasta burun kanaması ve 5 gündür yüksek seyreden ateş ile acil servise başvurdu. Hasta infektif endokardit ön tanısı ile yatırıldı. Hastanın yapılan transtorasik ekokardiyografisinde (Video 1) protez kapağın septum komşuluğunda dehisens olduğu ve bu bölgeden aort duvarının içine doğru uzanan 5.7x1.9 cm boyutlarında psödoanevrizma ile uyumlu bir yapı olduğu gözlemlendi. Dehisens olan bölgede psödoanevrizma içine akım mevcut ve vejetasyon ile uyumlu 4-5 mm çapında fibriler yapılar mevcuttu. Ampirik endokardit tedavisi için enfeksiyon hastalıkları tarafından teikoplanin ve ertapenem başlandı. Hastann yapılan trasnözefagial ekokardiyografisinde (Video 2) aort sol atriyum ve septum komşuluğunda psödoanevrizma ile uyumlu görünüm mevcut olduğu ve diseksiyonun olmadığı gözlemlendi. Hasta kalp-damar cerrahisi ile konsülte edilerek ameliyat kararı alındı. İnfektif endokardit (İE) kalp endotelinin bir enfeksiyonu olup yüksek mortalite ve morbidite riski olan ciddi bir hastalıktır. Protez kapağı olan hastalar İE açısından yüksek riskli hastalardır. İnfektif endokarditli olguların yaklaşık %10-30'u protez kapak endokarditli hastalardır. Protez kapak endokarditinde mortalite %20-80 arasında değişmektedir. Protez kapak infektif endokardit komplikasyonları; vejetasyon, protez kapak yetersizliği ve/veya ayrışması, apse formasyonu, fistülizasyon ve psödoanevrizma oluşumudur. Psödoanevrizmalar ince bir dokuyla ayrılmış, kardiyak siklüs boyunca karakteristik pulsatil hareket gösteren ve sol ventrikül çıkış yolu ile bağlantılı ekolüsen boşluklardır. Aort kökünün korunduğu durumlarda genellikle kapak ve aort kökü arasındaki dikiş hattında ayrışma, aort ile kapak grefti arasında kan geçişine bağlı ve genişleme eğiliminde bir boşluk ile sonuçlanabilir. Protez kapağı olan hastalarda süreğen ateş öyküsünün olması durumunda infektif endokardit akla gelmesi gereken önemli bir tanıdır. Ciddi komplikasyonlarının olduğu unutulmamalı ve hastanın tedavisine ivedilikle başlanmalıdır.

OPS2-28

Concomitant LAD and diagonal myocardial bridge in a patient with hypertrophic cardiomyopathy

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A myocardial bridge is found nearly in 10% of all coronary angiography procedures. Almost all of the bridges involve the left anterior descending coronary artery (LAD) and they are very rarely seen in the diagonal artery. Concomitant myocardial bridging is a very rare condition. Here we describe myocardial bridge involving both LAD and diagonal artery. 33 years old male patients was admitted to emergency department with presyncope. His ECG revealed inferior ST segment elevation and and left ventricular hypertrophia.his blood tests were unremarkable and the troponin values were normal. Transthorasic echocardiography revealed obstructive hypertrophic cardiomopathy (septal thickness 38 mm, LVOT peak systolic gradient 68 mmHg). Coronary angiography revealed both LAD and diagonal musculary bridge (Figure 1a, b). Holter recording revealed couplet ventricular premature complexes bot no nonsustained ventricular tachycardia. Patient's functional class was class 1. A decision was made to implant a DDD ICD for primary prevention against SCD. Myocardial bridging is defined as the tunneling of an epicardial coronary artery through the myocardium. The vast majority of the cases are seen in LAD. Myocardial bridging at the other coronary territories is very rare. The interesting feature of our case is the myocardial bridging seen both in LAD and diagonal artery at the same time.

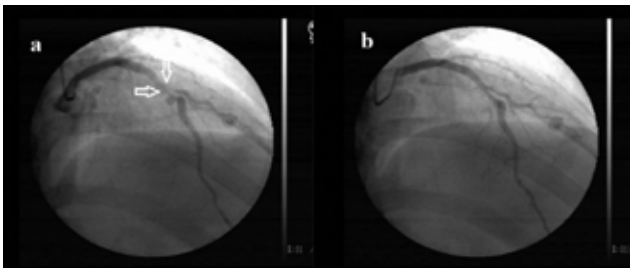

Figure 1. Myocardial bridging **(A)** systole **(B)** diastole.

OPS2-29

Percutaneous coronary intervention to left anterior descending artery in a patient with undeveloped right coronary artery and superdominant circumflex artery

Cengiz Ozturk, Hamidullah Haqmal, Turgay Celik, Ali Osman Yildirim, Mustafa Demir, Zekeriya Arslan, Murat Unlu, Atila İyisoy

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We reported a case of intervention to the narowed left anterior desending artery (LAD) due to the acute coronary syndrome with a right coronary artery (RCA) arising from circumflex artery (LCX) and superdominant left circumflex coronary artery continued as right coronary artery. A 82 year-old female patient was admitted to our outpatient clinic with past medical history of diabetus mellitus, evaluation of an exertional dyspnea and complaint of effort induced class II angina for 3 days. Transthoracic echocardiography showed anterior wall hypokinesia and mildly depressed left ventricular ejection fraction (55%), mild tricuspid and tricuspid valve insufficiency. There was 2/6 grade systolic murmur in the mesocardiac area and the lung was normal on physical examination. Cardiothoracic index was normal at X-ray. Systemic blood presure was 130/75 mmHg and pulse rate was 78 beat/minute and regular. Biochemical results such as complete blood count, sedimentation rate and other electrolytes tests were normal except elevated cardiac biomarker (troponin T, CKMB). The 12-lead electrocardiography revealed sinus rhythm with a rate of 103 beat per minute, diffuse ST segment depresion in the chest leads V1-V6. The patient was diagnosed with non ST segment elevation myocardial infarction (NSTEMI) and she was transferred to the angiography laboratory. Coronary and aortic root angiography did not show a vessel originating from the right coronary cusp. The left main coronary artery (LMCA) originated normally from left coronary sinus and divided into LAD and LCX arteries. LCX continued as right coronary artery and there was no RCA. The RCA was originated from LCX coronary artery and it was a terminal excursion of LCX. There was severe and diffuse lesion on LAD. Patient refered to open surgery treatment, but surgery was presumed to be high risk. We decided to perform percutaneous intervention for further treatment. The LAD was succesfully treated with use of two drug eluting stent without any complication.

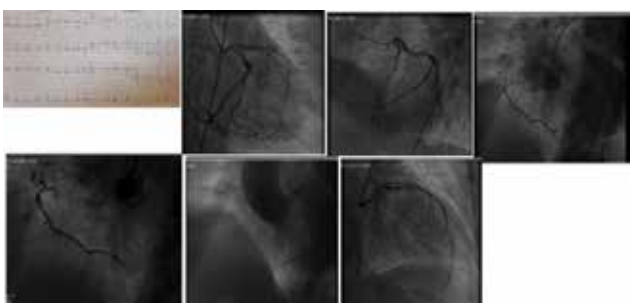

Figure 1.

Tartışma: Nadir bir konjanital kalp hastalığı formu olan koroner arter anomalileri çoğunlukla asemptomatik olmaları nedeniyle rastlantısal olarak saptanmaktadırlar. Ancak angina, senkop, disritmi, miyokard infarktüsü hatta ani ölüm ile klinik prezente olabilirler. Hastamızda efor anginası sebepli yapılan koroner anjiografi sırasında saptanan koroner anomali genel olarak benign gruptadır. LCx yokluğu görülme sıklığı olarak ~%0.003 olmakla birlikte koroner anomalilerdeki oranı ~%2'dir(3). Bu oranlarla en nadir rastlanan koroner anomaliler içerisinde sayılabilir. Koroner anomalilerinin malign çeşitlerinde tedavi cerrahi düzeltmedir. Ancak cerrahi tedavi endikasyonu olmayan bening seyirli anomalilerde her ne kadar tedavi prosedürleri net olmasa da anginal semptomların olduğu hastalarda nitrogliserin tedavisi ile koroner vazodilatasyonu sağlamak yararlı olabilir.

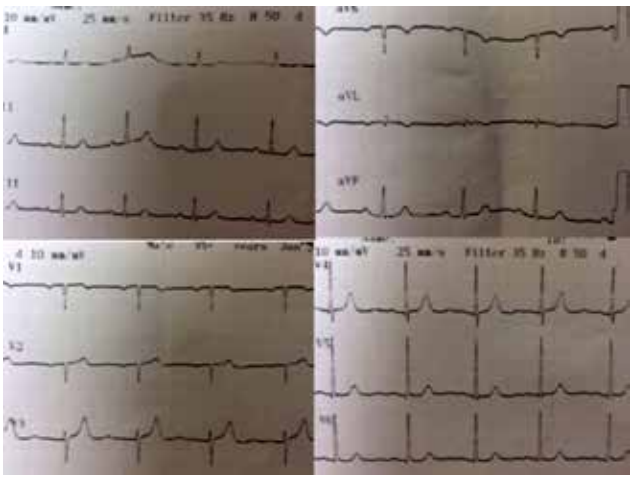

Figure 1.

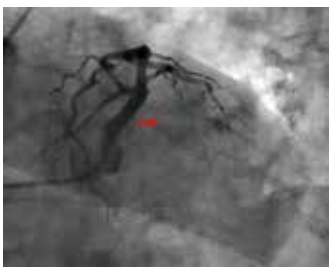

Figure 2.

Figure 3.

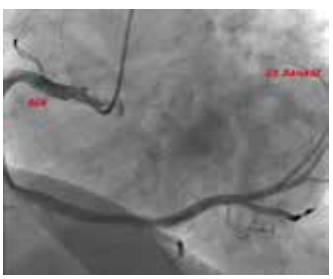

Figure 4.

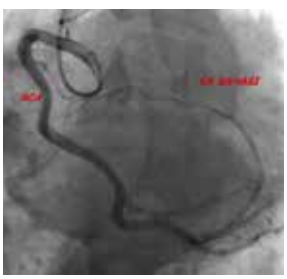

Figure 5.

OPS2-30

Nadir sayılabilecek bir koroner arter anomalisi, LCx yokluğu

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Trakya Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Edirne

Giriş: Koroner arter anomalileri anjiografik olarak ~%1 sıklıkta(1), otopsi serilerinde ise ~%3(2) sıklıkta rastlanan bir durumdur. Koroner çıkış yeri anomalileri çoğu zaman benign seyretmekle beraber klinik olarak değişik prezentasyonlarda karşımıza çıkabilirler.

Olgu Sunumu: Altmış beş yaşında bilinen koroner arter hastalığı hikayesi olmayan hasta, aralıklı olan CCS class 1-2 efor anginasının son dönemde CCS class 3 olması üzerine tarafımıza başvurdu. Elektrokardiyogramında normal sinüs ritmi saptanan hastada patolojik özellik saptanmadı(Şekil 1). Bunun üzerine hastaya ekokardiyografi planlandı. Ekokardiyografide sol ventrikül tip 1 diyastolik disfonksiyon ve sol ventrikül konsantrik hipertrofi dışında özellik olmayan hastaya iskemi araştırması için elektif koroner anjiografi planlandı. Hastaya yapılan sol diyagnostik koroner anjiografide LAD'nin sol valsalvadan tek başına çıktığı mid segment plaklı olduğu saptandı (Şekil 2, 3) ve LCx'in olmadığı görüldü. Sağ diyagnostik koroner anjiografide ise LCx'in RCA'nın terminal uzantısı olarak devam ettiği saptandı (Şekil 4, 5). RCA plaklı saptandı. Koroner arter anomalisi saptanan hastaya medikal takip kararı alındı ve vazodilatatör tedavi başlandı. Vazodilatatör tedaviden yanıt alındı.

OPS2-31

A case of rare anomaly of isolated right ventricular apical non-compaction

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Non-compaction of ventricular myocardium (NCVM) is a rare genetic disorder caused by intrauterine arrest of endomyocardial development. Left ventricle is the usual site of NCVM with very rare reports of isolated right ventricular non-compaction (IRVNC). We describe a case of asymptomatic IRVNC.

Introduction: Isolated right ventricular noncompaction (IRVNC) is a very rare disorder caused by the intrauterine arrest of endomyocardial development and affects both children and adults. The cardinal manifestations of IRVNC are heart failure, arrhythmia, and embolic events.

Case Report: Herein we describe an isolated right ventricular non-compaction in a patient who had an cardiac by-pass surgery before. A 60-year old man patient was evaluated in out-patient policlinic.He had a cardiac by-pass surgery one year ago.He hasnt any symptom now. His ECG

was normal.Echocardiographic examination revealed right ventricular apical non-compaction with deep trabeculations (Figure 1, 2, Video 1, 2). 24 hours ECG rhythm holter showed non-specific atrial premature depolarizations. We decided to medical treatment for this patient.

Conclusion: IRVNC is a rare congenital heart disease and it has several serious consequencies but still current treatment choices are limited.

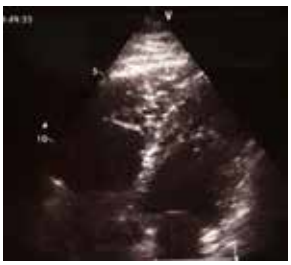

Figure 1. Right ventricular apical non-compaction.

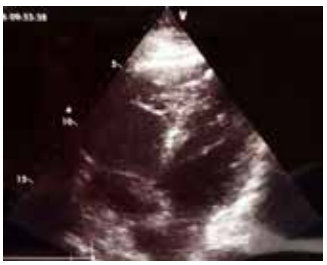

Figure 2. Right ventricular apical non-compaction.

OPS2-32

Myocardial bridging as a cause of myocardial ischemia in the patient with single coronary artery

Muhittin Demirel[1], Cüneyt Toprak[1], Lütfi Öcal[2], Özgür Yaşar Akbal[1], Alev Kılıçgedik[1]

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A 43-year-old female was admitted with effort angina Past medical history and physical examination was unremarkable. The electrocardiography (ECG) and lab results were normal. Transthoracic echocardiogram was normal. The exercise ECG test revealed evidence of ischemia. The patient was scheduled for coronary angiography on the following day. The coronary angiogram showed a patent left main coronary artery and a long and large-sized normal left circumflex (LCx) coronary arteries reached out to the right coronary artery (RCA) territory in the absence of RCA ostium. Cardiac computed tomographic angiography (CCTA), which was subsequently performed, revealed the presence of single coronary artery couring along the anterior side of the aortopulmonary trunk with myocardial bridging of LAD. In addition, the absence of RCA ostium were confirmed. She was discharged uneventfully with 100 mg aspirin and 100 mg metoprolol. She remained well at 6 months follow-up. Single coronary artery is rare, but potentially fatal anomaly. It can present with a broad range of clinical signs and symptoms. However, it may not be the underlying cause of the symptoms in certain circumstances. The myocardial bridge may cause ischemia and presyncope as in our case. Accordingly, thorough evaluation of symptomatic patients, including the course of anomalous coronary artery with CCTA as well as the assessment of ischemia, and structural heart diseases is mandatory.

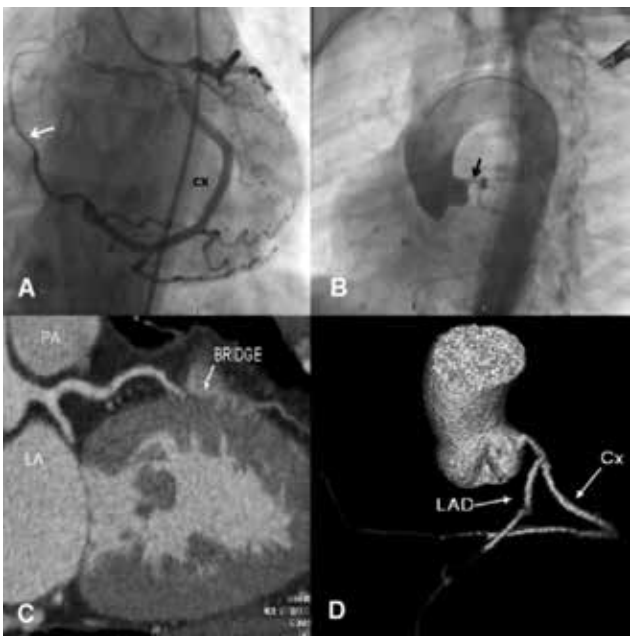

Figure 1. Conventional coronary angiogram showing a long and large-sized LCX coronary artery extending to the RCA territory (white arrow) **(A)**. The aortic root angiogram demonstrating a patent single coronary artery (black arrow) **(B)**. CCTA showing the single coronary artery coursing anterior side of the aortopulmonary with myocardial bridging of LAD **(C)**. The exact anatomical course of the anomalous coronary artery branches **(D)**. PA: Pulmonary Artery LA: Left atrium.

Konjenital kalp hastalıkları

OPS2-33

Functional single ventricle in a young patient: A rare congenital anomaly of the heart

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Single or common ventricle composes of 1.5% of all congenital heart diseases. Main symptoms are dyspnea and cyanosis. 50% of the patients die in te first month of their lives and 74% in the first 6 months. Pulmonary stenosis and great artery transpositions accompany with this anomaly frequently. A 32-year-old female patient admitted to our emergency clinic for dyspnea and palpitation in NYHA class II. 3/6 holosystolic murmur was audible in the mesocardiac area. Pulse rate was 80/min, arterial blood presure 120/80 mmHg and pO2 saturation 88%. There was no pretibial edema. She had history of single ventricle first diagnosed when she was 15 years old, receiving angiotensin receptor blocker and betablocker therapy. Chest radiogram showed mild cardiomegaly and increased pulmonary vascular bed (Fig. 1a). Diffuse ST segment depression, negative T waves in chest leads and right bundle branch block with a normal sinus rhythm were seen in ECG (Fig. 1b). Echocardiography revealed that she double-outlet single highly trabeculated ventricle in levoposition (Type AIII) (Fig. 1c, d). In absence of the interventricular septum completely, ejection fraction was 55% estimated by modified Sympson method. Moderate mitral and tricuspid valvular regurgitations existed with 4 m/s of tricuspid jet velocity. Inferior and superior vena cavae and portal vein were dilated. Functional single ventricle is a general term used to describe heterogeneous group of patients. The right or left ventricle is undeveloped or absent; leading to only one functional pumping chamber. Morphological, clinical and hemodynamic characteristics of the heart are variable and indicate the survival of the patient. These patients need repeated surgery, frequent hospitalization and have higher morbidity and mortality. Close hemodynamic monitorization is required to decide surgical correction.

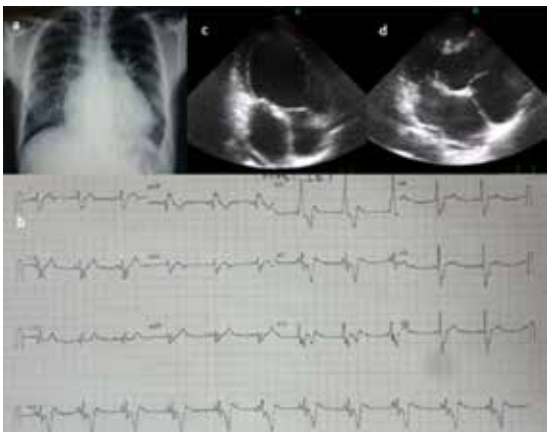

Figure 1.

OPS2-34

Sol ventriküle açılan çoklu koroner fistüller

Abdullah Özçelik[1], Sümeyye Fatma Özer[1], Kurtuluş Özdemir[2]

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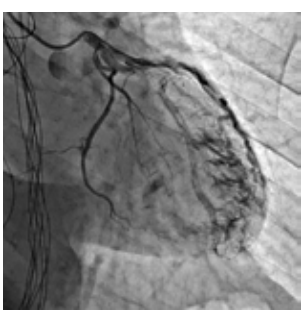
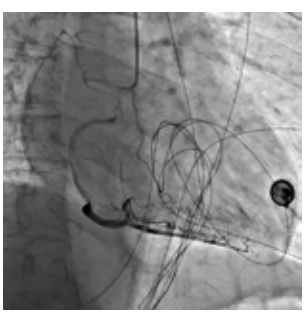
Koroner fistüller, koroner arterlerin düşük basınçlı vasküler boşluklar ya da kardiyak kaviteler ile bağlantıları olarak tanımlanır. Koroner fistüllerin çoğu, rastlantısal olarak koroner anjiyografi sırasında tanı alır. Hastamız, bilinen hipertansiyon öyküsü olan, 73 yaşında erkek hasta. Dış merkeze, baş dönmesi ve göğüs ağrısı ile başvurmuş. Dış merkezde yapılan koroner anjiyografide koroner fistül tespit edilmesi üzerine merkezimize yönlendirilmiş. Koroner anjiyografi incelendiğinde, LAD ve RCA'dan sol ventriküle multipl fistül tespit edildi. EKG'de iskemi bulgusu yoktu ve klinik takibinde göğüs ağrısı olmadı. Baş dönmesi nedeniyle 24 saatlik holter EKG monitörizasyonu yapıldı. Malign aritmi tespit edilmedi. koroner fistülün multipl olması ve izleminde şikayetlerinin tekrarlamaması üzerine medikal takip kararı alındı. Kameral fistüllerin %90'ı kalbin sağ tarafına açılır ve genellikle tektir. Sol kalp boşluklarına açılanlar ve multipl olanlar nadir görülür. Biz de bu vakayı, nadir görülmesi nedeniyle raporladık.



Şekil 1. LAD-LV fistül.



Şekil 2. RCA-LV fistül.

OPS2-35

İzole ventriküler divertikül

Mustafa Serkan Karakaş, Ali Yaşar Kılınç, Fatih Koç, Veysel Tosun

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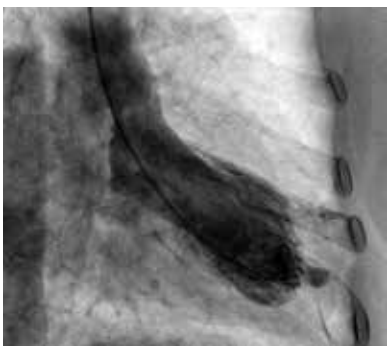
Giriş: Konjenital ventriküler divertikül (KVD) klinik pratikte nadir olarak görülür,daha çok sol ventriküldedir. KVD sistemik tromboemboli, endokardit, kardiyak rüptür, kalp yetersizliği, ventriküler aritmi ve ani kardiyak ölüm gibi komplikasyonlara yol açabilmektedir. Bu yazıda kliniğimizde rastlantısal olarak bulduğumuz sol vetrikül divertikülü olan bir hastayı sunduk.

Olgu Sunumu: Altmış yaşında kadın hasta 6 ay önce romatizmal mitral darlık nedeni ile perkütan mitral balon valvüloplasti yapılmış, 2 ay önce elektif anjiyografi sonucunda sağ koroner artere (RCA) stent implante edilmiş. Özgeçmişinde ayrıca diyabet, hipertansiyon, kronik obstrüktif akciğer hastalığı bulunmaktaydı. Hasta atorvastatin, perindopril, asetilsalisilik asit, klopidogrel, varfarin, furosemid, metoprolol ve bronkodilatatör ilaçlar kullanmaktaydı.Hastanın son 1 aydır olan efor anginası ve efor dispnesi mevcuttu. Fizik muayenesinde; S1 sertti ve kalp sesleri aritmikti.Diğer sistem muayeneleri doğaldı. EKG'de ritim atriyal fibrilasyon ve kalp hızı 110/dk idi. Hastanın ekokardiyografisinde EF %65, mitral kapak alanı planimetrik 2 cm^2, mitral kapak peak gradient 15, mean gradient 7 mmHg, olarak ölçülmüştür. 3-4. derece mitral, 1. derece aort ve 1-2. derece trikuspid yetersizliği mevcuttu. Triküspid yetmezlik velositesi üzerinden hesaplanan sistolik pulmoner arter basıncı 63 mmHg idi. Hastaya koroner anjiyografi ve kateterizasyon yapıldı. RCA'daki stenti patent saptanan hastanın yapılan sol ventrikülografisinde (SVG) sol ventrikül apeksinde 11x8 mm boyutunda divertikül olduğu görüldü (Şekil 1). Kapak cerrahisi planlanan hastanın mevcut şikayetleri ön planda kapak patolojisine bağlandı ve hastaya mitral kapak cerrahisi kararı verildi. Operasyon mortalitesini arttıracağı için divertikül eksizyonu planlanmadı.

Tartışma: KVD'lerin klinik olarak büyük bir kısmı asemptomatik olduğundan tanı koymak güçtür. KVD iki tipte olabilmektedir: fibröz ve musküler. Fibröz tipler kontraktil değildir, sol ventrikül bazaline ve genellikle submitral alana yerleşir. Sıklıkla kapak yetersizliğiyle birliktelik gösterir. Fibröz divertiküllere konjenital defektler eşlik etmez. Sol ventrikül divertiküllerinin %70'i musküler divertiküldür ve SVG'de apekse lokalizedir. Duvarında her üç kardiyak tabaka da yer alır. Musküler divertiküller; orta hat torakoabdominal anomaliler ya da konjenital kardiyak malformasyonlarla birlikte olduğundan tanı genellikle çocukluk çağında konulur. Kardiyak divertiküllerin etiyolojisi tam bilinmemektedir. İki boyutlu ekokardiyografi ile büyük çaplı divertiküllerin tanısı konulabilir fakat küçük çaplı divertiküllerin ekokardiyografiyle tanısını koymak zordur. KVD'lerin tanısında en faydalı yöntem SVG'dir. KVD olan semptomatik hastalarda ve ventrikül divertikülünün ek kardiyak anomalilerle beraber olduğu olgularda cerrahi tedavi öncelikle düşünülür. Fakat asemptomatik KVD'nin operasyon gerektirip gerektirmediği tartışmalıdır.



Şekil 1. Sol ventrikülografi: sol ventrikül apikalinde divertikule ait görünüm.

OPS2-36

A unique association of Wegener's disease with patent ductus arteriosus

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Patent ductus arteriosus is a congenital heart disease that connects the proximal descending aorta to the roof of the main pulmonary artery near the origin of the left branch pulmonary artery. Wegener's disease is a systemic inflammatory disorder of unknown aetiology, cardiac involvement of this disease includes pericarditis, arteritis, myocarditis, valvulitis and arrhythmias. We want to present a case of a first time of concomitance of PDA with Wegener's disease.

Introduction: Patent ductus Arteriosus (PDA) is a vascular structure that connects the proximal descending aorta to the roof of the main pulmonary artery near the origin of the left branch pulmonary artery. The incidence of PDA is 5-10% of all congenital heart disease. Ductus closure is clearly indicated in symptomatic patients with significant left to right shunting. Percutaneous closure of PDA remains standart practise since 1960s.Wegener's granulomatosis is a systemic inflammatory disorder of unknown aetiology. Cardiac involvement of this disease includes pericarditis, arteritis, myocarditis, valvulitis and arrhythmias. Up to now there is no published case report with Wegener's disease and PDA in the literature. We want to present a case of a first time of concomitance of PDA with Wegener's disease.

Case Report: A 42-year old female was admitted to emergency department with dyspne and hemoptysis. Pyhsical examination revealed continuous murmur at upper left sternal border. Chest roentgenography showed bilaterally heterogeneous density in basal segments. Thoracic computed tomography revealed multiple granulomatous nodules in bilaterally lung segments (Pic A). PDA was revealed in basal short axis view of transthoracic echocardiographic examination (Pic B). Anti-nuclear antibody (ANA) and Cytoplasmic-Antineutrophil Cytoplasmic Autoantibody (C-ANCA) were positive in her laboratory examination so Wegener's disease was found to be diagnosis in rheumatology consultation. After the diagnosis of PDA, the occlusion of PDA was considered and PDA was occluded successfully with percutaneous closure way with using occlusion coil (Pic C-D and E). To our knowledge, this is the first reported case of concomitance of PDA with Wegener's disease. Percutaneous closure of PDA with coil occluders remains standart practise since 1960s. Wegener's granulomatosis is a systemic inflammatory disorder of unknown aetiology. It defines a systemic small-vessels vasculitis, characterized by frequent involvement of upper and lower respiratory tract. The Cytoplasmic-type ANCA (C-ANCA) with Anti-Proteinase 3 specificity is observed in more than 90% of patients with Wegener's disease. Cardiac involvement of Wegener's disease include pericarditis, arteritis, myocarditis, valvulitis and arrhythmias. Up to now there is no published case report with Wegener's disease and PDA in the literature. To our knowledge, this is the first reported case of PDA with Wegener's disease.

OPS2-37

All three coronary arteries multiple fistulae draining to pulmonary artery and concomitant coronary anomaly

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Coronary to pulmonary fistulae are uncommon cardiac anomalies. 0.05–0.25% of patients undergoing diagnostic coronary angiography shows a fistula. Coronary artery fistulas are usually asymptomatic but larger and multiple fistula can lead to ischemia congestive heart failure, myocardial infarction and pulmonary hypertension. Although the usually congenital, it may be acquired forms. Frequently arises from the right coronary artery system (55%), less frequently left coronary artery system (35%), infrequently both coronary arteries (5%). The most common locations where drains, in decreasing frequency, are the right ventricle, right atrium, pulmonary artery, coronary sinus, left atrium, left ventricle, and superior vena cava. Although the fistulas are single, multiple fistulas was reported. Despite the fact that the most common type of coronary artery fistula in a study was coronary to pulmonary artery, whereas all three coronary artery to pulmonary artery multiple fistula and concomitant anomalous origin of coronary was less common related in fistula formation. We aimed to present this rare condition, a symptomatic patient with a congenital multiple coronary arteriovenous fistula, originating from the left anteriyor desending artery (LAD), left circumflex artery (LCx) and right coronary artery (RCA) draining into the pulmonary artery and concomitant anomalous origin of coronary which LCx arising from right coronary system. A 65 year old man admitted to the exertional dyspnea and typical anjina chest pain. Clinical examination revealed a grade 2 continuous murmur in the mesocardiac area. His medical history consisted of hypertension an dyslipidemia as the risk factor for coronary artery disease. Standard 12-lead electrocardiogram was no significant property. Transthoracic echocardiography examination demonstrated left ventricular relaxation impairment, left ventricular ejection fraction was 65%, significant valvular pathology was not observed and systolic pulmonary artery pressure was 45 mmHg. Subsequently, coronary angiography showed that normal epicardial coronary arteries whereas originating all three coronary arteries multiple fistulaes draining to pulmonary artery (Fig 1a, b) and concomitant coronary anomaly which LCx originating from the right coronary system (Fig. 2a). Furthermore, RCA is the origin of fistula in two different locations which mid level and conus branch (Fig 2b). Coronary artery fistula and ectopic origin of the LCx from the RCA are two different rare congenital anomalies. The cases with co-existed the two anomalies are more rare. We aimed to present this rare condition. To the best of our knowledge with literature review, the coronary artery fistula all three coronary artery to pulmonary artery between ectopic origin of the LCx from the RCA has not been previously reported.

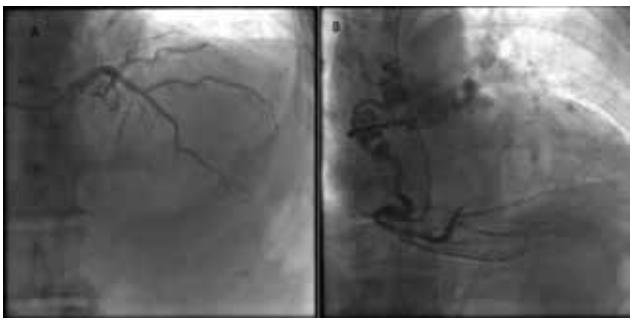

Figure 1. (A) Left coronary angiography demonstrated an anomalous coronary fistulae from the left anterior descending coronary artery to the pulmonary artery. **(B)** Right coronary angiography demonstrated an anomalous coronary fistulae from the RCA to the pulmonary artery.

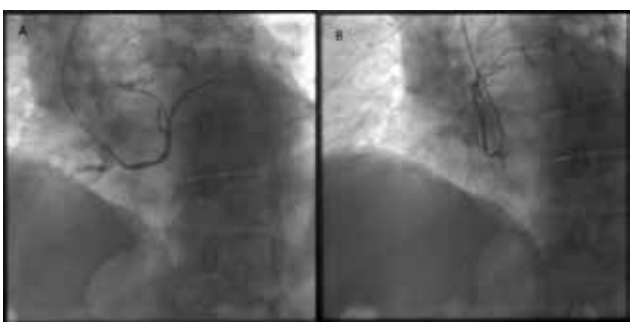

Figure 2. (A) Left coronary angiography demonstrated an anomalous coronary fistulae from the left anterior descending coronary artery to the pulmonary artery. **(B)** Right coronary angiography demonstrated an anomalous coronary fistulae from the RCA to the pulmonary artery.

OPS2-38

An aberrant ostium and subaortic course of left circumflex artery

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Department of Cardiology, Gülhane Military Medicine Academy, Ankara

Introduction: Coronary artery anomalies (CAA) are defined as congenital changes in their origin, course and structure and reported to be about 1% in the angiographic series. The most common coronary variation is the anomalous left circumflex artery (LCx), found in approximately 0.7% of all patients. LCx may be originated in a different positions; from a seperate ostium within the right sinus, from a main trunk with the right coronary artery or as a proximal branch of RCA. Such an anomaly may be associated with life-threatening conditions including arrhythmias, myocardial infarction, congestive heart failure, syncope and sudden cardiac death.

Case Report: A 36-year old male patient admitted to our outpatient clinic for chest discomfort similar to sore throat. He had no previous history of any cardiac symptom. His medical and family history was unremarkable. Cardiovascular exam revealed no any findings. Heart and lung ausculta-tions were normal. Systemic blood presure was 120/70 mmHg and pulse rate was 70 beat/minute. Biochemical results such as complete blood count, sedimentation rate and other electrolytes tests were normal. The 12-lead electrocardiography revealed normal sinus rhythm with a rate of 70 beat per minute. Transthoracic echocardiography (TTE) study showed normal left ventricle ejection fraction (65%) with mild tricuspid valve insufficiency. computed tomography angiography (CTA) scan performed for the patient due to doubtfully pozitive resulted exercise stress test. CTA revealed the right (RCA) and the left circumflex (LCX) coronary arteries originated from common orifice from right coronary sinus. The LCX artery proceeded posterioinferiorly and reach to left atrio-ventricular sulcus (Figure 1). The left anterior desending (LAD) coronary artery orifice originated from left coronary sinus in seperately. The CT angiogram for diagnosis of coronary artery anomaly is with high successful rate.

Conclusion: Coronary artery anomalies may cause symptoms similar to coronary artery diseases even if absent of atherosclerotic process. However these anomalies may also be a vulnerable envi-ronment for progress of the coronary artherosclerosis. Computed tomography angiography is one of the main diagnostic tool that can exhibit the coronary anomalies in detail.

Figure 1. CTA scaning images showing RCA and LCX arteries originated from common orifice from right coronary sinus. The LCX artery cruising to posterioinferiorly and reach to left atrioventricular sulcus.

OPS2-39

Cerrahi olarak atrial septal defekt kapatılmasından iki yıl sonra görülen sağ atriyum içerisinde iki adet hareketli trombüs

Cengiz Öztürk, Zekeriya Arslan, Hamidullah Haqmal, Mustafa Demir, Ali Osman Yildirim, Sevket Balta, Turgay Celik, Atila İyisoy

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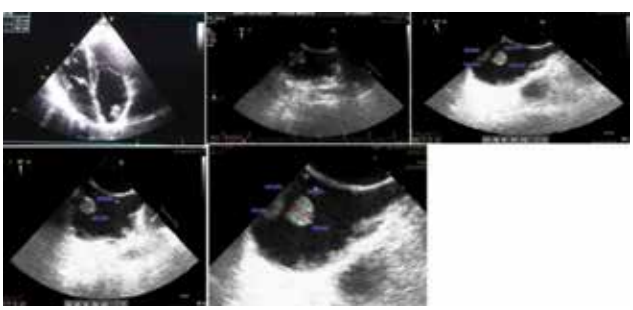
Yirmi üç yaşında erkek hasta iki yıl önce çarpıntı, baş ağrısı ve baş dönmesi yakınmaları ile dış merkeze başvurmuş. Transtorasik ekokardiyografide (TTE) sağ ventrikül çaplarında hafif dere-ce artış ve hafif-orta derecede triküspit yetmezliği saptanmış. İntraatriyal septum (İAS) üzerinde geniş ostium sekundum tipi atrial septal defekt (ASD) (defekt çapı 30 mm) saptanmış ve yapılan transözefajial ekokardiyografide (TEE) posterior rim yetersizliğinden dolayı perkütan kapama için uygun görülmemiş ve hastaya kalp damar cerrahi kliniği tarafından perikardial yama kullanılarak kapama işlemi gerçekleştirilmiş. Hasta kliniğimize rutin kontrol muayenesi amacıyla başvurdu. Fizik muayenesinde TA: 110/60 mmHg, Nb: 102/dk. TTE incelemesinde LVEF %62, sol ve sağ ventrikül çapları normal, sağ atriyum posterior ve lateral duvar tarafında iki adet hareketli kitle lezyonu izlendi. Lezyonun morfolojisini daha iyi değerlendirmek için TEE uygulandı. Üst ve orta özefajial uzun eksen görüntüde, interatrial septum intakt, cerrahi sütür materyaline ait görüntüler ve çapları 14.5x13.5 mm ve 13.5x8.5 mm boyutlarında hareketli ekojen iki adet kitle lezyonu izlendi. Üç boyutlu transözefajial ekokakardiyografide de hareketli lezyon gözlendi. Tanı olarak trombüs düşünüldü. Vakamızda görüntü olarak milsoma da düşünülebilirdi ancak görüntü olarak hem trombüse benziyor hem de cerrahi işlemden 2 yıl sonra görülmesi ve septal bağlantısının olmaması bize miksoma yerine trombüsü düşündürdü. Hastaya kalp damar cerrahisi konsultasyonu alındı ve elbette kesin tanı biopsi ile konulacaktır. Sonuç olarak, ASD perkütan ve cerrahi kapama sonrası hastalara yakın takip yapılmasının gerekli olduğunu düşünüyoruz.



Şekil 1.

OPS2-40

Triple cardiac defect in a young patient; perimembraneous and muscular type ventricular septal defect and wide atrial septal defect

Hamidullah Haqmal, Cengiz Ozturk, Murat Unlu, Ali Osman Yildirim, Mustafa Demir, Zekeriya Arslan, Turgay Celik, Atila İyisoy

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Atrial septal defects (ASDs) and ventricular septal defects (VSDs) are the two most common cardiac malformations. We reported triple cardiac defect (perimembraneous and muscular type ventricular septal defect and wide atrial septal defect) in a young patient. A 21-year old male was admitted to our outpatient with the complaint of short term angina and dyspnea related exercise in last one mounth. His symptoms completely resolve with rest. No previous history of any cardiac symptom is present. His medical and family history were unremarkable. His six minute efor capacity (walk test) result was 650 meters. Complete blood count, ESR and other electrolytes tests are normal. On physi-cal examination, a 4/6 pansystolic murmur was heard. Systemic blood pressure was 120/80 mmHg and pulse rate was 103/min. The 12-lead electrocardiography revealed sinus rhythm with a rate of 105 beat per minute, T wave invertion in chest leads V1-4 with right bundle branch block (RBBB) morphology, left axis deviation and high amplitude R wave and LV hypertrophy. Transthoracic echocardiography (TTE) study showed normal LV ejection fraction (60%), concentric hypertrophy, shunt from left to right ventricle with aneurysmal dilatation with septal pouch leading to diagnosis of perimembranuos type ventricular septal defect near the aortic valve, left to right shunt in the in-terosepta muscular part of LV and presence of wide (3x3cm) atrial septal defect. Also mild tricuspid valve insufficiency with right side dilatation was seen. QP/QS was >2.7 and systolic pulmonary artery presure was 45 mmHg. Computed tomography scaning (CT) for further investigation was made because of the patient's intolerance of transesophageal echocardiography. The CT showed the presence of perimembranuos and muscular type VSD and wide ASD. Tomographic angiography of coronary arteries showed normal anatomy. Following consultation with cardiovascular surgeons with these finding, the patient was considered for surgical treatment. The association of two ven-tricular and one atrial septal defects is a rare condition in a patient. Carefully examination and multimodality approach are advised to these patients in order not to make underdiagnosis.

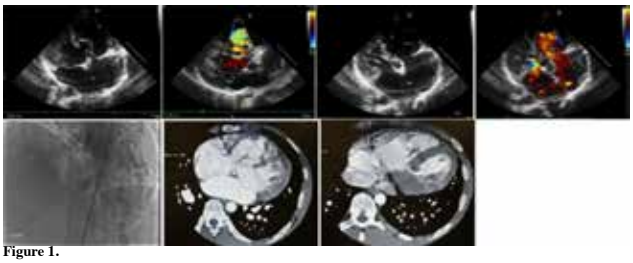

Figure 1.

OPS2-41

Quadricuspid pulmonary valve detected by CT angiography

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Introduction: Quadricuspid pulmonary valve is a rare anomaly with incidence of one in 400 to 2000 autopsies. It's been reported as a silent pathology which rarely produces significant valvular dysfunction and it has been diagnosed mainly post mortem. Due to its position, echocardiographic diagnosis is difficult because of not easy to identify the three cusps at the parasternal views. Recent advances in cardiac imaging, such as computed tomography (CT) and magnetic resonance imaging (MRI), made more cases being discovered incidentally in living patients.

Case Report: We present a case of an asymptomatic 23-year old male who was referred for moder-ate pulmonary regurgitation which diagnosed by transthoracic echocardiogram. He had no previ-ous history of any cardiac symptom and his medical and family history was unremarkable. 2/6 systolic murmur on the pulmonary and ¼ diastolic murmur on the left sternal area were heard at auscultation. The lung was normal. Cardiothoracic index was mildly increased at X-ray. Blood pressure was 127/76 mmHg and pulse rate was 64 bpm. The 12-lead electrocardiography revealed normal sinus rhythm with a rate of 60 beat per minute. Transthoracic echocardiography (TTE) showed normal left ventricle ejection fraction (65%), mild tricuspid and moderate pulmonary valve regurgitation. There was a 20 mmHg peak systolic gradient on the pulmonary valve with minimal right ventricular dilatation. QP/QS was 1,4/1. (Fig. 1). Computed tomography angiography also re-vealed that pulmonary root was dilated (35x30 mm) and pulmonary valve consists of four leaflets. The coronary artery anatomy was normal. Echocardiographic follow-up was recommended for the patient in terms of valvular insufficiency.

Conclusion: Quadricuspid pulmonary valve is a rare congenital anomaly. Diagnosis of quadricus-pid pulmonary valve by TTE is very difficult, because of the anatomical disposition of the valve with respect to the thoracic wall. Hence it has almost always been diagnosed at autopsy. Thanks to development of the imaging technologies, this type of anomalies could have been caught before early stages of the life.

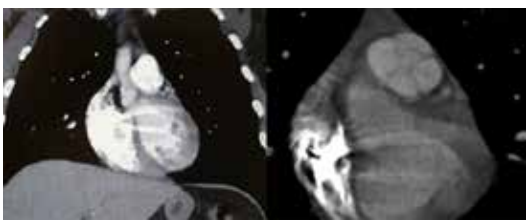

Figure 1. The CTA scanning image shows dilated pulmo-nary root and quadricuspid pulmonic valve.

OPS3-01

Şiddetli ve tekrarlayıcı öksürük atağı sonrası gelişen koroner arter disseksiyonu

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A 42-year-old man with a history of smoking was referred to our emergency department with complaints of severe chest pain after severe coughing episodes. Cardiac auscultation revealed 2/6 grade systolic ejection murmur over the tricuspid area. Electrocardiogramshowed a sinus rhythm with inferior ST-segment elevation and reciprocal ST-segment depression in the lateral leads. Acute inferior ST segment elevation myocardial infarction (STEMI) was the suggested diagnosis and we started a 300 mg of aspirin and 600 mg of clopidogrel, and 5000 U of heparin. Coronary angiography (CAG) revealed normal left main artery, left anterior descending (LAD) artery, and left circumflex artery but CAG showed that the mid segment of right coronary artery (RCA) had dissection with thrombosis and preserved distal RCA flow (Figure 1). Successful percutaneous coronary intervention (PCI) to RCA with a everolimus-eluting stent was done (Figure 2). Spontaneous coronary artery dissection (SCAD) is a rare cause of acute myocardial infarction (MI) and accounting for 0.1 to 4.0% of all acute coronary syndromes (ACSs). Patients are typically young, female, and lacking traditional coronary risk factors. STEMI occurs in 48% of cases. The pathophysiology and etiology of SCAD is poorly understood and felt to be multifactorial in etiology (atherosclerosis, the peripartum period, multiparity, menopause, oral contraceptive use, connective tissue disease, trauma, psychophysical stress, vascular malformations, cocaine use, vasculitis, fibromuscular dysplasia, polycystic kidney disease, and certain medications). Revascularization is typically considered in cases refractory to conservative management. Coronary stenting has been employed in several cases. Coronary artery bypass grafting is generally reserved for cases with dissection of the left main or multiple vessels. A wide variety of complications related to recurrent coughing have been reported. Cardiovascular complications of coughing include syncope, arrhythmias and rupture of subconjunctival and nasal veins. Additionally SCAD associated with coughing only one patient with cystic fibrosis have been reported previously in literature however in patients with not having chronic diseases have not been reported previously.

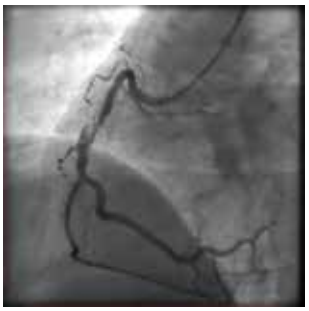

Figure 1. Right coronary angiogram showing right coronary artery dissection and thrombus of mid segment.

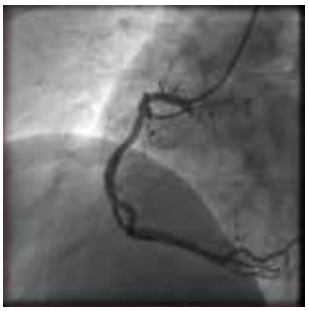

Figure 2. Right coronary angiogram. After implantation of a everolimus-eluting stent in the mid right coronary artery..

OPS3-02

Acute inferior myocardial infarction related to blunt chest trauma

Celal Kilit[1], Taner Şen[2], Adnan Doğan[1], Muhammed Oylumlu[1], Mehmet Özgeyik[1], Hüseyin Burhan Sarısoy[1], Türkan Paşalı Kilit[3]

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Introduction: Despite the frequent occurrence of blunt chest trauma (BCT), associated cardiac injuries are relatively rare. A wide variety of cardiovascular pathologies have been associated with BCT, including myocardial contusion, aortic transection or less often aortic dissection, trauma-induced ventricular arrhythmia or commotio cordis in young children, hemopericardium with tamponade, and aortic valve leaflet avulsion. Acute myocardial infarction (AMI) is a rare but well-described complication of BCT. Since the advent of primary percutaneous intervention, there have been several reports of AMI due to total epicardial thrombotic occlusion following BCT and its successful treatment by percutaneous intervention. We report a case of acute inferior wall myocardial infarction following BCT.

Case Report: A 46-year-old male with no relevant medical problems was transferred to our medical center for retrosternal chest pain after BCT due to a traffic accident. Upon arrival to the emergency department patient was still complaining of chest pain. ECG demonstrated ST segment elevation in the inferior leads with reciprocal changes in the lateral leads all consistent with active ischemia (Figure 1). After rolling out rib or sternum fracture, haemorrhage and aortic dissection, patient was loaded with ASA, ticagerlor, heparin and was emergently taken to the cardiac catheterization lab. Coronary angiography demonstrated total thrombotic occlusion in the proximal right coronary artery with TIMI 0 flow distally (Figure 2). After balloon predilatation of the occlusion, dense thrombus formation was observed in coronary artery (Figure 3). After thrombus aspiration (Figure 4), an atherosclerotic plaque was noted on the angiogram that was successfully stented.

Discussion: BCT is among the most commonly seen problems in traumatology. In addition to the ubiquitous musculoskeletal thoracic injuries, a number of less common injuries that clinicians must be aware of, have been reported. Among those are blunt cardiac injuries which can present as a spectrum that spans from cardiac "concussion" and contusion to acute posttraumatic valvular and coronary artery changes lead to AMI. Proposed mechanisms for AMI in this setting have included intimal injury due to shear forces imparted by the blunt trauma, plaque rupture, coronary artery dissection, and coronary vasospasm. Patients with blunt trauma have greater numbers of circulating procoagulant microparticle and increased in vitro thrombin generation that correlated with injury severity. The treatment approach to AMI following BCT is immediate coronary angiography and revascularization with percutaneous intervention and continuation of dual antiplatelet therapy. Although significant chest pain would not be unexpected following severe blunt trauma injury to the chest, the possibility that ongoing pain represents myocardial ischemia should be considered, and a screening ECG should be considered to identify rare cases of AMI.

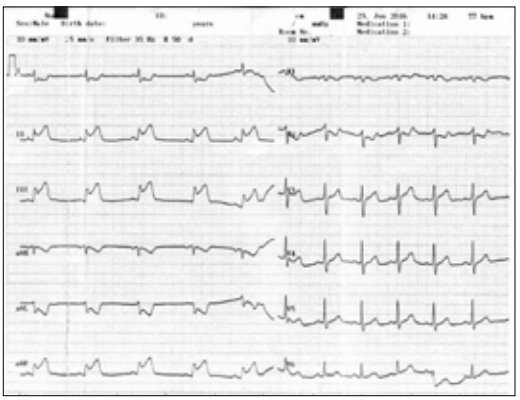

Figure 1. ECG demonstrated ST segment elevation in the inferior leads with reciprocal changes in the lateral leads.

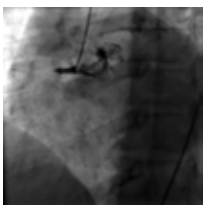

Figure 2. Coronary angiographic image demonstrated total thrombotic occlusion in the proximal right coronary artery with TIMI 0 flow distally.

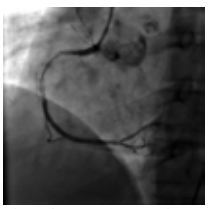

Figure 3. Dense thrombus formation observed in right coronary artery.

Figure 4. Retrieved thrombotic material from right coronary artery.

OPS3-03

Çift sağ koroner arter vakası

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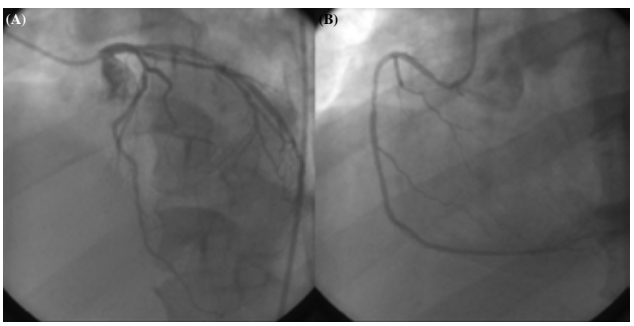
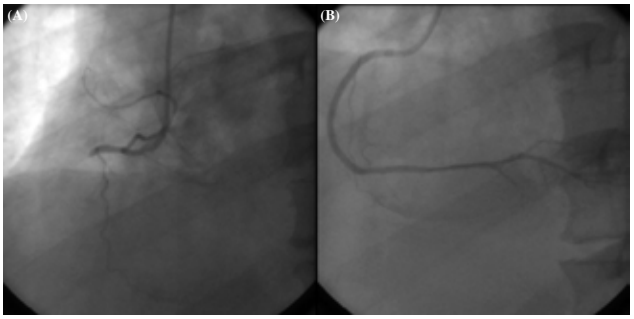
Koroner arterlerin sayısı, şekli ve ostiumlarının lokasyonları çeşitli şekillerde olabilir. Koroner arterlerin çıkış anomalisi nadir görülen anomalilerdir ve çoğunun klinik önemi yoktur. 56 yaşında erkek hasta kliniğimize yaklaşık 6 saat önce başlayan göğüs ağrısı şikayeti ile başvurdu. Çekilen EKG'de inferior derivasyonlarda ST segment elevasyonu, D1 ve aVL derivasyonlarında resiprokal ST segment çökmesi izlendi. Ekokardiyografide sol ventrikül ejeksiyon fraksiyonu %50, inferior duvar hafif hipokinetik izlendi. Hasta primer perkutan koroner girişim planlanarak koroner anjiografi laboratuvarına alındı. Sol koroner arterlerde nonkritik lezyonlar izlendi (Şekil 1a). Sağ koroner arterde (RCA) de krtitik darlık izlenmedi (Şekil 1b). Göğüs ağrısı şikayeti ve EKG de ST elevasyonu devam eden ve circumflex arteri dominant olmayan hastanın RCA'sının da ince olması üzerine başka bir RCA olabileceğini düşünerek RCA'nın tekrar değerlendirilmesine karar verdik. RCA nın tekrar görüntülenmesinde başka bir ostiumdan çıkan ikinci bir RCA'nın olduğunu görüntüledik (Şekil 2a). Tam tıkalı olan ikinci RCA'ya perkutan koroner girişim yapıldı (Şekil 2b). Çift RCA koroner anomalilerin nadir görülen bir çeşitidir. Bizim vakamızda akut inferior MI nedeni ile koroner anjiografi yapılan hastada çift ostiumlu RCA mevcut olup ilk görüntülenen RCA normal ikinci RCA nın ise %100 tıkalı olduğu tespit edildi. Akut koroner sendrom vakalarında koroner anjiografide ciddi darlık görülmediğinde devam eden göğüs ağrısı veya ST yüksekliği var ise koroner arterlerde çıkış anomalisi olabileceği akılda tutulup, ek pozlarla inceleme yapılması düşünülmelidir.



Şekil 1. (A) LAD ve circumflex arterlerde kritik darlık izlenmedi **(B)** RCA da kritik darlık izlenmedi.



Şekil 2. (A) Tam tıkalı olan ikinci RCA **(B)** Tam tıkalı RCA'nın başarılı perkutan koroner girişim sonrası görüntüsü.

OPS3-04

Kounis syndrome presenting with acute inferior wall myocardial infarction and cardiogenic shock secondary to intravenous ampicillin/sulbactam administration

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Kounis syndrome (allergic angina or myocardial infarction) is described as the concurrence of myocardial infarction with mast cells activation. KS is induced by allergic and anaphylactic reaction and considered as a rare cause of coronary artery spasm. Patients with KS commonly show a good course and discharge without a complication. We present a patient with KS who presented with acute inferior wall myocardial infarction and cardiogenic shock. A 44 year old male patient was admitted to our center with a complaint of severe chest pain lasting for 1 hour. He was administered with a treatment of 1 g intravenous ampicillin/sulbactam with a diagnosis of upper respiratory tract infection in a primary health center. He was a quite active before this illness and did not have any history of allergy or traditional risk factors for coronary artery disease. Ten minutes after the injection, he felt a severe and squeezing retrosternal chest pain. On physical examination, he was pale looking. He did not have pruritus or a rash. His blood pressure and heart rate were 77/48 mmHg and 104 bpm respectively. Electrocardiogram (ECG) showed ST segment elevation in leads D2, 3, aVF, V4R, V5R and V6R and ST segment depression in leads D1 and aVL (Figure 1 a, b). A bed side echocardiogram revealed a moderate reduction in left ventricular ejection fraction (45%), inferior and inferoseptal wall hypokinesia and a depression in right ventricular systolic function. The patient was allowed to catheterization laboratory with a diagnosis of acute myocardial ischemia. Coronary angiography (CAG) revealed diffuse constriction of left anterior descending artery (LAD) and left circumflex artery (Cx) and total occlusion of right coronary artery (RCA) (Figure 2a, 3a, 4a). All constrictions were considered as coronary artery spasm. Despite the deep hypotension, intracoronary nitroglycerin was administered to confirm the coronary artery stenosis. Intra-arterial blood pressure monitoring was revealed that his blood pressure picked up to 108/73 mmHg. By this time his chest pain was alleviated and control CAG showed complete normalization of coronary artery spasm (Figure 1b, 2b, 3b). The patient was allowed to coronary care unit and a combination treatment of intravenous 25 mg prednisolone, 50 mg ranitidine and 50 mg diphenhydramine were administered with the diagnosis of KS. He felt better and his chest pain was completely resolved over the next 15 minutes. Control ECG showed normalization of ST segment elevation (Figure 5). The patient was managed for KS and isosorbit-5-mononitrat and ciprofloxacin 400 mg once a day were administered. The patient's eosinophil count was in normal range (120/μL) and cardiac biomarkers and serum tryptase level were elevated as follows; troponin I: 3 ng/dL, creatine kinase-MB: 52 U/L and 146 ng/mL. The patient was observed closely over the next two days and he did not develop a further chest pain or complication.

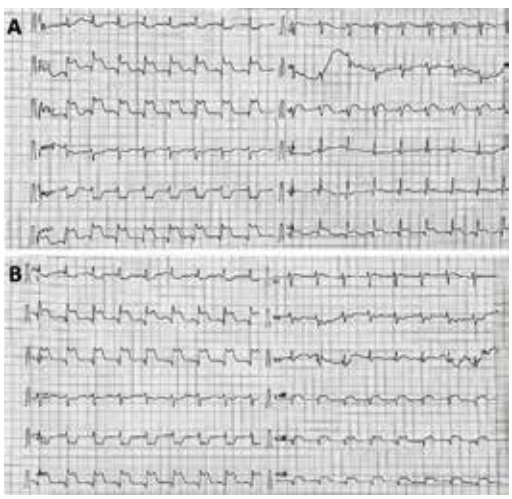

Figure 1. (A, B) Admission electrocardiograms show ST segment elevation in inferior leads and V4R, V5R, V6R.

Koroner arter hastalığı / Akut koroner sendrom

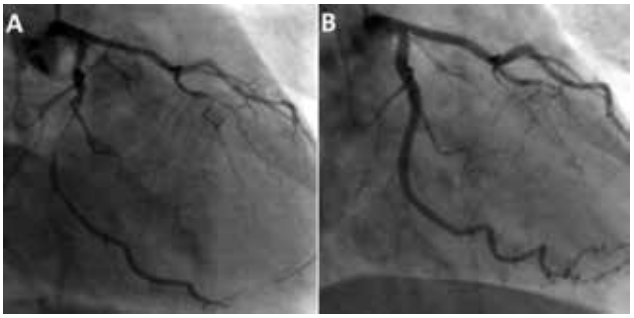

Figure 2. (A, B) Initial and control coronary angiograms show diffuse right coronary artery spasm and its normalization.

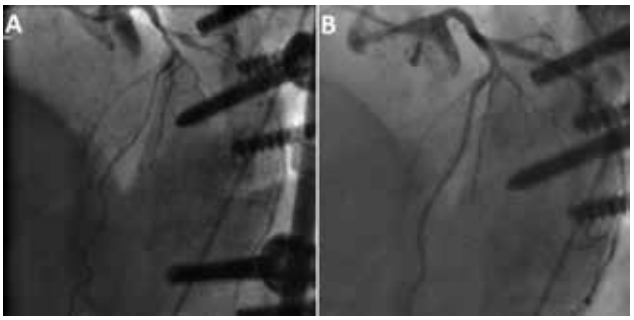

Figure 3. (A, B) Initial and control coronary angiograms show diffuse left anterior descending and left circumflex arteries spasm and its normalization.

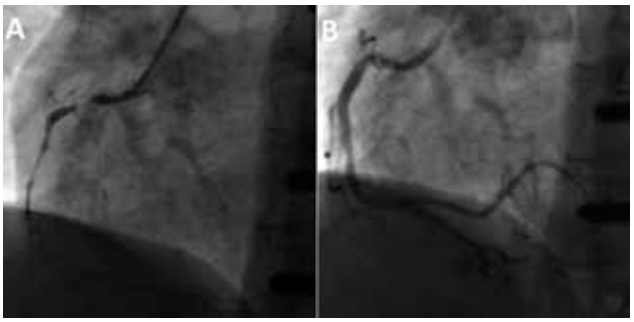

Figure 4. (A, B) Initial and control coronary angiograms show diffuse left anterior descending and left circumflex arteries spasm and its normalization.

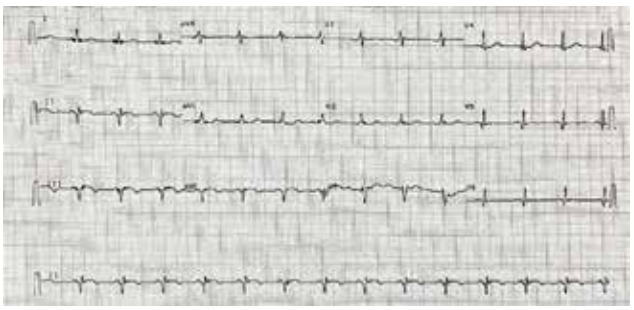

Figure 5. Control electrocardiogram shows normalization of ST segment elevation after antihistaminics and corticosteroid treatment.

OPS3-05

Kounis syndrome developing during postoperative residual curarization

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The Kounis Syndrome was first described in 1991 by Kounis and named the "allergic angina syndrome". Drugs and various conditions can lead to the Kounis Syndrome. A 21- year- old man admitted to orthopedic clinic because of his left ankle flexion contracture. Result of testing, it was decided to perform the operation. In patient history there was no allergic and other comorbid disease history. During present induction of anesthesia, anesthesiologist began general anesthesia with four different anesthetic drugs; 250 mg propofol, 100 mcq fentanyl, 40 mg lidocaine and 8

mg vecuronium. During the operation, they used nitrous oxide and sevoflurane for maintenance of anesthesia and the vital signs were normal. When the operation finished, anesthesiologist gave 1.5 mg neostigmine and 0.65 mg atropine for the purpose of postoperative residual curarization (PORC). Immediately after implementation of the drugs for PORC, it was denoted that the patient had instability, excessive sweating, palpitation and dyspnea. Besides ST depression occurred in ECG monitoring, the patient had tachycardia and hypertension in the same time. After that, his respiratory effort and verbal contact were good, he was successfully extubated; but the patient had typical chest pain in the retrosternal region. The ECG showed minimal ST-segment elevation in leads V1-V3 and ST-segment depression in leads V4-V6 (Fig. 1). According to the blood results taken in the postoperative care unit after surgery, CK-MB and troponin T were high, so the patient was transferred to the coronary care unit. In coronary care unit, physical examination revealed generalized erythema and mild urticarial rash as a possible clinical sign of allergic reaction. Cardiovascular and respiratory auscultation findings were unremarkable. Peak hs Troponin T (690 pg/mL) and CK-MB level (31.3IU/L) were high. Echocardiography showed mild septal wall hypokinesia and LVEF 55%. We gave immediately 5 mg sublingual nitrate, first generation antihistamine pheniramine maleate (45.5 mg) and 40 mg prednisolone by intravenous route. Then we started anti-ischemic treatment with 6000 IU enoxaparin, 100 mg acetylsalicylic acid, 300 mg clopidogrel and 20 mg atorvastatin therapy. During the follow up in coronary unit the patient's chest pain was relieved and ST segment depression was resolved in leads of V4-V6. However, we observed T wave inversion in leads of V1-V2 and biphasic T wave in leads V3 on ECG (Fig. 2). CAG performed 24 hours later revealed normal coronary anatomy (Fig. 3a, b). No recurrent angina was observed during the coronary care unit, the regional wall motion abnormality was greatly improved and he was transferred to the orthopedic clinic again without any symptoms. We suggested that the hypersensitivity reaction following PORC probably can result in the anginal attack and ECG changes and thereby, the diagnosis of Kounis I syndrome might be possible. So the patient was recommended to undergo allergy testing after discharge.

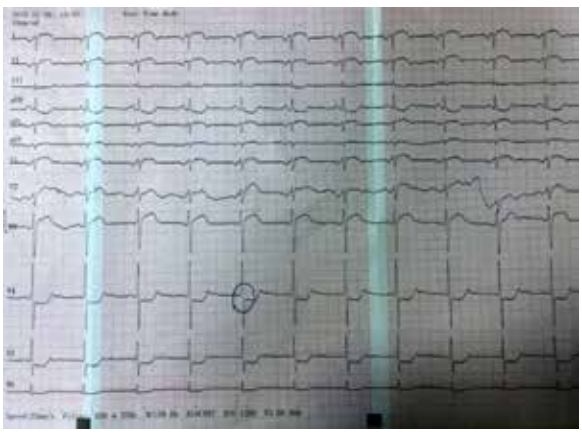

Figure 1. Take ECG immediately after the postoperative residual curarization and showed that horizontal ST-segment depression in leads V4-V6.

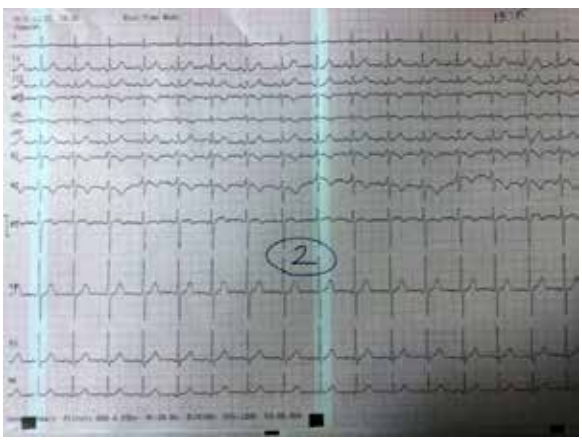

Figure 2. ECG after the patient's chest pain was relieved in coronary care unit and showed in leads of V1-V2 T wave inversion and biphasic T wave at V3.

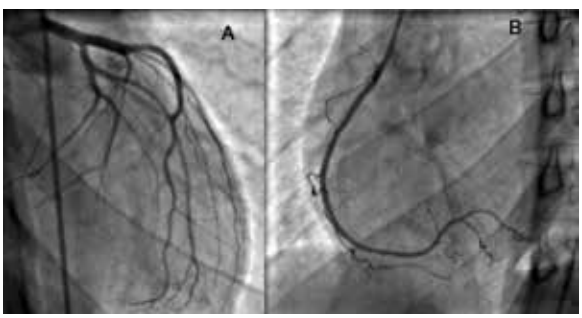

Figure 3. Coronary angiography revealed normal left **(A)** and right **(B)** coronary arteries.

Koroner arter hastalığı / Akut koroner sendrom

OPS3-06

Two life-threatening clinical conditions: Coexistence of myocardial infarction and acute ischemic stroke

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Introduction: Acute ischemic stroke in patients with acute coronary syndrome is uncommon but devastating. it is the single strongest risk factor for in hospital mortality (1,2). There is little information on the type of optimal reperfusion therapy and the antithrombotic therapy to be applied to patients with ST segment elevation myocardial infarction (STEMI) and acute ischemic stroke within a time. We present a case of tissue plasminogen activator thrombolytic therapy in a 69 year-old man admitted to the Emergency Department with STEMI and ischemic stroke.

Case Report: A 69-year-old man was admitted to the emergency department with 30 minute history of chest discomfort, right-sided hemiplegia and aphasia. He had a history of smoking, hypertension and 5 years before cerebral infarction. The physical examination showed a heart rate of 75 beats per minute, blood pressure of 110/70 mmHg. Neurological examination revealed right sided hemiplegia with a grade 0 motor power in the right upper and lower limbs and aphasia. The 12-lead electrocardiogram showed anterior ventricular ST-segment elevation (Figure 1a). A computed tomography (CT) excluded intracranial hemorrhage (Figure 2). The patient was diagnosed as STEMI and acute ischemic stroke. Because the two diseases occurred simultaneously within a time, the patient was first considered for thrombolytic therapy. We started intravenous tPA treatment with the recommended dose for acute STEMI (15 mg I.V. bolus, followed by 50 mg infused over the next 30 min, and then 35 mg infused over the next 60 min.). ST-segment resolution was approximately 40% at 90 minutes after starting of tPA (Figure 1b). 2 hours after thrombolysis, the strength of right upper and lower limbs improved. 24 hours later, the motor deficits almost disappeared. The brain CT at 12 and at 72 hours, presented no hemorrhage. Echocardiography showed global severe hypokinesia of the left ventricle with ejection fraction (EF) of 20% and no intracardiac thrombi. İn the third day, angiography was performed via to right radial access. Coronary angiography showed critical stenosis in left anterior descending artery (LAD) (Figure 3a). LAD was implanted with a drug eluting stent (Figure 3b). MRI showed the acute ischemic stroke image in different sequence after the 48 hours (Figure 4). Control echocardiography showed partially improved left ventricular systolic function with EF of 40%. The patient was discharged with functional capacity of New York Heart Association II and almost fully recovered from hemiplegia but partially recovered from aphasia.

Discussion: The use of intravenous tPA for patients with acute ischemic stroke 3 to 4.5 hours after onset showed a significant treatment benefit; however, there is little information on the type of optimal reperfusion therapy for the specific scenario of STEMI with acute ischemic stroke within a time. So that the systemic fibrinolytic therapy may be the optimal reperfusion therapy for these patients.

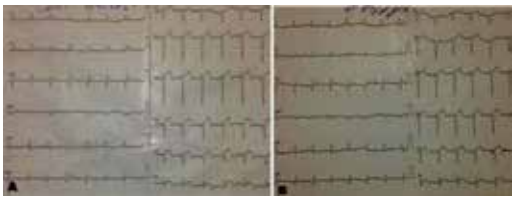

Figure 1. (A) ECG on admission to hospital shows ST-segment elevation in the anterior leads. **(B)** ECG after intravenous thrombolysis shows ST-segment resolution.

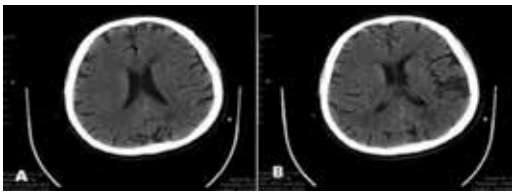

Figure 2. (A, B) Brain CT shows no hemorrhage CT: Computed Tomography.

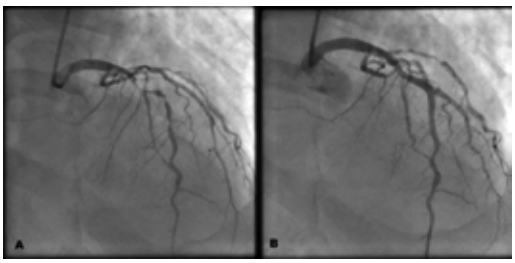

Figure 3. (A) Coronary angiography shows severe stenosis in the LAD. **(B)** After stent implantation LAD LAD: left desending artery.

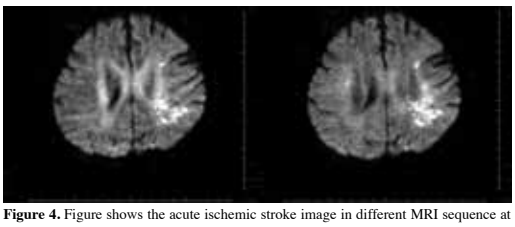

Figure 4. Figure shows the acute ischemic stroke image in different MRI sequence at 48 hours after onset.

OPS3-07

The association between lymphocyte-to-monocyte ratio and coronary artery disease severity in patients with stable coronary artery disease

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Aim: Inflammation plays an important role in the pathogenesis of atherosclerosis. Lymphocyte-to-monocyte ratio (LMR) may reflect systemic inflammatory status. We investigated the association between LMR and coronary artery disease in patients with stable angina pectoris.

Methods: A total of 221 consecutive patients who were routinely referred to coronary angiography for stable angina pectoris and 72 patients with normal coronary artery were included in the present study. We analyzed the relation between LMR and angiographic severity of CAD. The SYNTAX score was used for assessing the severity of coronary atherosclerosis.

Results: The neutrophil-to-lymphocyte ratio (N/L ratio), platelet size distribution width (PDW), neutrophil and uric acid levels were significantly higher in the coronary artery disease group than in the control group. The LMR was significantly lower in the coronary artery disease group than in the control group (4.5±3.2 vs. 6±2.9, p<0.001). Patients with elevated SYNTAX score (≥32) had lower LMR values (3.2±1.5 vs 4.6±3, p=0.002). Using multivariate logistic regression analysis, we found that the LMR was independent predictor of the presence of coronary artery disease in patients with stable angina pectoris.

Conclusion: Lymphocyte-to-monocyte ratio, an inexpensive and easily measurable laboratory variable, is significantly associated with the presence of coronary artery disease and high SYNTAX score in patients with stable angina pectoris.

OPS3-08

Wandering myocardial infarction

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A 54 year old man presented to emergency room with typical angina. The electrocardiogram revealed an ST elevation in inferior leads and complete AV block (Figure 1). On the way of catheterization laboratory the patient developed ventricular fibrillation which was successfully defibrillated. Angiogram didn't show any obstructive atherosclerotic disease. The patient was thought to have vasospastic disease but a provocative test wasn't performed. The patient was monitorised in the coronary care unit and prescribed diltiazem and isosorbid mononitrate. At 5 a.m. the patient suffered a typical angina again with a hyperacute ST elevation on anterior chest leads (Figure 2). Bedside echocardiogram revealed a severe hypokinesis of anterolateral walls. The patient immediately transferred to catheterization lab and left angiogram revealed total occlusion of left anterior descending and circumflex arteries (Figure 3a). At a dose of 100 microgram intracoronary nitroglycerin was performed resulting in complete patency of the left coronary system (Figure 3b). As the patient was demonstrated to have severe coronary spasm he was prescribed nitroglycerin patch, nifedipine 60 mg and sertraline. An implantable cardioverter defibrilator was implanted due to history of sudden cardiac death. His 3-month follow-up was free of arrhythmia and angina.

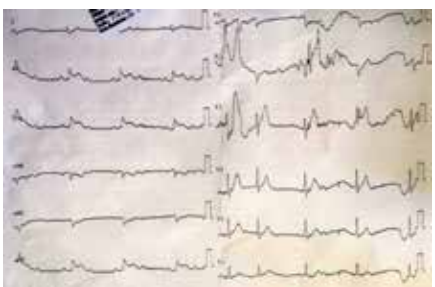

Figure 1. EKG.

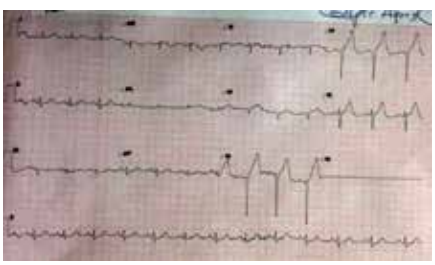

Figure 2. EKG-2.

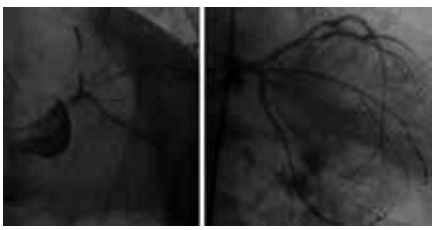

Figure 3. Anjio.

OPS3-09

Acute myocardial infarction after a blunt chest trauma in a patient without cardiovascular risk factors

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[2]*Department of Cardiology, Dumlupınar University Kütahya Evliya Çelebi Training and Research Hospital, Kütahya*

Introduction: Large vessels dissection and laceration after chest trauma is a common entity, however coronary artery injury is a rare complication. It may be misdiagnosed as musculoskeletal injury in emergency department especially in patients without cardiovascular risk factors. We report a case of acute myocardial infarction (AMI) due to the occlusion of left anterior descending artery (LAD) in a patient without cardiovascular risk factors who was referred to emergency department after a blunt chest trauma with chest pain and misdiagnosed as musculoskeletal injury.

Case Report: A 41-year-old male patient was referred to the emergency department after a car accident. He had moderate chest pain as pressure sensation and fullness. His pulse rate was 92 beats/min and blood pressure was 128/79 mmHg. The physical examination showed minimal bruises on the chest wall. There was no pathology in thorax computed tomography. However, 12-lead ECG was not performed in emergency department. Patient was considered as musculoskeletal trauma and discharged from emergency department. After discharge, chest discomfort was continued for 6 hours and the patient was admitted to the emergency department again. 12-lead ECG showed ST-segment elevation in V1-V4 leads (Figure 1). Emergency coronary angiography demonstrated total occlusion of LAD with normal left main, circumflex and right coronary arteries (Figure 2). Primary percutaneous coronary intervention was performed to the culprit lesion. After pre-dilatation of lesion with a balloon catheter, a 3.0 mm x 20 mm drug-eluting stent (CYPHER, Cordis Corporation, Warren, NJ) was implanted and TIMI 3 flow was achieved. Aspirin with a loading dose of 300 mg, ticagrelor with a loading dose of 180 mg and 8000 IU un-fractioned heparin were given before the procedure. Three days after the coronary intervention, patient was discharged from the hospital without any complication.

Discussion: Blunt chest trauma may cause many cardiovascular injuries. A wide range of complications may occur including dissection, aneurysm, intimal tear and sub-intimal hemorrhage of aorta and coronary arteries, ruptured of cardiac chambers, cardiac valves injuries, and pericardial effusion with or without tamponade. However coronary artery injury causing AMI is a rare outcome after blunt chest trauma. AMI may be seen due to occlusion of coronary artery by dissection, spasm or thrombus. The most frequently affected coronary artery after trauma is LAD artery (72%). ECG must be seen in all patients with chest traumas even if patients are younger and have no cardiovascular risk factors. Determining the cause of chest pain in these patients may be difficult due to trauma. Serial electrocardiograms and troponin measurement should be considered in cases where clinical suspicion continues. Percutaneous coronary intervention is the preferred treatment modality in these cases. Avoiding from thrombolysis is advisable because of the bleeding risk.

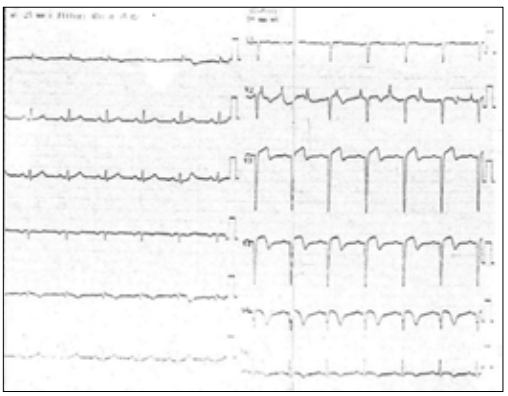

Figure 1. ECG at second administiration to emergency department.

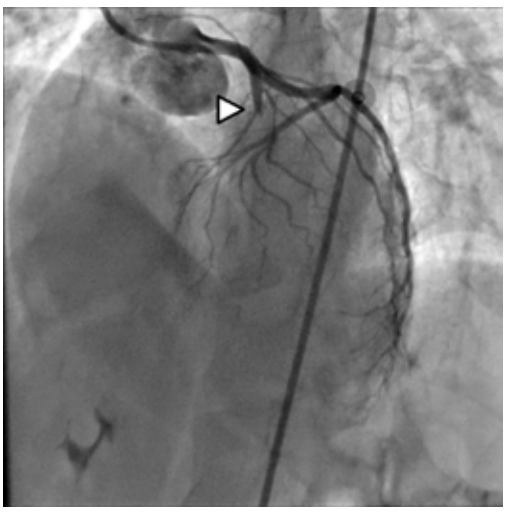

Figure 2. Total occlussion of LAD (Arrow Head).

OPS3-10

Triamsinolon kullanımına bağlı gelişen Kounis sendromu

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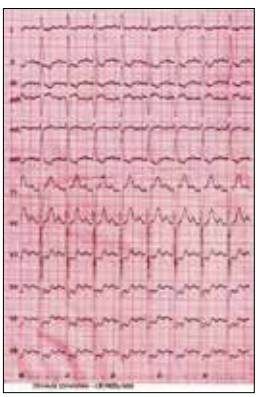
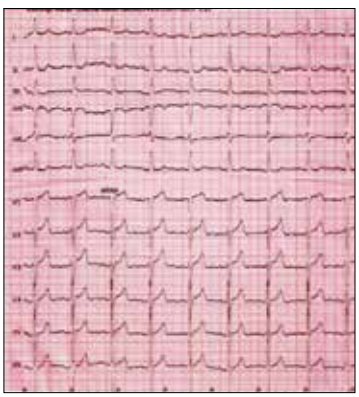
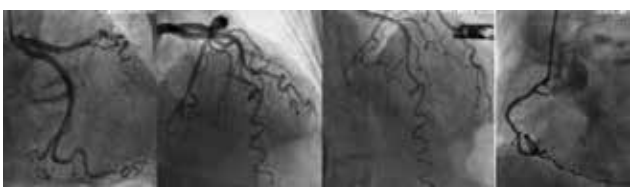
Akut koroner sendromlar ile alerjik veya hipersensitivite reaksiyonlarının birlikte görülmesi klinik pratikte gittikçe artmaktadır. 1991'de Kounis ve arkadaşları "alerjik anjina sendromunu" tarif etmiştir. Braunwald 1998'de alerjik reaksiyonlarda histamin ve lökotrienlerin koroner damar düz kaslarında spazm oluşturabileceğine işaret etmiştir. Herhangi bir alerjik maruziyet sonrası mast hücre aktivasyonu sonucu ortaya çıkan histamin, triptaz, lökotrienler, tromboksan gibi açığa çıkan bir takım mediyatörler aracılığı ile koroner vazospazm görülebilmektedir. Günümüzde alerjik anjina veva alerjik miyokart enfarktüsü "Kounis Sendromu" olarak adlandırılmakta ve koroner arter spazmının sebebi olarak kabul edilmektedir. Antibiyotik, analjezikler dâhil birçok ilaç, kontrast maddeler, arı ve böcek sokması, insektisit maddeler ve bazı gıdalar alerjik nedenli koroner vazospazm yaparak Kounis sendromuna neden olabilmektedir. Prednol ve antihistaminiklerle vazospazm düzelmekte ve klinik cevap alınabilinmektedir. Bu olgumuzda bir kortikosteroid olan triamsinonolon kullanımı sonrası gelişen kounis sendromu hastamızdan bahsedilecektir. Olgumuz 52 yaşında bayan hasta. Hastamız kliniğimize dış merkezden non-ST elevasyonlu miyokard infarktüsü tanısıyla sevkedildi. Hasta göğüs ağrısı öncesinde dış merkezde kafa derisinde döküntü ve kaşıntı nedeni ile dermatoloji kliniğine başvurmuş. Dermatoloji uzmanınca yapılan değerlendirme sonucunda hastaya dermatit tanısıyla, Triamsinolon asetonid 40 mg/ml (Kenacort-A Retard IM Ampul) tedavisi reçete edilmiş. Tedavinin ilk dozundan yaklaşık 15 dakika sonra hastanın tipik bir anjinası başlamış. Hasta anjina nedeni ile dış merkez acil kliniğine başvurmuş. Çekilen EKG'sinde DII, DIII, aVF, V3-V6 derivasyonlarında belirgin ST çökmesi olması üzerine kliniğimize sevk edilmiş. Hasta kliniğimize geldiğinde ağrısı oldukça azalmış ve EKG değişiklikleri devam etmekteydi. Biyokimyasal testlerinde troponin pozitifliği dışında özellik yoktu. Hastanın ağrısı triamsinolon intramuskuler tedavisi sonrası başladığı için alerjik bir vasospazm düşünülerek 1 ampul antihistaminik (Feniramin maleat 45.5 mg/2ml, Avil amp) yapıldı. Aynı zamanda standart tüm antiiskemik tedavi verildi. Hasta koroner anjiografi laboratuarına alındı. Yapılan koroner anjiografisinde koroner arterler normal olarak değerlendirildi. Bu nedenle hasta kounis sendromu olarak kabul edildi. Önerilerde bulunarak taburcu edildi. Kounis sendromu tedavisinde antihistaminikler ve prednol gibi steroidler kullanılmaktadır. Bir kortikosteroid olan triamsinolon ile gelişen kounis sendromu bu vakamız bu yönüylede oldukça farklı bir vakadır.



Şekil 1. Hastanın geliş EKG'si.



Şekil 2. Hastanın takip EKG'si.



Şekil 3. Koroner anjiografi görüntüleri.

OPS3-11

Ketiapin induced Kounis syndrome

Çağlar Kaya, Servet Altay, Mustafa Adem Yılmaztepe, Fatih Mehmet Uçar

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Kounis syndrome has been defined as an acute coronary syndrome that manifests as unstable vasospastic or nonvasospastic angina, and even as acute myocardial infarction triggered by the release of inflammatory mediators following an allergic insult. A 28-year-old male patient with known history of food allergy was admitted to the emergency department with dyspnea, nausea, and chest pain. Blood pressure and heart rate were 90/60 mmHg and 74 bpm respectively. On physical examination there was generalized urticaria, and dyspnea In the emergency department ECG showed ST segment elevation leads II, III, AVF (Figure 1).An immediate coronary angiography was performed with the diagnosis of inferior miyocardial infarction. Normal coronary anatomy was detected (Figure 2). From the detailed history, it was learned that the patient was a heavy smoker. the patient was using 200 mg ketiapin for sleeping.The patient also showed symtoms of allergy, dispnea and urticaria. With these signs and symptoms the diagnosis was thought to be Kounis syndrome induced by ketiapin.The main pathophysiological mechanism of coronary spasm in Kounis syndrome is the inflammatory mediators released during a hypersensitivity reaction triggered by food, insect bites or drugs. Here, we report a case of coronary spasm secondary to allergic reaction following ketiapin intake.To the best of our knowledge, this is the first case of ketiapin induced Koinus syndrome.

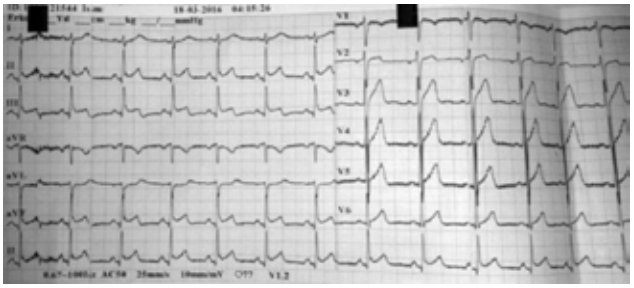

Figure 1. ECG shows ST segment elevation leads II, III, AVF.

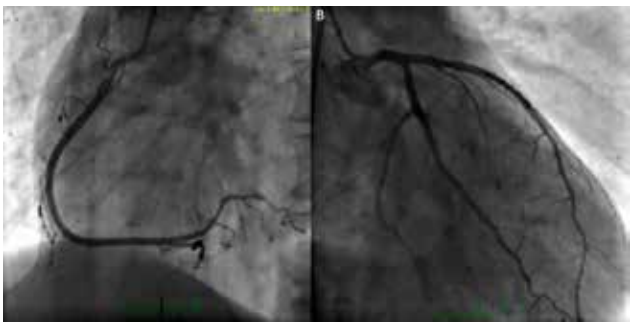

Figure 2. Coronary Angiography: Normal coronary arteries.

OPS3-12

Diagnostic dilemma in emergency department: acute aortic dissection versus acute myocardial infarction

Mert İlker Hayıroğlu, Muhammed Keskin, Koray Demir, Göksel Çinier, Ahmet Okan Uzun, Mehmet Eren

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Acute myocardial infarction and acute aortic dissection are both emergent circumstances requiring rapid intervention. Differential diagnosis plays a vital role in order to rescue patients. Here we discuss 72 years-old man admitted to our emergency department with crushing chest and back pain started an hour ago. The first electrocardiogram (ECG) revealed nothing significant. Chest X-ray showed expanded mediastinum; fast bedside transthoracic echocardiography (TTE) demonstrated ascending aorta aneurysm with moderate to severe aortic insufficiency, ejection fraction was also normal. Urgent contrast enhanced thoraco-abdominal computerized tomography was performed with acute aortic dissection preliminary diagnosis. Despite aortic aneurysm, dissection flap could not be determined in CT. While waiting for the laboratory results, repeat electrocardiograms revealed ST segment elevation on the precordial derivations. Patient was immediately transferred to the catheter laboratory, coronary angiography showed critical stenosis in the left main coronary artery. Patient underwent urgent surgery, left internal mammary artery- left anterior descending artery bypass in addition Bentall was performed successfully.

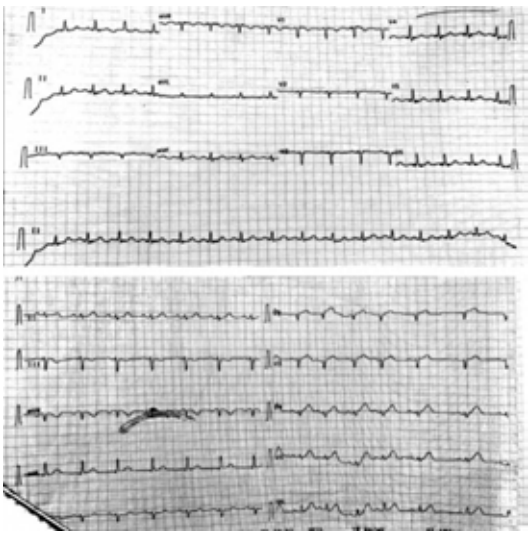

Figure 1. ECG recorded at the beginning, ECG recorded after increased pain.

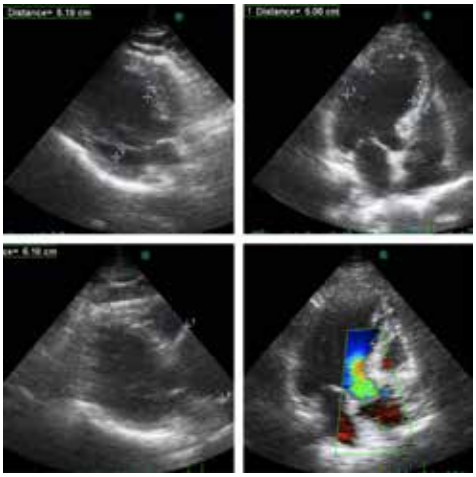

Figure 2. Ventricle is not dilated, aortic dilatation and aortic insufficiency were demonstrated.

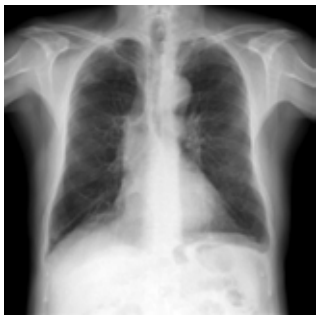

Figure 3. Chest X-ray showing dilated mediastinum.

OPS3-13

Diffuse coronary aneurysms in both of three vessels in a patient with acute inferior myocardial infarction

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[2]*Department of Cardiology, Mersin Univensity Faculty of Medicine, Mersin*

Case: A 63 year old male patient with a medical history of hypertension was admitted to emergency department for chest pain for 2 days. Electrocardiogram revealed acute inferior myocardial infarction with q waves and 1 mm ST elevation in inferior leads (Figure 1). On physical examination there was a diastolic decrescendo murmur on the Erb point. Thransthorasic echocardiography showed ejection fraction 45% and mild aortic regurgitation. PCI was planned and coronary angıography was performed. Proximal segments of left anterior descending (LAD), circumflex (Cx) and right coronary artery (RCA) were aneurysmatic and there was marked stenosis in the distal RCA with coronary slow flow and thrombus form through the lumen of proximal RCA and total occlusion in PDA (Figure 2a-d). After balloon intervention to the culprit lesion, the patient was treated with tirofiban and unfractured heparin infusion (Figure 3a, b). Physical examination revealed no specific findings and his chest pain was relied. He was discharged with dual antiplatelet theraphy in addition to his antiischemic therapy.

Discussion: The best treatment strategy for coronary artery aneurysms is still controversial and unclear, as there is no single management strategy. Available options are either a more conservative approach; using antiplatelet and anticoagulation therapy with regular follow up, percutaneous coronary intervention with possible stenting, or surgically using coronary artery bypass grafting. In this case we tried to draw attention that patients should be managed individually according to the location of the aneurysm and the clinical context.

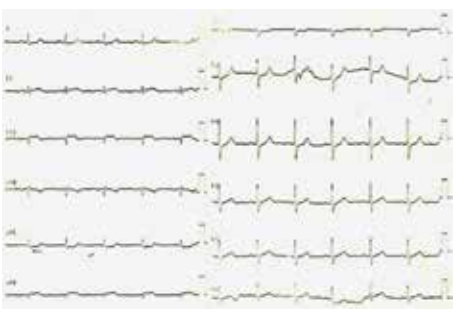

Figure 1.

OPS3-14

Acute myocardial infarction associated with the induction of general anesthesia

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[2]*Department of Cardiology, Van Erciş State Hospital, Van*

Introduction: Cardiac complications are the most common cause of postoperative morbidity and mortality. One of them is perioperative acute myocardial infarction(AMI); is a rare but important and fatal complication. Although there are many risk factors for development of perioperative AMI, it may occur without any conventional risk factors. Recognizing this situation may be difficult due to the influence of general anesthesia.

Case Report: A 56-year-old female admitted to urology department and diagnosed the right nonfunctioning kidney with the repetative urinary tract infection. For these reasons the nephrectomy operation was planned.After intubation, the patient has bradycardia and hypotension with ST segment elevation in DII lead. 12 lead ECG showed ST segment elevation in inferior leads and depression in V1-3(figure1). The echocardiographic examination revealed that left ventricular dysfunction with ejection fraction 40%-45%, hypokinetic inferior and posterior wall.Acute inferoposterior myocardial infarction was diagnosed and the angiography showed that proximal portion of the Cx was occluded by thrombus, LAD and RCA were plaque. Percutan coronary angioplasty and the stenting resulted in successful dilatation of the Cx and the ST changes improved instantly(figure 2). She was discharged without any complication and the operation postponed 6 months later.

Discussion: Several factors can cause the myocardial ischemia in perioperative period. Martinez et al. reported that the optimization of supply-demand balance in myocardial oxygen decreased the mortality rate in sixth months. Patients with abnormal preoperative ECG findings such as in our case, has more cardiovascular risk than those with normal ECG findings. Also the close monitoring ST segment and the hemodynamic values are very important to recognize AMI during the operation despite to the influence of general anesthesia. Because of increasing levels of the catecholamines and the corticol after hemodynamic changes or as a result from pain,surgical trauma,anemia and hypothermia, heart rate, blood pressure and vascular shear stress can increase and these conditions can lead to plaque instability. Also it is well known that some anesthetic agents sensitizes the myocardium to the catecholamines. It was reported that the adrenaline (A) and the noradrenaline (NA) stimulate platelet aggregation such as thromboxane A2, ADP and collagen. High level of the A and NA values are partially related to increased platelet activity, by stimulating thromboxane A2. Thereby ventricular arrhytmias and other adverse events such as perioperative AMI, can be seen during the surgical stress period.

Conclusion: Close monitoring of the hemodynamic parameters and the ST segment during operations are very important for recognizing perioperative cardiac complications which are the most common cause of postoperative morbidity and mortality. Early diagnosis and treatment of these complications can decrease the morbidity and mortality.

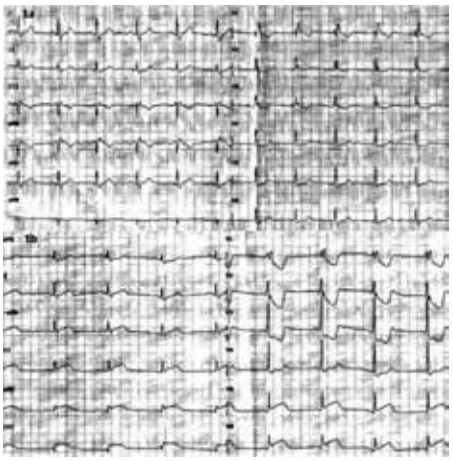

Figure 1. (A) Sinus rhythm with right bundle branch block, **(B)** ST segment elevation in inferior leads and depression in V1-3.

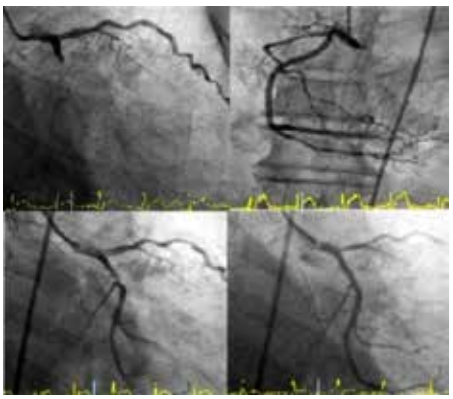

Figure 2. Proximal portion of the Cx was occluded, LAD and RCA were plaque. Percutaneous transluminal coronary angioplasty and the stenting resulted in successful dilatation of the Cx.

OPS3-15

ST elevation myocardial infarction as first manifestation of acute leukemia

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[2]*Department of Cardiology, Mersin State Hospital, Mersin*

Intraduction: Acute myocardial infarction refers to myocardial necrosis occurring as a result of acute myocardial ischemia, and is the main cause of death and disability in adults. Acute myocardial infarction is is divided into five groups based on etiology in the Third Universal Definition of Myocardial Infarction. Type 2: In instances of myocardial injury with necrosis where a condition other than coronary artery disease contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension. Anemia is a major reason of Type 2 myocardial infaction. Acute myeloid leukemia is a group of hematopoietic malignancies in which abnormal myeloblast proliferation results in the disruption of erythrocyte and platelet production in the bone marrow.

Case Report: A 66-year-old female patient was admitted to our clinic with complaints of fatigue, exertional chest pain and shortness of breath. The patient's complaints had started about 1 month earlier and worsened over the 12 hours before admission. ECG showed peaked T-wave in leads V1–V6 and then significant ST-wave elevation in the anterior leads and ST-wave depression in the aVR lead (Figure 1a). Echocardiography showed left ventricle EF of 45% and hypokinesia in the anteroseptal wall (Figure 2). Without waiting for the results of her blood tests, a primary percutaneous coronary intervention was planned and urgent coronary angiography was performed. Narrowing of the epicardial coronary arteries which would cause ischaemia was not observed on the coronary angiography (Figure 3). Her hemoglobin was 6 g/dL and platelet count was 55.000/μL, other chemistry panel was within normal limits Because of the lack of severe stenosis in the coronary arteries, we diagnosed secondary myocardial infarction due to anemia. A peripheral blood smear, was consistent with Acute myeloid leukemia, and the patient was transferred to the hematology department for chemotherapy.

Discussion: In patient with the diagnosis of ST elevation myocardial infarction, sometimes normal coronary arteries may seen. We believe that the imbalance between oxygen supply and demand due to impaired myocardial perfusion led to evolvement of type 2 myocardial infarction at this case. The prognosis in accompanying myocardial infarction and Acute myeloid leukemia is clearly worse.

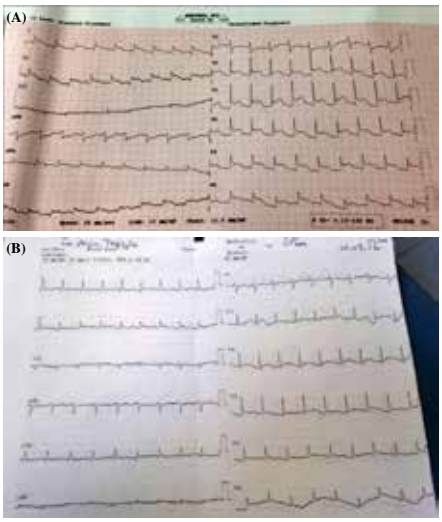

Figure 1. (A) Admission electrocardiography. **(B)** The normalized electrocardiogram after red blood cell transfusion.

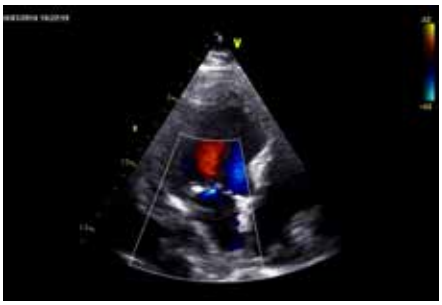

Figure 2. Admission echocardiography.

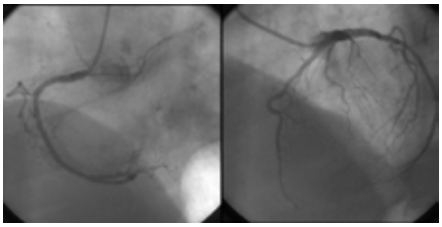

Figure 3. Normal epicardial coronary arteries on coronary angiography.

OPS3-16

VEGFR tirozin kinaz inhibitörü sunitinib ile ilişkili stent trombozu vakası

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VEGFR tirozin kinaz inhibitörleri sunitinib ve sorafenib böbrek hücreli karsinom ve gastrointestinal tümörlerde sık kullanılan ajanlardandır. Bu ajanların etkinlik ve advers etkilerinin incelendiği çalışmalarda kardiyovasküler olay riskini arttırdığı belirlenmiştir. Ön planda konjestif kalp yetersizliği ve kardiyomiyopatiyi arttırdığı görülse de bu ajanları kullanan hastalarda akut miyokard infarktüsü sıklığında da artış meydana gelmiştir. Tirozin kinaz inhibitörlerinin stent trombozuna neden olması ise nadir bir durumdur.

Olgu Sunumu: Altmış üç yaşında bayan hasta acile baskı tarzında göğüs ağrısı ve kusma şikayetiyle başvurdu. Göğüs ağrısı 3 saattir mevcuttu. Hikayesinde 6 yıldır hipertansiyon hastalığı olduğu, 35 paket/yıl sigara öyküsü olduğu ve anne ve babasında koroner arter hastalığı mevcut olduğu öğrenildi. Gastrointestinal tümör nedeniyle 3 kez opere olduğu ve idame tedavi olarak sunitinib kullanmaya devam ettiği belirlendi. Fizik muayenede ciltte solukluk ve tansiyon düşüklüğü (80/40 mmHg) saptandı. Elektrokardiyogramında D2-3-aVF derivasyonlarında ST yükselmesi görüldü. Hasta akut inferior miyokard infarktüsü tanısıyla primer revaskülarizason amacıyla kateter laboratuarına alındı. RCA distal segmentte total oklüzyon saptanan hastaya ilaç kaplı stent ile girişim uygulandı ve revaskülarizasyon sağlandı. Hastaya koroner girişim sonrası asetilsalisilik asit, tikagrelor, metoprolol ve atorvastatin başlandı. Aynı zamanda kullanmakta olduğu sunitinib tedavisine devam edildi.AMI sonrası 4. günde hastanın şiddetli göğüs ağrısı olması üzerine çekilen elektrokardiyogramda inferior derivasyonlarda yeni gelişen ST yükselmesi görüldü ve RCA distal segmentteki stent içinde trombüs ve RCA proksimal bölgede %70 darlık görüldü. RCA distal stent içine PTCA yapıldı ve proksimal lezyona direk stent implantasyonu yapıldı. İkinci işlemden 3 gün sonra şiddetli göğüs ağrısı yaşayan hastada 3. kez akut inferior miyokard infarktüsü saptandı ve kateter laboratuarına alındı. Hastanın RCA mid segmentte 2 stent arasında subtotal lezyon olduğu görüldü ve direk stent implantasyonu yapıldı. Hastanın kullanmakta olduğu sunitinib ile ilgili literatür taraması yapıldı. VEGFR tirozin kinaz inhibitörü sunitinib/sorafenib ajanlarının kardiyovasküler olay sıklığında artışa sebep olduğu anlaşıldı. Ancak daha önce stent trombozu ile ilişkili olduğu vaka bildirimi bulunamadı. Medikal onkoloji ile konsülte edilen hasta için sunitinibe bağlı stent trombozu riski olabileceğinden sunitinib kesilmesi uygun görüldü. Sunitinib kesilen hastada 1 haftalık yoğun bakım takibinde akut koroner sendrom tekrarlamadı. Hastanın 1. ay ve 3. ay kontrollerinde yeni bir akut koroner sendrom gelişmediği görüldü. Hastanın klinik seyri ve literatür bilgileri ışığında vakanın sunitinibe bağlı tekrarlayan akut inferior miyokard infarktüsü olduğu anlaşılmaktadır. Nadir görülen bu durumun klinisyenlerin bilgisine sunulması ve bu tür vakalarda gözlem süresinin uzatılmasının düşünülmesi açısından bu baka bildirimi önemlidir.

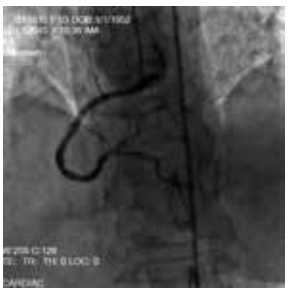

Figure 1. 2. akut koroner son hali. 2. revaskülarizasyon sonrası.

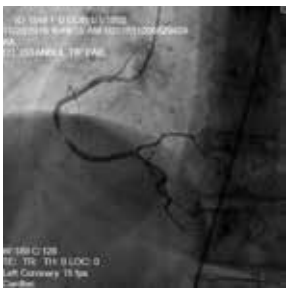

Figure 2. 3. akut koroner anjiografisi. 3. MI anjiosu.

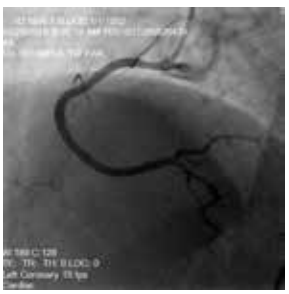

Figure 3. 3. revaskülarizasyon son hali. 3. revaskülarizasyon sonrası durum.

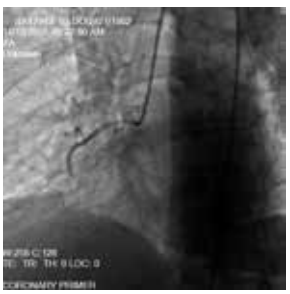

Figure 4. Anjio 1 başlangıç. İlk akut koroner sendrom görüntüsü.

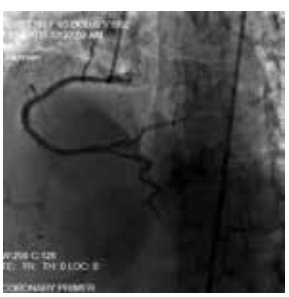

Figure 5. Anjio 1 son. 1. revaskülarizasyon sonrası.

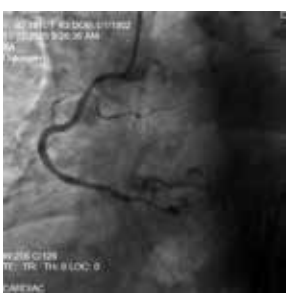

Figure 6. Anjio 2 başlangıç. 2. akut koroner sendrom koroner anjiografisi.

OPS3-17

Ischaemia induced preexitation resolved by ranalozine

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A 52- years- old man presented with exercise induced chest pain and palpitation. He a history of CABG surgery 8 years ago and a postero – septal ablation for WPW 6 months ago. ECG during angina revealed SR, left axis and WPW pattern with short PR, delta waves and 2mm ST depression in precordial derivations. After IV nitrate (200 mcg bolus) chest pain and ECG changes resolved (Fig. 1). With elevated cTnT levels he was hospitalized as nonSTEMI. Coronary angiography totally occluded LAD and Cx proximally and RCA at mid portion with patent LIMA-LAD and Ao-CXPL grafts and an occluded vein graft to RCA. Heart team decided for optimal medical treatment he was given ASA 100 mg, Ticagrelor 2x90 mg, Metoprolol tartarate 50 mg, zofenoprile 15 mg, atorvastatine 40 mg and iv nitrate. During hospitalization he had recurrent chest pain episodes induced by minimal effort accompanied with same ECG changes so that iv nitrate bolus had to be given and the maintenance dose was increased and a long acting nifedipine 30 mg was added to therapy. Efforts to change to oral nitrate was unsuccessful. As he continued to have ischaemic episodes Ranolozine 375 mg twice a day was added after which he became symptom free and discharged two days later. The appearance of the WPW pattern during angina can be explained by transient ischaemia blocking/prolonging conduction in the AV node and activating the accessory pathway. This may indicate an ablation failure as well as a second accessory way.

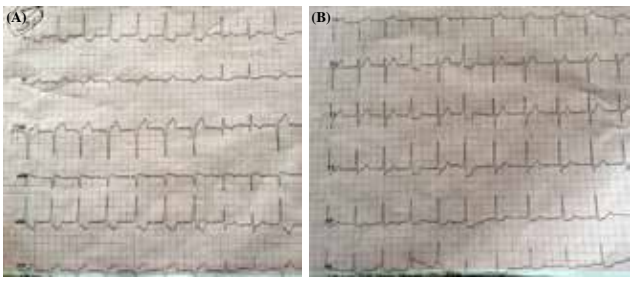

Figure 1. (A, B) Ischaemia induced WPW.

OPS3-18

A rare complication of hemorrhage into rectus abdominis sheath due to cough in an elderly patient under dual antiplatelet therapy and anticoagulation

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Patient was a 72 years old female who presented with acute dyspnea to the emergency room. Ortopnea and coughing was remarkable on the inspection. Oxygen saturation was 88% and she had inspiratory rales on the chest auscultation. ECG revealed ST segment depression on inferior lateral leads without any co-existing ST segment elevation. Cardiac troponin and CKMB was measured high on the laboratory test. Treatment targeting acute heart failure high probably due to acute myocardial ischemia was initiated. Dual antiplatelet therapy including acetylsalysilic acid and clopidogrel and anticoagulation by enoxaparin were initiated in coronary care unit. Although the patient's dyspnea was improved her cough did not relieved. In the morning patient started to complaint about acute abdominal pain characterized as stabbing. Her abdomen was tender. Patient was consultated by general surgery. We observed that a few localized lumps with tenderness have already developed on her abdomen. Ultrasonograhic examination of soft tissue on the abdomen revealed that there has been echo-free areas which were compatible with fluid on the subcutaneous tissue (Figure 1). These echo free areas were spontaneously developed hemorrhages which were limited between the rectus abdominis sheaths. It was triggered by persistent coughing while the patient was on dual antiplatelet therapy and anticoagulation. Lumps were resolved in several days without leaving any deficit. Most significant characteristics of lumps were being limited into sheath and absence of ecchymosed and discoloration of skin probably due to limitation of hemorrhage into sheath. Hemorrhage into rectus abdominis sheath is a rare complication which may be overlooked in elderly patients with acute abdominal pain. Usage of dual antiplatelet therapy and anticoagulation may promote such hemorrhagic complications. Coughing may trigger hemorrhage by increasing intrathoracic and intraabdominal pressure and also in vascular structures around these systems.

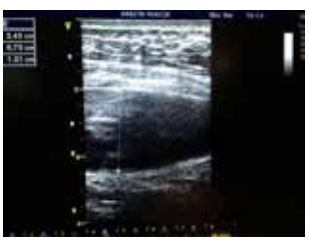

Figure 1. Hemorrhage into rectus abdominis sheath due to persistent coughing in an elderly patient under dual antiplatelet therapy and anticoagulation.

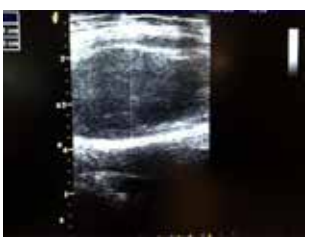

Figure 2. Echo free area compatible with hemorrhage into muscle sheath.

OPS3-19

A scorpion sting mimicking inferior myocardial infarction; Kounis syndrome

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Scorpion stings incidence are highest in rural regions of hot countries.The most common manifestations are local symptoms including severe pain, erythema, and burning sensation at the site of the sting. Nevertheless, severe cardiac complications for instance myocardial infarction (MI), acute pulmonary edema, cardiogenic shock myocarditis and even death occur rarely. Acute coronary syndromes after envenomation exposures are referred to as the Kounis syndrome or allergic myocardial ischemia and infarction.We present here in a case report with the clinical manifestations following scorpion bite mimicking acute myocardial infarction. A 55-year-old man with no previous disease or cardiac risk factor was stung by a scorpion in his garden. A few minutes after the sting, he felt severe local pain on his right leg, as well as several episodes of emesis, vomiting, dizziness. He had a mild chest pain at admission to emergency room. The vital signs were: heart rate 65 beats/min, blood pressure 110/54 mm Hg, and pulse oxymetry 96%. Cardiac and pulmonary auscultation were normal. Electrocardiogram revealed sinus rhythm with ST segment elevation in leads DII, DIII, aVF (Figure 1). Both cardiac troponins I and CK-MB were pozitive; 0.087 ng/ml, 21.3 ng/ml respectively. After scorpion antivenom was intravenously administered, the patient was admitted to the catheterization laboratory. Selective coronary angiography was performed revealing normal coronary arteries with preserved left ventricular function (Figure 2). The patient recovered very well and was discharged home on the following day with a seven day antihistamine treatment. The reported cases are attributable to hypersensitivity, and are referred to as the Kounis syndrome, with two identified subdivisions: type I, occurring in patients with angiographically normal coronary vessels (as was the case in our patient); and type II, occurring in patients in whom concomitant atheromatous lesions are found. Possible mechanisms include imbalance in blood pressure and coronary vasospasm caused by the combination of sympathetic excitation, scorpion venom-induced release of catecholamines, and the direct effect of the toxin on the myocardium. The management of cardiac ischemia in this setting is uncertain.

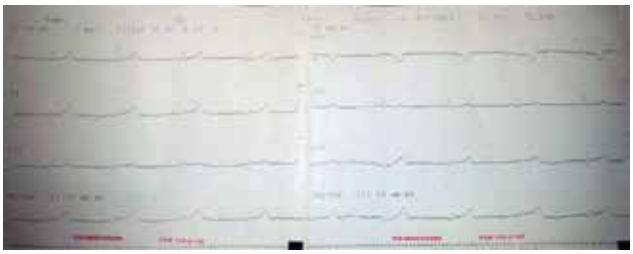

Figure 1. ECG.

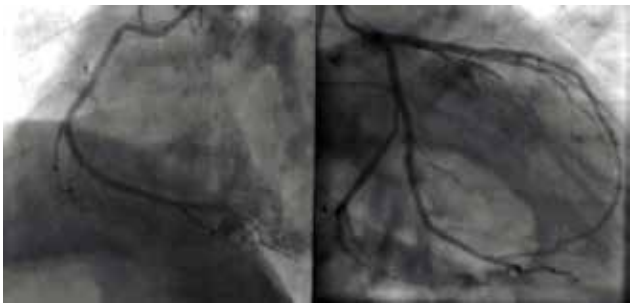

Figure 2. Coronary angiogram.

OPS3-20

Balon fraktürü vakası

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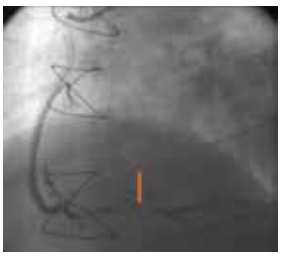
Mersin Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Mersin

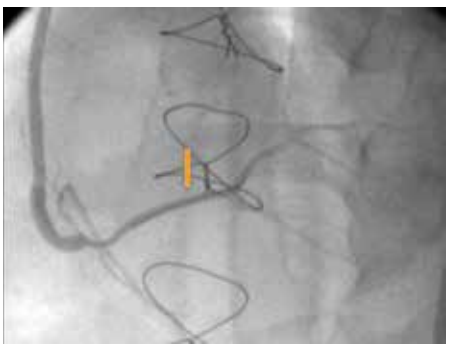
Dünyada koroner girişimlerin artması ile işlemde kullanılan malzemeler ile ilgili komplikasyonlarda artmaktadır. Burada perkütan koroner girişim (PKG) sırasında gövdeden kopan ve balonun lezyona sıkışıp kaldığı bir olguyu sunduk. On yıl önce üç damar koroner arter bypass greft operasyonu öyküsü olan altmış yedi yaşında erkek hastaya ST-yükselmesiz miyokard infaktüsü (MI) tanısı ile yapılan koroner anjiografi+bypass greft kontrolünde; Aort-RCA-safen greft anastomoz sonrası nativ damara PKG kararı alındı (Şekil 1). Aynı seansta 1.5x20 semikompliyan balon ile predilatasyon planladı. Lezyondan balon ile geçilip indeflator ile şişirilmeye başlanınca indeflator basınç göstergesinin yükselmediği ve balonun şişmediği izlendi. İndeflator kaynaklı olabilecek mekanik problem ihtimaline karşı indeflator değiştirilip tekrar balon şişirilmesi denendi. Buna rağmen balonun şişmemesi üzerine balonun değiştirilmesi planlandı. Balon çıkartılması için geriye doğru çekilirken balonun yetmişinci cm den kırılmış olduğu ve ilk bölümünün koroner lezyondan başlayıp guide kateter içine uzandığı izlendi. Hastanın semptomlarının artması ve elektrokardiyografik değişikliklerinin belirginleşmesi üzerine ikinci bir floppy tel ile lezyon ve balon seviyesi geçilip sarmal ile balonun geri çekilmesi planlandı fakat başarısız olundu. Sonra snare gönderilerek balon tutulmaya çalışıldı fakat snare tortiyoz ve kalsifik safen damarda takılıp kaldı. Daha sonra balon kateterin şaftının bir ucunun kateter içinde görülmesi üzerine başka bir kılavuz tel safene uzatıldı, 4x15 mm nonkompliyan bir balon alınarak kateter uç kısmında kırılan balonun gövde kısmı ile

guide kateterin proksimali arasında balon şişirildi. Böylece kırılan balon, guide kateter ile non-kompliyan balon arasında sıkıştırıldı. Sonrasında guide kateter ve içindeki bütün sistem ile birlikte başarılı bir şekilde çıkarıldı.(Şekil 2) Ardından Aort-RCA-safen greft anastomoz sonrası lezyona PKG uygulandı (Şekil 3). PKG işlemi sırasında oluşan komplikasyonlar MI, inme ve hatta ölüm ile sonuçlanabilmektedir. PKG sırasında balonun kırılması durumunda, kırılan parçayı çıkarmak için snare, forseps veya çift tel gibi teknikler kullanılabilir. Bu teknikler başarısız olursa cerrahi tedavi alternatif bir yöntemdir. Bizim vakamızda balonun kırıldığı yer guide kataterin içinde görüldü ve kırılan balonun gövdesi yeni bir balon ve katater arasına sıkıştırılarak çekildi. Girişimsel işlemler ile uğraşan kardiyoloğun işlem sırasında bir komplikasyon ile karşılaştığında soğukkanlı bir şekilde müdahale edecek bilgi ve tecrübeye sahip olması gerekmektedir.



Şekil 1. RCA'daki kritik lezyon.

Şekil 2. Kırılan balon.



Şekil 3. Başarılı PKG sonrası RCA.

OPS3-21

A very young acute myocardial infarction case after the use of low-dose oral contraceptives

Abdullah Nabi Aslan[1], Hüseyin Ayhan[2], Hakan Süygün[2], Serdal Baştuğ[1], Hacı Ahmet Kasapkara[2], Murat Akçay[2], Telat Keleş[2], Tahir Durmaz[2], Engin Bozkurt[2]

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Shortly after the introduction of oral contraceptives (OCs) in the 1960s, myocardial infarction (MI) was found to be associated with OC use. Although several but not all of the early studies showed evidence of increased risk of MI with current OC use, these findings were based on the use of OCs containing higher doses of estrogen than those now commonly prescribed. Third generation OCs containing low doses of estrogen and new progestagens are associated with a reduced risk of MI. In this case, we presented a 20-year-old young woman presenting with acute inferior MI and receiving a third generation low-dose OCs (drospirenone 3 mg and ethinyl estradiol 0.03 mg) for a duration of just one month. A 20-year-old non-smoker young girl was admitted to our polyclinic with a 3-day complaint of bilateral hand numbness and a retrosternal sharp pain. Physical examination was unremarkable. Electrocardiography showed 1-2 mm ST elevation at D2, D3 and aVF derivations that was accordance with an acute inferior MI (Fig. 1). Echocardiography also confirmed a wall motion disturbance at the inferior and posterior wall of the heart with a left ventricular ejection fraction of 48%. Her medical history revealed no cardiovascular (CV) risk factor including family history. Her body mass index was 24 kg/m^2. Biochemical investigations, which were done before the admittance, were as follows: glucose 81 mg/dL, blood urea nitrogen 21 mg/dL, creatinine 0.55 mg/dL, total cholesterol 182 mg/dL, triglyceride 69 mg/dL, high-density lipoprotein-cholesterol 53 mg/dL, low-density lipoprotein-cholesterol 115 mg/dL. She did not consume any drugs of abuse. She has been using orally a low-dose OCs including drospirenone 3 mg and ethinyl estradiol 0.03 mg for 1 month due to the complaint of dysmenorrhoea. Patient was taken to the catheterisation laboratory for the aim of primary percutaneous coronary intervention and loading dose of clopidogrel 75 mg and asetylsalicylic asit 300 mg was given. At coronary angiography, left anterior descending artery and circumflex artery was normal (Fig. 2a) but a total thrombotic occlusion at the distal of right coronary artery (RCA) was observed (Fig. 2b). Then, after passing through the lesion with a floppy guide-wire, percutaneous balloon angioplasty with a 2.0x12 mm balloon was performed (Fig. 2c). After the balloon angioplasty, coronary dissection was developed (Fig. 2d). Because, there was thrombolysis in myocardial infarction (TIMI) 3 flow in the RCA, stenting was not performed and the patient was transferred into coronary intensive care unit. A dose adjusted 12-hour tirofiban infusion with a bolus dose was started immediately. Despite having no other identifiable risk factors for an acute CV event and normal biochemical and hematological investigations, acute inferior MI was thought to be related to the using OCs. On the 3th day of admittance, she was discharged from the hospital because of her stabile condition.

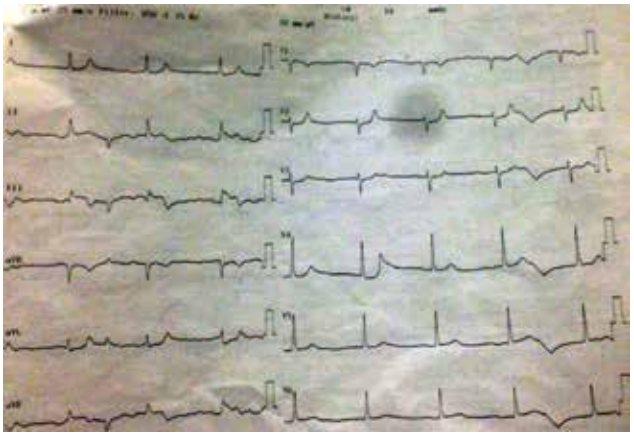

Figure 1. Electrocardiogram showing ST elevations in D2, D3 and aVF leads together with the reciprocal ST depressions in D1 and aVL leads.

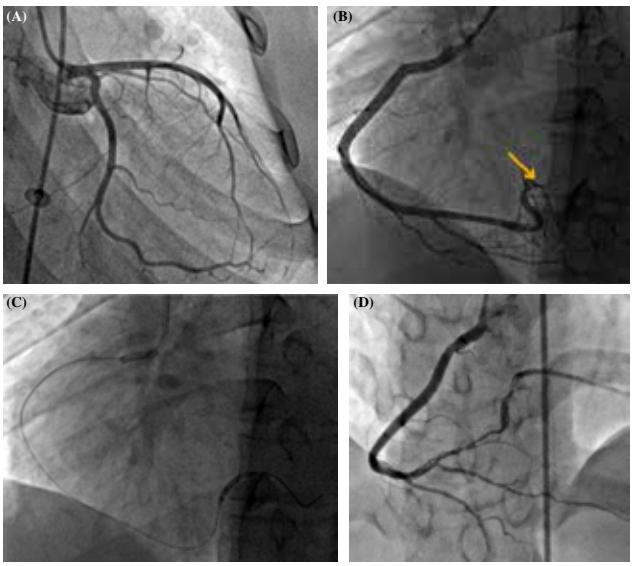

Figure 2. (A) Coronary angiography right caudal view showing normal left anterior descending and circumflex artery. **(B)** Coronary angiography left anterior oblique view showing a distal thrombotic occlusion at the right coronary artery (arrow). **(C)** Coronary angiography image showing balloon angioplasty. **(D)** Right coronary artery dissection developed after balloon angioplasty.

OPS3-22

Circumflex coronary artery totally occluded to that in acute inferior myocardial infarction in patients with right coronary artery anomaly originating from circumflex artery

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Aim: We offer a inferior miocardial infarction case because of circumflex artery total occluded which give right coronary artery.

Methods: A 56-year-old men patient with known history of hypertansion andsmoking was admitted to the emergency departmant with chest pain and vomiting. Blood pressure and heart rate were 130/85 mmHg and 98 bpm respectively. In the emergecy department ECG showed ST segment elevation leads D2, D3, aVF.

Results: An immediate coronary anjıography was performed with the diagnosis of inferior miocardial infarction. Because of electrocardiographic findings support the total RCA occlusion have begun working with left diagnostik catheter. During the process, circumflex artery was totally occluded from the mid segment. Left diagnostic process ending was passed to the right coronary diagnostic process in patients but could not find the RCA osteoma. Aortograf was performed whereupon (Figure 1a, b). Failure to follow the RCA outputs on aortagrafi was decided to attempt Cx artery. In patients undergoing percutaneous intervention Cx artery RCA native vessels were observed to watch as a continuation of the Cx artery (Figure 2a, b). Follow expired healing the patient was discharged.

Conclusion: RCA origin of the anomaly is a rare coronary artery anomaly has been reported as % 0.026. Most commonly it was found that out of the left sinus of Valsalva. Even if atherosclerosis RCA origin abnormalities have been reported to be associated with angina, myocardial infarction and sudden cardiac arrest. Acute inferior myocardial infarction patients is shows the ECG findings totaly occluded RCA but RCA field could not be displayed the possibility that RCA may have originated from the Cx should not be ignored.

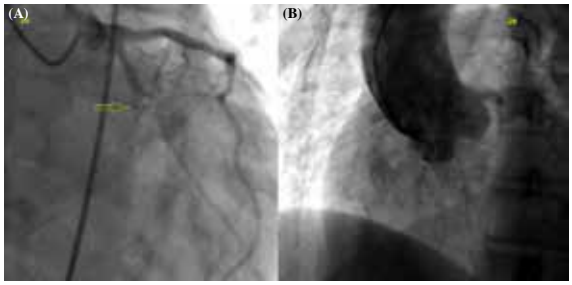

Figure 1. (A) Ocluded Circumflex artery. **(B)** Unknown RCA osteoma.

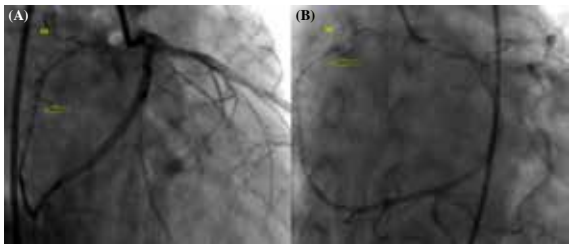

Figure 2. (A, B) RCA originated from Cx.

OPS3-23

Niemann-Pick disease type B and dyslipidemia in an adult patient

Abdullah Nabi Aslan[1], Hakan Süygün[2], Hacı Ahmet Kasapkara[2], Serdal Baştuğ[1], Hüseyin Ayhan[2], Murat Can Güney[2], Melike Polat[2], Murat Akçay[2], Telat Keleş[2], Tahir Durmaz[2], Engin Bozkurt[2]

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Niemann-Pick disease (NPD) type B is an autosomal recessive disorder. Here, we reported a NPD type B patient who presented with headache, weakness and fatique. He had been followed-up for NPD since 5 years of age. At physical examination, the liver was palpable at a 5-cm-breadth below the right costal margin. Because, he had splenectomy operation due to the traffic accident 12 years ago, the spleen was absent. His blood pressure was 120/70 mmHg and cardiovascular examination was normal. Electrocardiography was unremarkable. Echocardiography was normal. At laboratory investigation, the levels of the aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were elevated to 145 IU/L and 163 IU/L, respectively. His serum lipid profiles showed that total cholesterol was 481.5 mg/dL (normal range <200 mg/dL), LDL-cholesterol was 407 mg/dL (normal range <130 mg/dL), triglyceride was 298.7 mg/dL (normal range <200 mg/dL), and HDL-cholesterol was 15 mg/dL (normal range 35–55 mg/dL). 2 weeks later, the lipid assay was repeated to prevent a probable laboratory mistake and it confirmed that total cholesterol was 456 mg/dL (normal range <200 mg/dL), LDL-cholesterol was 386.3 mg/dL (normal range <130 mg/dL), triglyceride was 278 mg/dL (normal range <200 mg/dL), and HDL-cholesterol was 14.1 mg/dL (normal range 35–55 mg/dL). Before beginning the treatment of dyslipidemia, carotis Doppler ultrasonography was performed. Carotid intima-media thickness at the right carotid artery was detected as 1.2 mm with a local-intimal thickening and fibrofatty plaque. Abdominal ultrasonography showed hepatomegaly (18 cm). After all, rosuvastation 40 mg once a day was commenced for the treatment of hyperlipidemia and the patient was re-evaluated for both efficacy and potential side-effect of rosuvastatin 2 weeks later. The levels of the aspartate aminotransferase (AST) and alanine aminotransferase (ALT) had been decreased to 88 IU/L and 98 IU/L, respectively. There was also a decrease in the lipid profile of the patient. At follow-ups, the level of liver enzymes have been normalized and the lipid profiles was decreased to near-normal levels (total cholesterol 242 mg/dL, LDL cholesterol 168 mg/dL, HDL cholesterol 28 mg/dL and triglyceride 230 mg/dL) within 3 months after the therapy. Here, we have reported a NPD type B patient with dyslipidemia. He was treated with rosuvastatin, which resulted in improved lipid profiles and decreased levels of liver enzymes just after 3 months of therapy. Therefore, we can conclude that, rosuvastatin is safe and effective in the treatment of dyslipidemia in patients with NPD type B. However, the natural history of this disorder is unknown and efficacious dosage and adverse effects of the lipid-lowering agent have not been documented partly due to the hepatic dysfunction. Thus, further studies are required for the evaluation of long-term efficacy of lipid-lowering agents in NPD type B patients.

OPS3-24

Homozigot ailevi hiperkolesteroleminin uzun dönem komplikasyonu; İleri aort darlığı

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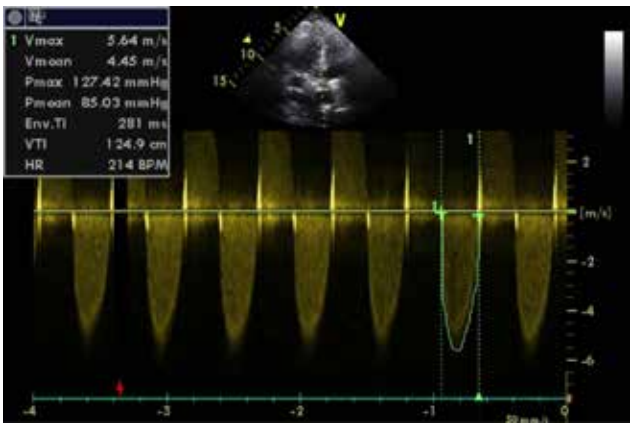
Ailevi hiperkolesterolemi (AH), erken ateroskleroza yol açan aşırı yüksek kolesterol düzeyleri ile karakterize genetik bir hastalıktır. Homozigot AH (HoAH) bulunan olgularda çocukluk döneminden itibaren kardiovasküler olaylar gelişebilmektedir. HoAH'lı hastalara tanı geç konmaktadır. Aferez tedavisine geç başlandığı için ateroskleroz ve aort darlığına ilerlemektedir. Bu vakada takipsiz HoAH hastasında uzun dönemde gelişen ileri aort darlığı anlatılacaktır.

Olgu Sunumu: Yirmi yaşında ve 24 yaşında iki kez bypass cerrahisi geçiren uzun süredir takiplere gelmeyen rosuvastatin 40 mg ve ezetimib 10 mg kullanan HoAH tanılı 30 yaşındaki bayan hasta göğüs ağrısı kliniğimize başvurdu. Akraba evliliği bulunan hastanın kızı da HoAH tanısı almış. Fizik muayenesinde göz etrafında ksantolezmalar ve sol ön kol ekstensör bölgede ksantom izlenen hastanın aort odağında 3/6 sistolik ejeksiyon üfürümü duyuldu. Hastaya yapılan transtorasik ekokardiyografisinde; ejeksiyon fraksiyonu %55, sol ventrikül hipertrofik, aort kapak ileri kalsifik ve kapak açılımı kısıtlı izlendi. Aort kapakta peak gradiyent 127 mmHg, mean gradiyent 85mmHg ölçüldü (Şekil 1). Aort kapak alanı <1 cm² hesaplandı. Yapılan karotis vertebral arter dopplerinde sağ internal karotis arterde %30 darlık, sol internal karotis arterde %50 darlık izlendi. Hasta endokrinoloji bölümüne danışıldı. Hastaya lipid aferezi tedavisi uygulanmasına karar verildi. Hastanın periferik vasküler yetersizliği nedeniyle lipid aferez yapmak için 22F kalıcı subklavian kateter takıldı. Hasta lipid afereze alındı. İşlem sonrası kontrol LDL değeri 93 mg/dl ölçüldü. Hastaya koroner anjiyografi sonrasında aort kapak operasyonu planlandı. Hasta tedaviyi kabul etmedi. Hasta lipid aferezi programına alındı. Takiplerimizde kateter enfeksiyonu gelişen hasta exitus oldu.

Tartışma: Ailevi hiperkolesterolemi, LDL-reseptör geninde mutasyon sonucu ortaya çıkan çok yüksek LDL düzeyi ve erken aterosklerozla karakterize bir hastalıktır. Yüksek düzeydeki lipitler deri altında, özellikle damarların proksimal bölgelerinde ve aort kapakta birikme eğilimindedir. Bu vakada lipid aferezi tedavisine geç kalmış bir hastada meydana gelen ileri aort kapak darlığı mevcuttu. Homozigot hastalarda aort darlığının engellenebilmesi için afereze çok erken yaşlarda başlanması gerekir. Bunun dışında lipid aferezi invaziv bir tedavi metodu olduğundan komplikasyonları da mevcuttur. Yeni gelişen yakın zamanda kullanıma girecek anti-lipid ilaçlar HoAH hastalarında lipid afereze alternatif olacaktır.



Şekil 1. Aort kapakta gradiyent.

OPS3-25

Supravalvular aortic stenosis in a patient with homozygous familial hypercholesterolemia

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Introduction: Homozygous familial hypercholesterolemia (FH) is an autosomal dominant disorder, in which number and function of LDL-receptors are decreased due to mutations, resulting in very high levels of LDL-cholesterol and premature coronary artery disease. Homozygous FH also affects aortic valve and aortic root.

Equipment and Methods: A 24-year-old male patient presented for a control examination. He had the diagnosis of homozygous FH and had bypass surgery at the age of 14. He was on ezetimibe, rosuvastatin, cholestiramin, aspirin and β-blocker therapy. Cardiac auscultation revealed an ejection murmur at the right upper sternal border. His lipid profile was as follows: Total cholesterol: 584 mg/dl, LDL-cholesterol: 493 mg/dl, HDL-cholesterol: 69 mg/dl. Other biochemical parameters were normal.

Results: On transthoracic echocardiography, the aortic wall was thickened from Valsalva sinuses to sinotubuler junction and the aortic lumen was narrowed (Video 1 and 2). Peak continuous wave Doppler velocity was 2.6 m/s and the mean systolic pressure gradient was 18 mmHg at the suprasternal level. The aortic valve was tricuspid with minimally thickened cusps and mild aortic regurgitation. Left and right ventricular functions were normal.

Conclusions: Homozygous FH patients present with premature coronary artery disease. The coronary artery stenosis site is typically ostial. On physical examination, cholesterol rich fat deposition in eyelids (xanthelasma palpebrarum), in the outer margin of the iris (arcus senilis corneae), and in the tendons of the hands, elbows, knees and feet, particularly the Achilles tendon (tendon xanthoma) can be seen. These patients may also have valvular or supravalvular aortic stenosis, secondary to cholesterol deposition. Aortic valve/root involvement is rarely seen in heterozygotes. Diagnosis is made by echocardiography, computed tomography, magnetic resonance imaging or aortic angiography. Supravalvular aortic stenosis may cause or aggravate anginal symptoms and aortic root replacement/enlargement is the mode of treatment when stenosis is severe. Transthoracic echocardiographic follow-up is recommended to screen aortic valve and aortic root complications in homozygous FH patients.

OPS3-26

In cases of doubt endocarditis PET / CT plays an important role

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Aim: We present the diagnosis of non bacterial thrombotic endocarditis by PET/CT

Methods: A 72-year-old female known history of hypertansion and diabetes mellitus was admitted to the emergency depatmant with weakness and feeling faint. Blood pressure and heart rate were 105/60 mmHg and 102 bpm respectively. Diffusion MR shot due to weakness was observed spot diffusion limitation and echocardiyography planned with the diagnosis of septic emboli. PML calcified root of Echocardiography image of the monitored patient was admitted for further examination.

Results: When transosephageal echocardiography was performed two mobile isoechogenic structure (Figure 1a-d). When examined in detail for duke criteria only 1 major findings except, no major and minor criteria has not been demonstrated. Patients in diagnosing the presence of active infection around the valve or exclude taken PET / CT showed irregular FDG uptake was increased level of mitral valve (Figure 2a, b). Patient was adopted nonbacterial thrombotic endocarditis. After diagnostic coronary angiography performed to the patients, coronary arteries bypass graft operation and mitral valve replacement was applied and the patient was discharged with healing.

Conclusion: DUKE criteria for infective endocarditis is the only vegetation in patients who do not meet criteria for minor and major especially to confirm the diagnosis of non bacterial endocarditis PET/CT can play a very important role.

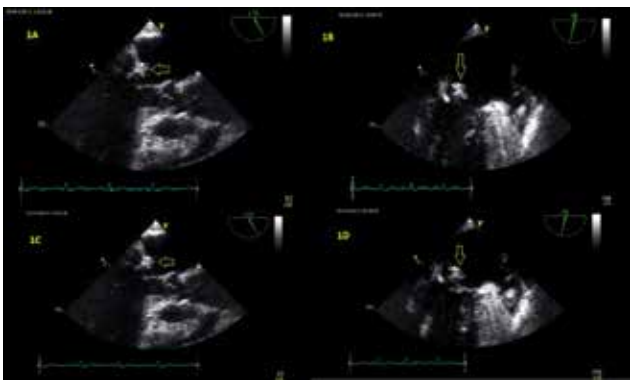

Figure 1. (A-D) Vegetation at echocardiography.

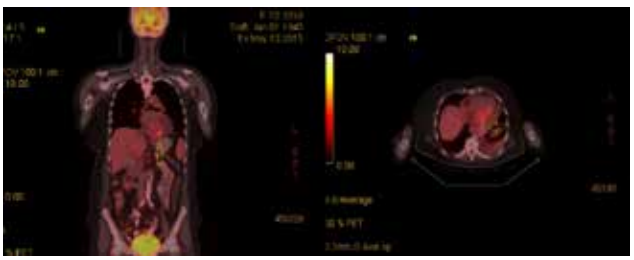

Figure 2. (A, B) FDG uptake at PET/CT.

OPS3-27

The importance of physical examination and a very rare cause of heart failure: SLE/scleroderma overlap syndrome

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A 32-year-old female patient applied to outpatient clinic with heart failure symptoms of NYHA class III. She had had a history of heart failure for one year, however, the interval of hospitalizations had increased for the last six months. The patient was prescribed ramipril 10 mg, carvedilol 12.5 mg bid, spironolactone 25 mg, digoxin 0.25 mg and furosemide 40 mg bid. Electrocardiography denoted sinus rhythm and echocardiography revealed an EF of 38% with a globally dilated left ventricle (LV), systolic pulmonary artery pressure (sPAB) of 65mmhg and moderate pericardial effusion. On physical examination, digital ulcers on second and third fingers of left hand and digital ulcer on the index finger of right hand were noticed. Afterwards, a more detailed inspection was made and an increased thickness of the skin of fingers and hand was ascertained as well as a slight and faint malar rash. Laboratory results revealed that thrombocyte count was 72.000/mm^3, anti-Scl70 and antinuclear antibody were positive. The patient was having findings of both systemic lupus erythematosus (SLE) and scleroderma. Thus, the patient was consulted to rheumatology department. After a comprehensive evaluation, the patient was diagnosed with SLE/Scleroderma overlap syndrome and hospitalized to rheumatology department. After six months of hydroxychloroquine sulfate, steroid, cyclophosphamide and iloprost therapy, echocardiography revealed an EF of 45% with a systolic pulmonary artery pressure of 37mmhg and without any pericardial effusion. Moreover, the patient was having NYHA class I-II symptoms. Our case emphasises the ongoing importance of physical examination in the era of many innovative medical imaging instruments in course of diagnosis. This case is a striking one to draw attention of junior cardiologists to how physical examination could play an important role in the process of diagnosis and treatment. Because this patient had had an intractable heart failure despite an optimal medical therapy for one year. Physical examination revealed the etiology of heart failure and resulted in resolution of symptoms after the treatment of underlying disease. Moreover, heart failure due to SLE/scleroderma overlap syndrome is a very rarely encountered condition. Rheumatologic diseases should also be kept in mind as an etiologic factor of heart failure, particularly, in patients with global dysfunction of LV, pericardial inflammation and/or markedly elevated sPAB.

OPS3-28

A case of hypereozinophilic syndrome presenting with acute heart failure

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Introduction: Eozinophilic myocarditis and eozinophil-mediated cardiac damage are manifestations of cardiac involvement in hypereozinophilic syndrome (HES) and a rare cause of acute heart failure.

Case Report: A 52 year-old man presented to our clinic with acute dyspnea. His previous medical history was insignificant except bronchial asthma. Physical examination revealed a S3 and fine inspiratory crackles in both lung zones. His ECG was consistent with sinus tachycardia and non-spesific ST segment abnormalities in precordial leads. The chest X-ray revealed alveolar edema and bilateral pleural effusion. His cardiac biomarkers were elevated. Blood tests revealed an elevated C-reactive protein of 150 mg/L (normal range 0-5 mg/L), erythrocyte sedimentation rate of 48 mg/dl (normal range 0-20 mm/hour), mild leukocytosis and elevated eozinophil count of 2.68x10^9 cells/L (normal range 0-0.7×10^9 cells/L). A coronary angiogram was performed and yielded normal coronary arteries. The echocardiogram showed diffuse global hypokinesia, moderate pericardial effusion and left ventricular apical thrombi. A cardiac MRI was performed and was nugatory except diffuse left ventricular wall motion abnormalities and apical trombi. The patient was investigated for secondary eozinophilia causes like parasitic infections, allergic and pulmonary diseases. None of them were present. A bone marrow biopsy was performed and showed material rich in eozinophilic granulocytes (%21 eozinophils). However, FIP1L1/PDGFRA fusion gene and JAK2 V617F mutation were absent. In the light of these findings, hypereozinophilic syndrome with cardiac involvement was suspected and corticosteroid treatment was initiated. Following treatment, the eozinophil count and C-reactive protein returned to normal. However, no change was observed in left ventricular function and the apical thrombi continued to increase in size despite adequate anti-coagulation. At follow-up, the patient died because of a massive mesenteric emboli.

Discussion: In conclusion, HES is a heterogeneous group of conditions defined by elevated eosinophil count of >1.5×10^9/L and subsequent organ damage. Cardiac involvement is frequent and a major determinant of prognosis in HES. Eozinophilic myocarditis and associated acute necrosis are observed during the initial phase of HES and should be suspected when there is an elevated eozinophil count and acute heart failure.

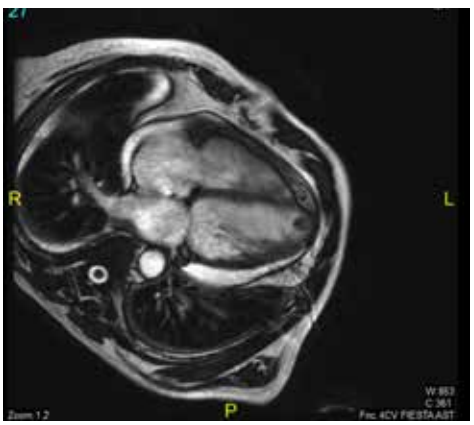

Figure 1. Left ventricular apical thrombi on cardiac MRI.

Figure 2. Left ventricular apical thrombi on TTE.

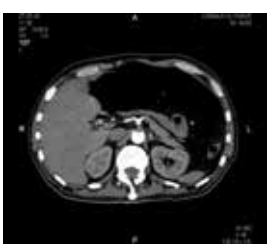

Figure 3. Superior mesenteric arterial thrombi on abdominopelvic CTA.

OPS3-29

An unexpected benefit of left ventricular assist device implantation: Spontaneous return to sinus rhythm in a patient with chronic atrial fibrillation

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Left atrial remodelling is the main pathophysiologic mechanism responsible for the maintenance of atrial fibrillation in mitral stenosis. Left ventricular assist devices act by decompressing the left ventricle, and by doing so, they also induce a reduction in left atrial volume. We report a patient with left ventricular failure and rheumatic mitral stenosis, whose rhythm was chronic atrial fibrillation unresponsive to cardioversion. A 47 years old patient was followed up by heart failure clinic due to severe, unresolving exertional and rest dyspnea for three years. He had severe mitral stenosis and left ventricular systolic function, with a valve area of 1.2 cm^2 and an ejection fraction of 20%. His rhythm was AF at his initial admission and during follow up visits (Figure 1a), and a rate-control strategy was adapted after two failed attempts of cardioversion. Six months before LVAD implantation, he underwent emergency atrial repair due to atrial perforation after a failed percutaneous mitral valvotomy attempt. He was previously accepted to heart transplantation program, but a decision to proceed with LVAD implantation was given due to unresolving NYHA Class IV - INTERMACS 4 symptoms and multiple (three times within six months) hospitalizations, along with a general lack of suitable heart donors. Prior to the operation, two and three dimensional echocardiograms were obtained using a Philips iE33 platform (Philips Healthcare, Andover, MA), which showed severe LV and LA dilatation, an ejection fraction of 20%, a LA anteroposterior (AP) diameter of 5.1 cm and a LA volume of 122.8 ml (Figure 1b and 1c). Mitral valvotomy was not planned prior to operation to avoid an unnecessary increase in operative duration and an increased mitral valve area was not expected to improve the patient's heart failure symptoms or overall survival. The operation was uncomplicated, and the patient was discharged from the hospital two weeks after the operation. At discharge, his heart failure symptoms had decreased substantially, but his rhythm was still AF and there were no remarkable changes on transthoracic echocardiogram except a reduction in LV dimensions. On a follow up outpatient visit at first month, his rhythm reverted back to sinus rhythm, with LA AP diameter of 4.5 cm and a left ventricular EF of 25% on two dimensional echocardiogram. His ECG's taken at third and sixth month visits also revealed sinus rhythm (Figure 1a), and a 3D transthoracic echocardiogram using the same echocardiography platform repeated at the sixth month showed a LA AP diameter of 3.6 cm and LA volume of 64.7 ml (Figures 1b and 1c). In addition to the reduction in left atrial size, his ejection fraction had increased to 45%. During his interview at sixth month, the patient was totally asymptomatic and had no palpitations for the previous five months.

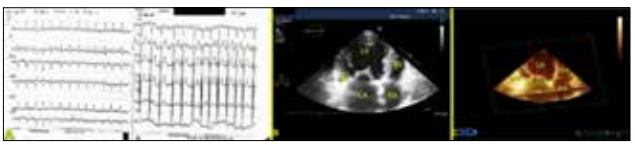

Figure 1. Electrocardiographic and echocardiographic findings of the patient prior to implantation of left ventricular assist device. His rhythm was compatible with coarse-wave atrial fibrillation, with a heart rate of 145 beats/minute **(A)**. Left atrium, as well as left ventricle was dilated on two dimensional echocardiography **(B)**, and his left atrium was measured as 122.8 ml with three dimensional transthoracic echocardiography **(C)**. MV, mitral valve; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

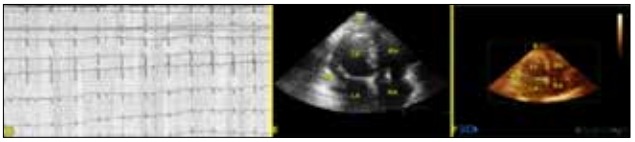

Figure 2. Electrocardiographic and echocardiographic findings of the patient six months after implantation of left ventricular assist device. His rhythm converted back to sinus rhythm at the ifrst month and sinus rhythm was maintained at sixth month, with a heart rate of 70 beats/minute **(A)**. Both left atrium and left ventricle was considerably smaller, as compared to right ventricle and atrium on two dimensional echocardiography **(B)**, while the left atrial volume was measured as 64.7 ml **(C)**. Note the left ventricular assist device inflow canula (arrow) within apical part of left ventricle **(B, C)**. MV, mitral valve; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

OPS3-30

Imatinib induced congestive heart failure and electrical storm in a young woman

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Introduction: Imatinib is a standard first-line therapy for chronic myeloid leukemia (CML). There have been sporadic reports of imatinib-induced cardiotoxicity, ranging from asymptomatic subclinical abnormalities and a decline in left ventricular ejection fraction to life-threatening events like congestive heart failure and acute coronary syndromes. In our case, we present a young woman who developed irreversible dilated cardiomyopathy following imatinib theraphy.

Case Report: A 25 year old woman was diagnosed with CML at the age 19. Following the diagnosis, imatinib was started. However, the patient stopped taking her medication because of an intervening pregnancy. The patient was diagnosed with pre-eclampsia during the seventh month of her pregnancy and had a C/S. One year later, she presented with generalised edema. A transthoracic echocardiography revealed poor left ventricular function and dilated ventricles. Imatinib was stopped and medication was started for heart failure with reduced ejection fraction. Also an ICD was implanted following a succesfully resuscitated VF and cardiac arrest during one of her hospitalizations for decompansation. During follow-up, the patient had a progression to blastic phase and hospitalized for acute leukemia. During her hospitalization, the patient had an electrical storm with multipl ICD shocks and cardiac arrest following a sustained ventricular tachycardia attack. She was successfully resussicated. Interrrogation of her device revealed 88 ICD shocks for VT and VF and EOL. Her ECG's revealed an excessively prolonged QTc interval of 680 msn. An immediate battery replacement of her ICD was performed, over drive pacing at 100 beats/minute was performed until the QTc interval returned to normal. At follow-up, the patient stabilised, QTc interval returned to normal and she had no further ICD shocks.

Conculusion: In conclusion, although it is extremely rare, imatinib and potentially also other ablkinase inhibitors might induce congestive heart failure. Aging is considered as a risk factor for the cardiotoxicity of imatinib. Our case is a rare example of imatinib induced cardiotoxicity occuring in a young patient. Also our case is the first in the literature, demonstrating a patient with imatinib induced heart failure and electrical storm.

OPS3-31

Akut anterior myokard i˙nfarktusu bulguları veren i˙nfluenza virus (H1N1) ilişkili fulminan myokardit

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Giriş: Akut miyokardit vakaları çok farklı etkenler sebebi ile ve değişken klinik tabloda presente olabilir. Fulminan myokardit ise nadir olmak ile birlikte oldukça mortal seyretmektedir. Biz vakamızda akut koroner sendromu taklit eden, etken olarak olarak H1N1 düşünülen fulminan myokardit vakasını sunacağız.

Olgu Sunumu: 46 yaşında kadın hasta,ani gelişen göğüs ağrısı yakınması ile acil servise başvurdu. Fizik muayenesinde akciğer bazallerde ralleri mevcuttu. Elektrokardiyogramında geniş QRS'li ritm, RBBB ile uyumlu, anterolateral derivasyonlarda iskemik olduğu düşünülen ST-T dalga değişiklikleri saptandı. Hastaya yapılan ekokardiyografide EF %45, apeks, anterior duvar ve lateral duvarın mid apikali hipokinetik saptandı. Hasta akut koroner sendrom ön tanısı ile koroneranjiografiye alındı ve koroner patoloji saptanmadı (Şekil 2, 3). Hasta koroner yoğun bakıma alındı. Laboratuvar tetkiklerinde akut faz reaktanlarının yüksekliği ile birlikte kardiyak trop I >55 ng/ml saptandı. Hastaya antiplatelet tedavi ve i.v düretik başlandı. Koroner yoğun bakım takibinde hastanın kliniği progresif olarak kötüleşti. Kardiyak trop I halen >55 ng/ml idi. Hastanın konjesyon bulgularının efektif doz düretik tedaviye rağmen gerilememesi hatta artması üzerine pozitif inotrop başlandı. Bir hafta sonra tekrar ekokardiyografi yapıldı ve EF <%35, LV global belirgin hipokinetik saptandı (Şekil 4). Hastada fulminan myokardit düşünülerek,antipaltelat tedavi kesildi,geniş spektrumlu antibiyotik ve antiviral tedavi başlandı. Öncesinde gribal semptomlarının olmadığının öğrenilmesine rağmen mevsimsel olarak uygun etken olduğundan influenza H1N1 suşu için boğaz sürüntü örneği gönderildi. Bu sürede hasta pozitif inotrop bağımlı olması ve konjesyon bulgularının progresif artması nedeni ile uygun merkeze invaziv dolaşım destek cihazı takılması açısından değerlendirilmek üzere yönlendirildi. H1N1 PCR sonucu pozitif olarak tarafımıza bildirilen hasta dış merkez takibinde kardiyojenik ve septik şok tablosunda exitus olmuştur.

Tartışma: Viral etyolojik nedenli fulminan myokardit vakaları nonspesifik semptomlarla başvurabileceği gibi, vakamızda olduğu gibi akut koroner sendromu da taklit edebilir. Fizik muayende kalp yetmezliği bulguları saptanır. Tanıda görüntüleme tetkikleri yanında şüphelenilen spesifik etkene yönelik viral pcr gibi uygun tetkiklere başvurulabilir. Tedavide yoğun bakımda maksimum hemodinamik desteğe rağmen mortalite %39 civarındadır. Eğer tedavi direnci var ve mekanik dolaşım desteği sağlanamıyor ise mortalite %89'a kadar çıkmaktadır. Eğer hastalar hayatta kalabilir ise uzun dönem prognozları iyidir. Myokardit'in viral etyolojileri arasında H1N1 mutlaka düşünülmelidir.İmmunsupresif tedavinin yeri tartışmalıdır. Vakamızda olduğu gibi atipik başvuran vakalarda fulminan myokardit tanısı unutulmamalıdır.Erken tanı konulması, mümkün olan en erken zamanda agresif hemodinamik desteğin ve antiviral tedavinin başlanması oldukça önemlidir.

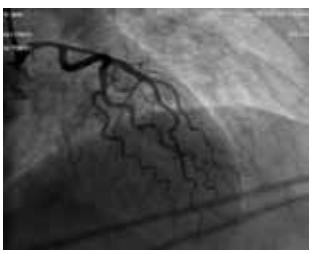

Figure 2. Koroner anjiografi.

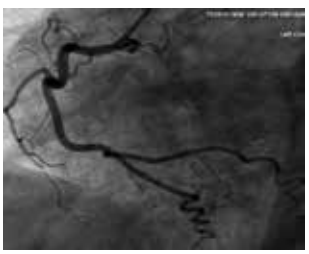

Figure 3. Koroner anjiografi.

OPS3-32

Conn sendromu ile ilişkili dilate kardiyomiyopati

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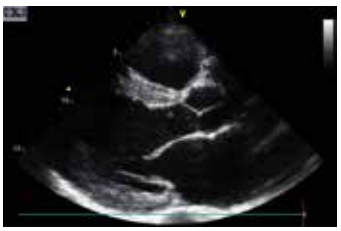
Giriş: Dilate kardiyomiyopati, bir ya da her iki ventrikülün dilatasyonu ve sistolik disfonksiyonunun görüldüğü durumdur. Hipertansiyon dilate kardiyomyopatinin genç yaşta önemli nedenlerdendir. Conn sendromu nadir görülen primer hiperaldosteronizmdir. Ciddi hipertansiyon, hipernatremi, derin hipokalemi ve metabolik alkalozla ilişkilidir. Conn sendromunda ciddi hipertansiyona bağlı dilate kardiyomiyopati oluşabilmektedir. Bu vakada genç yaşta Conn sondromuna bağlı gelişen dilate kardiyomiyopati olgusu anlatılacaktır.

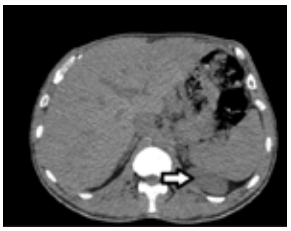
Olgu Sunumu: 31 yaşında erkek hasta nefes darlığı, bacaklarda şişlik ve baş ağrısı ile acil servise başvurdu. Acilde değerlendirilen hastanın tansiyonu 200/110 mmHg ölçüldü. Ortopneik ve takip-

neik olan hastanın bilateral akciğer orta zonlara kadar ralleri mevcuttu. Biyokimyasal değerler Na: 145 mEq/L, K: 2.1 mEq/L, Cl: 101 mEq/L, Ca: 9.3 mg/dl, kreatinin 0.9 mg/dl ölçüldü. Kan gazında pH: 7.50, bikarbonat 30.4 mmol/L ölçüldü. Yapılan transtorasik ekokardiyografide; ejeksiyon fraksiyonu %35, sol ventrikül (SV) diyastol sonu çapı 63 mm, SV sistol sonu çapı 50 mm, interventriküler septum 17 mm, posterior duvar 14 mm ölçüldü (Şekil 1). Orta düzeyde mitral kapak yetersizliği ve hafif düzeyde triküsbit kapak yetersizliği izlendi. Elektrokardiyografide ise sol ventrikül hipertrofi bulguları izlendi. Hasta koroner yoğun bakım takiplerinde kompanse hale geldi. Hastanın genç yaşta ciddi hipertansiyon ile beraber hipernatremi, hipokalemi ve metabolik alkalozunun olması nedeniyle primer hiperaldosteronizm açısından endokrinoloji bölümüne danışıldı. Serum aldosteron/renin oranı ≥%50 hesaplanması nedeniyle yapılan bilgisayarlı tomografide 10x15 mm sol sürrenal kitle görüldü (Şekil 2). Endokrinolojik cerrahlar tarafından 10x10 mm' lik sol sürrenal kitle başarı ile eksize edildi. Patoloji sonucu adrenokortikal adenom ile uyumlu geldi. Hastanın kontrol Na: 142 mEq/L, K: 3.9 mEq/L Cl: 109 mEq/L ölçüldü. Serum aldosteron/renin oranı <20 olarak hesaplandı. Kalp yetersizliği tedavisi düzenlenerek taburcu edilen hastanın takibi devam etmektedir.

Tartışma: Ciddi hipertansiyon dilate kardiyomyopati yapmaktadır. Endokrinolojik hipertansiyon genç yaşta ortaya çıkan hipertansiyonun en önemli bir nedenlerindendir. Derin hipokalemi ile ciddi hipertansiyon durumlarında primer hiperaldosteronizm düşünülmeli endokrinolojik açından değerlendirilmelidir. Conn sendromu nadir de olsa hipertansiyona sekonder dilate kardiyomiyopatiye neden olabileceği akılda tutulmalıdır.



Şekil 1. Transtorasik ekokardiyografi görüntüsü.



Şekil 2. Bilgisayarlı tomografi sol sürrenal kitle.

OPS3-33

An acute coronary syndrome induced by varenicline usage

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A 29-year-old, male patient with typical angina pectoris was admitted to our hospital. An electrocardiogram showed biphasic T wave in anterior precordial leads. There was no risk factors for coronary artery disease apart from smoking. The patient was a heavy smoker for 3 years until he joined the smoking cessation program with varenicline. He took varenicline for last twent-five days. His signs and physical examination were unremarkable. Laboratory analyses showed elevated troponin I (7 ng/ml). Transthoracic echocardiography (TTE) in parasternal short-axis wiev revealed a regional wall motion abnormality in the anterior segments of the left ventricle. The chest pain persisted and the patient was transferred to the coronary angiography unit following 300 mg acetylsalicylic acid, 600 mg clopidogrel, and one puff of sublingual glyceryl trinitrate spray. Coronary angiography showed critical left coronary artery (LAD)-diagonal one (D1) bifurcation lesion (Figure a). The bifurcation lesion,after evaluation of the lesion in the main vessel and side-branch, was a true bifurcation and culotte technique was applied (Figure b,c) and two stents were successfully deployed on bifurcation lesion (Figure d). It was considered that the myocardial infarction was probably secondary to triggered by the release of inflammatory mediators following an allergic insult due to varenicline. Kounis syndrome is a group of symptoms that manifests as unstable vasospastic or nonvasospastic angina and even as acute myocardial infarction. It is caused by inflammatory mediators such as histamine, neutral proteases, arachidonic acid products, platelet activating factor and a variety of cytokines and chemokines released during the activation process. The release of mediators during allergic insults has been incriminated as a cause of coronary artery spasm and/or atheromatous plaque erosion or rupture. Many cases were reported regarding Kounis syndrome caused by varenicline.

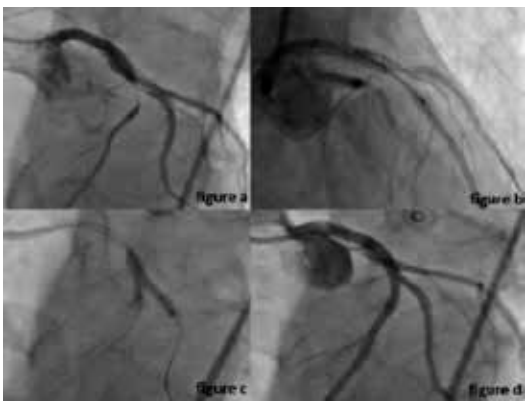

Figure 1. (A) Image shows the true bifurcation lesion on LAD. **(B)** Image shows stent deployment on the main vessel. **(C)** Image shows final kissing percutaneous transluminal coronary angioplasty. **(D)** Image shows the last view of succesfull revascularization of LAD true bifurcation lesion.

OPS3-34

A coronary collateral to lung... How much do we care about? Case report

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64 years old man without known disease was admitted to emergency service with substernal chest pain, diaphoresis, nausea and vomiting. There was no ST segment elevations or depressions on his electrocardiography. As a result of significant increase in troponin values, NSTE-ACS with GRACE score: 95 was diagnosed. Coronary anjiography was done a day after and showed critical lesions at proximal portion of left anterior descending artery (LAD) and circumflex artery (CX) (Figure 1) During right coronary artery viewing; a coronary collateral to left lung and nodular image at the end of that artery was seen. Revascularization procedure haven't done at that time. Thorax CT images showed multiple nodular lesions, the biggest ones were 19 mm in diameters at left lung, 15 mm at right lung and thought as metastasis (Figure 2). Abdominal MR with contrast was taken for primary focus searching and showed 5 cm nodular image with malign character as local extension to perirenal fascia at right kidney, renal cell carsinom was the first diagnosis (Figure 3). He was consulted with urology department and operation was thought as elective surgery. So revascularization procedure done with two bare metal stent, as 3.5/20 mm to LAD and 4/25 mm to CX lesions. Dual antiplatalet therapy for one month as asetylsalicylic asid and klopidogrel was the treatment of choice. After one month without any cardiac complaint, kidney resection operation done safely.

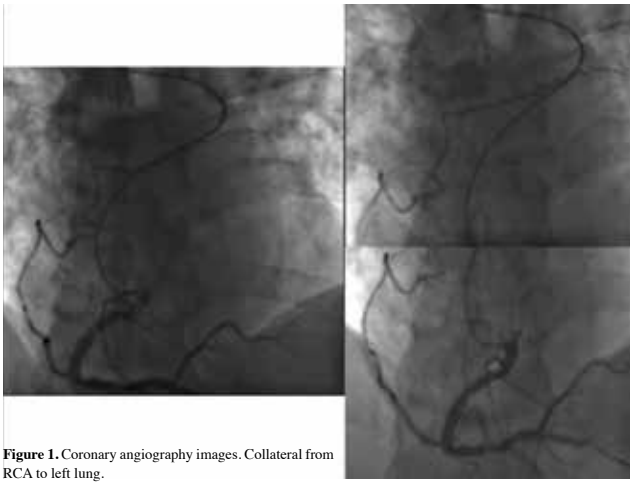

Figure 1. Coronary angiography images. Collateral from RCA to left lung.

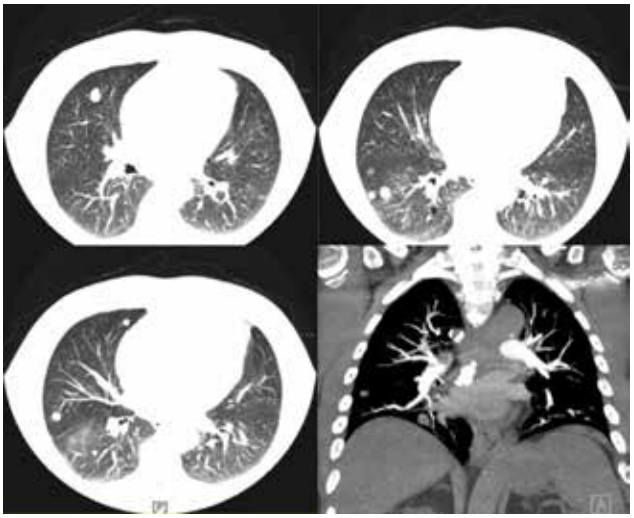

Figure 2. Thorax CT images shows bilateral lung metastasis.

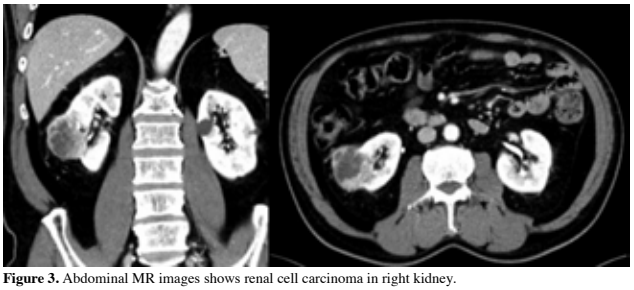

Figure 3. Abdominal MR images shows renal cell carcinoma in right kidney.

OPS3-35

A catastrophical complication during PCI: Iatrogenic LMCA dissection

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İstanbul University Haseki Cardiology Institute, İstanbul

66 year old male with evidence of anterior myocardial ischaemia on myocardial perfusion scintigraphy was referred for coronary angiography, which showed a significant stenosis in the mid-part of the left anterior descending coronary artery (LAD). A 4.0 left Judkins guiding catheter was inserted via 7F right femoral sheath. Engagement of the LMCA was rather difficult due to its superior location but was finally acomplished after some manipulation.Unfortunately the first contrast injection showed an an antegrade dissection, extending from proximal to the distal LMCA (Figure 1). Coronary flow was halted abruptly resulting in hypotension and chest pain. We negotiated a wire in the true lumen of the LMCA and a bare metal stent (Integrity 4.0x12 mm) was subsequently placed from the ostium with minimal protrusion into aorta re-establishing TIMI 3 flow in both the LAD and the circumflex artery (Figure 2). The angiographic result was optimised with non-compliant balloon post-dilatations (Sprinter NC 4.5x15 mm). Meanwhile the patient became completely symptom free and hemodynamically stable so a DES (2.75x20 mm) was implanted to the culprit lesion in mid LAD and postdilated. The patient was transferred to the coronary intensive care unit for further monitoring under medical treatment and discharged two days later in a well overall condition without ECG changes or wall motion abnormalities. Iatrogenic LMCA dissection results from mechanical injury to the arterial wall during catheter manipulation or passage or deployment of an interventional device.Vigorous dye injection from a noncoaxial catheter is another underlying mechanism. Once dissection occurs, rapid deterioration may occur shortly after because of abrupt flow compromise due to progressive dissection or superimposed thrombus formation. Immediate intervention with left main stenting is the treatment of choice but in some cases advancement of the wire into the true lumen may be impossible necessitating emergent by pass surgery.

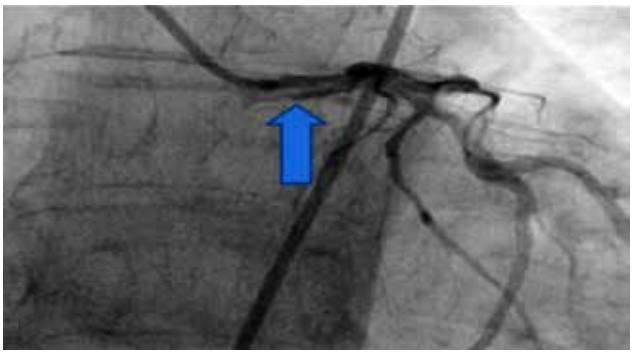

Figure 1. Iatrogenic coronary artery dissection extending from proximal to distal LMCA (blue arrow).

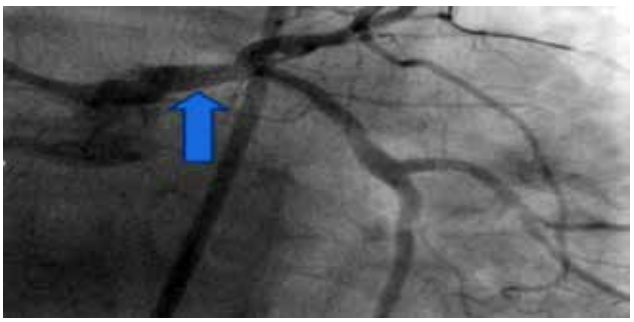

Figure 2. LMCA dissection was limited after stent implantation (blue arrow).

OPS3-36

Acute Inferior myocard enfarctus concomitant complete heart block due to generalise vasospasm

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[2]*Department of Cardiology, Adana Numune Training and Research Hospital Seyhan Application Center, Adana*

The patient was 61 years old women. She admitted to emergency service with retrosternal chest pain. Electrocardiogram (ECG) was shown both ST segment elevation on derviation D2, D3, AVF and complete heart block at emergency service (Figure 1). We were diagnosed acute inferior myocard enfarctus and a-v complete block and immediately taken to coronoray intensive unite (CIU). Coronary angiography (CAG) was performed and determined non-critical occlussion before 1 month, she had hypertansion and smooking priority. Again we performed CAG. We were demonstreated generalise vasospasm in left anterior descedending artery (LAD) and circumflex (CX) artery (Figure 2). There were non-critical occlussions in right coronary artery (RCA) (Figure 3). Vasospasm was disappear after given 2 cc nitroglycerine (Figure 4). We didn't think any intervention because of vasospasm. The patient was taken to CIU and ECG was shown normal (Figure 5). She was excharged after 2 days with isosorbid didnitrate.

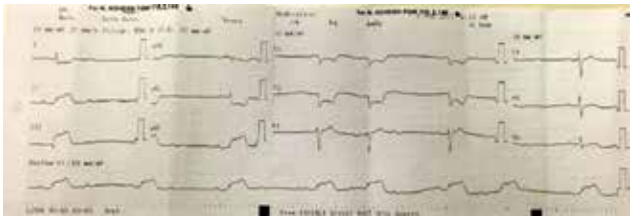

Figure 1. Complete Heart Block on EKG.

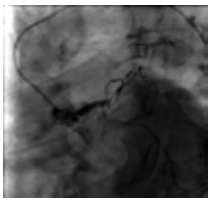

Figure 2. Vasosapsm in LAD and CX.

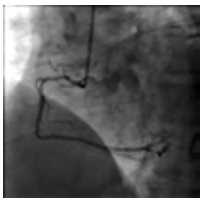

Figure 3. Non-critical occlussion in RCA.

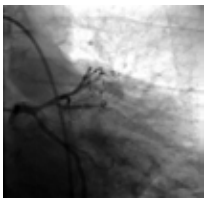

Figure 4. LAD and CX artery after 2 cc nytrogliserine.

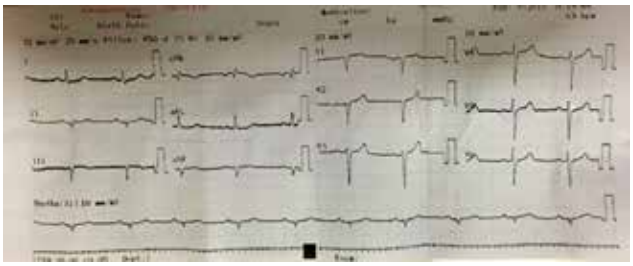

Figure 5. Normal ECG after 2 cc nytrogliserine.

OPS3-37

Single coronary artery; Anomalous origin of the right coronary artery as a branch of the left anterior descending artery

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Case: 55 year- old female known to have diabetes mellitus, hypertension admitted to our clinic with a complaint of chest pain which is lasting for 3 months. Cardiac sounds were normal, The blood pressure was 135/80 and pulse rate was rhythmic and70 beats per minute. Electrocardiogram revealed sinus rhythm. Echocardiogram showed normal ejection fraction (65%) and no valvular disease. She had had a positive stress and was scheduled for cardiac catheterization. Left coronary angiography revealed plaque formation in left anterior descending (LAD) artery and circumflex artery with anomalous right coronary artery (RCA) arising from proximal LAD. Aortagraphy rule out any coronary artery arising from right coronary sinus. The patient was treated medically with acetylsalicylic acid 100 mg, atorvastatin 10mg and ramipril 5mg.

Discussion: Anomalous right coronary artery is a very rare entity with a prevelance of 0.26%. The anomalous origin of RCA usually arises from the left sinus valsalva. An anomalous RCA arising from the LAD artery is very rare. It is usually known as a benign entity but can have clinical importance due to its course between the aorta and pulmonary artery which may cause myocardial ischemia or sudden cardiac death.

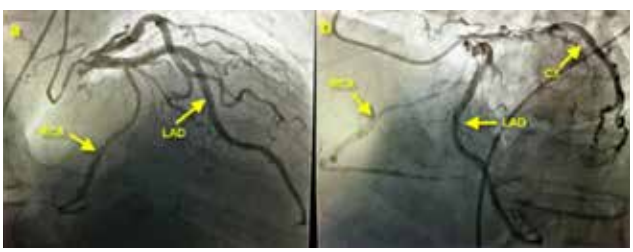

Figure 1. Coronary angiogram showed anomalous origin of right coronary artery arising from left anterior descendin artery (1a: anteroposterior-cranial view 1b: left cranial oblique view).

OPS3-38

Akut inferior miyokard enfarktüsünde RCA-safen grefti anastomoz yeri hizası revaskülarizasyon sonrası gelişen nativ RCA akımının total oklüzyonu sonucu anastomoz yerinden RCA ya retrograd perkütan başarılı girişim uygulaması

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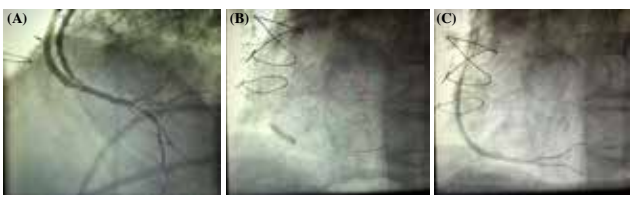
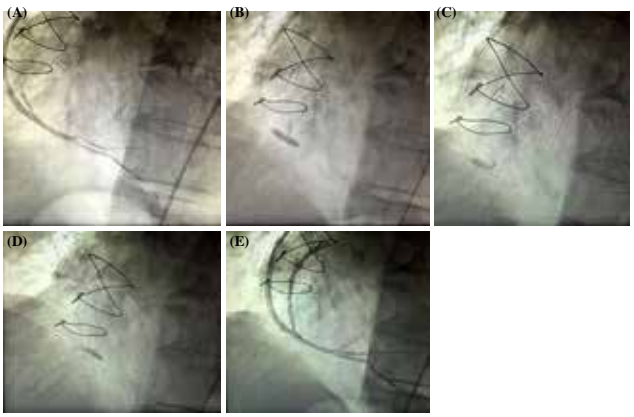
Giriş: Koroner arter hastalığı ön tanısı ile yapılan koroner anjiografilerin % 20 kadarında kronik total oklüzyon izlenir. Kronik total oklüzyonların %60-65 kadarı anterograd yolla %35-40 kadarı ise retrograd yolla revasküarize edilmektedir. Retrograd yol olarak da septal, safen ven ve epikardiyal kollateraller kullanılmaktadır. Bu vakada 80 yaşında erkek hasta akut inferior miyokard enfaktüsü ile acil servise başvurduktan sonra yapılan koroner anjiyografisinde RCA 'ya Ao-safen ile anastomoz yeri tıkanıklığı üzerinden revaskülarizasyon işlemi sonrası retrograd nativ akımın kesilmesi üzerine anastomoz sonrası total oklüzyona retrograd başarılı PTCA işlemi uygulanmıştır.

Olgu Sunumu: Seksen yaşında erkek hasta bilinen koroner arter hastalığı ve kalp yetersizlği olup acile tipik angina şikayeti ile başvurmuştu. Ekokardiyografide EF %35, 2/4 egzantrik mitral yetersizliği ve 2/4 aort yetersizliği izlendi. EKG sinde inferior derivasyonlarda ST elevasyonu izlenen hastaya yapılan koroner anjiografide LMCA plaklı, LAD proksimalde %100 tıkalı, OM2 başında %40 darlık, RCA PDA başında %40-50 darlık izlendi. LİMA-LAD grefti açık, Ao-safen-RCA grefti açık ve anastomoz yerinde %70-80 darlık izlendi. Nativ RCA retrograd RCA safen üzerinden tamamen doluyor idi. Koroner anjiografiyi takiben RCA ostiumuna kılavuz telle oturuldu. Ao-RCA-safen anatomoz yerindeki %70-80 lezyondan kılavuz telle geçildi. Lezyona 3.5x12 mm balon ile PTCA uygulandı. PTCA sonrası lezyona 4,5x12 mm zotarolimus kaplı stent implante edildi. Tam açıklık sağlandı. Stent sonrası nativ RCA' da anastomoz öncesinde total oklüzyon izlendi (Şekil 1a-c). İşleme son verildikten sonra hasta yoğun bakım ünitesine tirofiban ve heparin infüzyonu altında alındı. Hastada VF gelişince hasta defibrile edildikten sonra acilen koroner anjiografiye tekrar alındı. Anastomoz sonrası oklüzyon izlenmesi üzerine hasta tekrar koroner anjiografi işlemine alındı. Ao-safen-RCA crux öncesi %100 tıkalı olduğu izlendi. Ao-safen-RCA grefti crux öncesi %100 lezyondan kılavuz tel ile geçildi. Lezyona 4.0x12 mm balon ve RCA anastomoz hattına 3.0x12 mm balonlar ile kissing PTCA yapıldı. PTCA sonrası nativ RCA anastomoz yerinde %20 darlık izlendi. (Şekil 2a-e). Başarılı PTCA sonrası RCA akımı retrograd sağlandıktan sonra hasta yoğun bakım ünitesine alındı. VT/ VF tekrarı olmadı. Hastaya antikoagülan tedavi aspirin 100 ve klopidogrel 2x75 başlandı. Bu vakamızda safen greftine PTCA+ stent sonrası gelişen nativ RCA akımının ortadan kalkmasını retrograd yolla perkütan koroner işlemle başarılı bir şeklide sonlandırdık.



Şekil 1. (A-C)



Şekil 2. (A-E)

OPS3-39

Coronary intervention with extra back-up catheter to anomalous RCA

Yahya Kemal İçen[1], Gökhan Gözübüyük[1], Mevlüt Koç[2]

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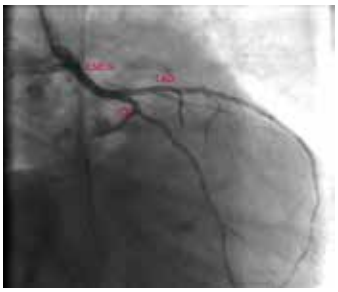
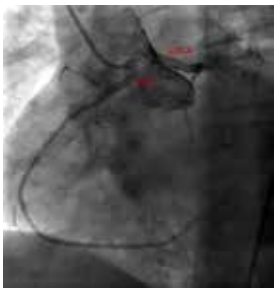
The patient was 61 years old man. He was admitted to emergency service with retrosternal chest pain. His ECG an cardiac markers were normal. He had hypertansion,smooking and performed coronary angiography but didn't known result. He was hospitalized because of ongoing chest pain. We were performed coronary angiography. Left coronary system was shown with left judkins catheter as non-critical occlussions. Right coronary artery (RCA) and left coronary sistem was shown right Judkins catheter as non-selective at the same time. Both systems originated from same sinüs.

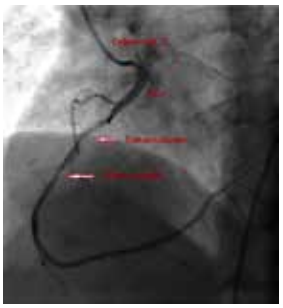
RCA was demonstrated as selective with right amplatzer catheter-2. There were two critical lesion at mid portion RCA. We weren't placed any right side guiding catheter (judkins right and amplatzer right catheter) for intervention. So, we decided to triying left side guiding catheters. We used extra back up catheter (EBU) and placed at RCA. Finally, lesions in RCA were treated succesfully with two stents. The patient was excharged with medical treatment after 1 day.



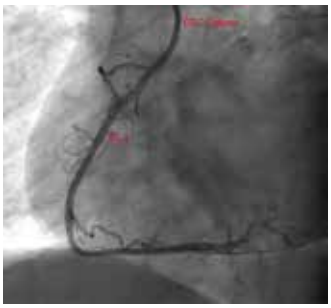
Showing left coronary system with FL4.



Showing both left and right coronary system with FR-4.



Showing right coronary artery with AR-2.



Treated and showing rihgt coronary artery with EBU.

OPS3-40

A rare presentation of acute coronary syndrome:Simultaneous thrombotic occlusion of native right coronary artery and saphenous vein graft

Servet İzci, Emrah Acar, Muhittin Demirel, Mehmet Fatih Yılmaz, Ahmet Güler, Ender Özgün Çakmak, Regayip Zehir, Selçuk Pala

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A 42-year-old-male patient presented to our emergency service with chest pain,shortness of breath and vomiting.He had a history of coronary artery by-pass graft surgery (CABG) five years ago. He had also diabetes mellitus and essential hypertension. ECG showed ST segment elevation in leads II, III and aVF with reciprocal ST depression in leads I and aVL. The patient was diagnosed with acute myocardial infarction. Coronary angiography showed simultaneous thrombotic occlusion of both native right coronary artery (RCA) (Figure 1a) and saphenous vein graft to the obtuse marginal branch of the circumflex artery (OM-SVG) (Figure 2a). We firstly engaged into the RCA ostium with a 7F right guiding catheter. The occlusion was crossed with a floppy wire (Figure 1b). Then we inflated a 2.0x15 mm semi-compliant coronary baloon catheteter for predilatating the lesion. After that a 3.0x38 mm drug eluted stent was deployed for covering the lesion. Then we postdilateted the stent with a 3.5x23 mm non-compliant coronary baloon catheter. The vessel had a TIMI-III flow at last (Figure 1c). After the successfull revascularization of the native RCA, subsequently we engaged with the same guiding catheter into the OM-SVG ostium.Then we crossed the lesion with a wire fixed to distal embolic protection device (EPD) (Figure 2b). EPD was opened on a safe landing zone distal to the occlusion site. We aspirated the thrombus burden with a manuel aspiration thrombectomy catheter. A great part of fresh white thrombus was aspirated (Figure 2c). Then we took an angiographic scene for understanding the extent of the lesion and it showed a very long lesion with large thrombus burden in spite of successfull aspiration (Figure 2d). So we injected an intracoronary tirofiban bolus dose adjusted to the patient's weight. After that we deployed a 3.5x32 mm bare metal stent on the distal part of the lesion, a 3.5x24 mm bare metal stent on the mid part of the lesion and a 4.0x15 mm bare metal stent on the proksimal part of the lesion. After that EPD was retrieved. We lastly took angiographic view showing that distal flow was achieved (Figure 2e). But still it had a bit of thrombus so intravenous tirofiban infusion was decided to administer for a 18 hours. He did not complain any symptom, ST elevation in electrocardiogram was decreased and returned to baseline. Also we did not want to increase the risk of contrast induced nephropathy,so we did not perform a control angiogram after tirofiban infusion. He was dicharged without any complication and he is in the outpatient clinic follow-up regularly.

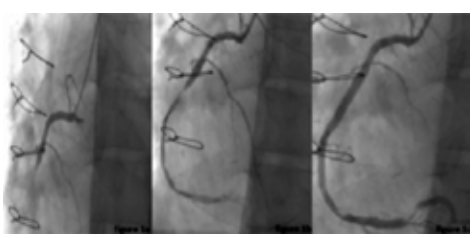

Figure 1. (A) RCA occlusion in the mid segment figure **(B)** The lesion after the wire crossing figure **(C)** The view of the vessel after stent deployment, distal flow was achieved.

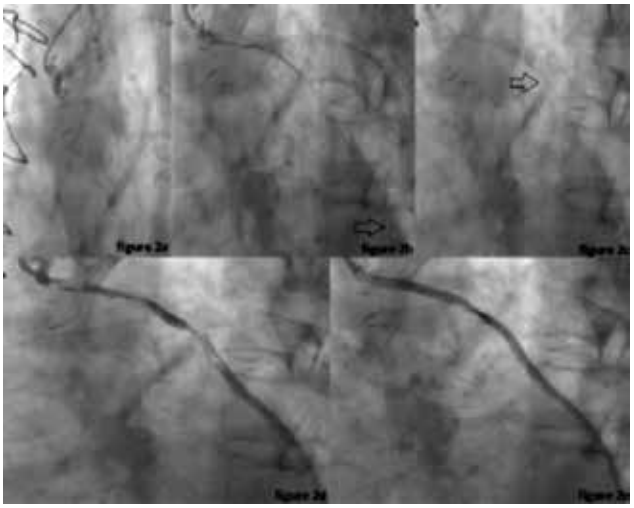

Figure 1. (A) OM-SVG occlusion proximally. **(B)** After the crossing of the lesion with the EPD and openening of the EPD(arrow),there was no flow yet. **(C)** A manuel aspiration catheter(arrow) was introduced in the thrombus and sucked figure. **(D)** After the aspiration there was a long lesion and related great burden of thrombus figure. **(E)** Three apart stents were deployed for the full covering the lesion.In spite of successfull revascularization still a bit of thrombus persisted but distal flow was achieved.

OPS3-41

Nadir bir olgu: Sol koroner sinüsten çıkan tek koroner arter

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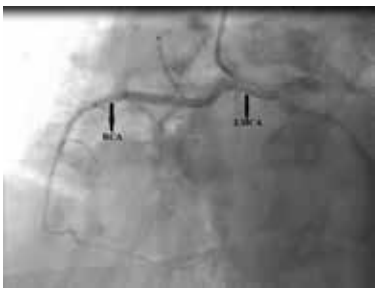
Daha önce bilinen kardiyovasküler hastalığı olmayan 61 yaşında erkek hasta, tipik göğüs ağrısı ile acil serviste değerlendirildi. Risk faktörü olarak hiperlipidemisi mevcuttu. Hastanın hemodinamisi stabil ve kardiyovasküler sistem muayenesi doğaldı. EKG inferior derivasyonlarda T negatifliği olan ve kardiyak biyomakerları negatif olan hasta unstabil angına pektoris ön tanısı ile kardiyoloji bölümüme yatırıldı. Çekilen ekokardiyografide sol ventrikül sistolik fonksiyonları normal ve ejeksiyon fraksiyonu %65 olarak saptandı ve koroner anjiyografi yapıldı. Koroner anjiyografi sonucunda ise sol koroner sinüsten tek ostiyumla sağ koroner arter ve sol ana koroner arterin birlikte çıktığı gözlendi. Sağ koroner arterin ortak kökten çıktıktan sonra normal dağılım bölgesine doğru seyrettiği görüldü (Şekil 1). Sol ana koroner normal, LAD, sirkumfleks ve RCA plaklı olarak saptandı. Koroner arterlerin aortadan çıkış anomalileri konjenital kalp defekti olmayan hastalarda en sık karşılaşılan koroner arter anomalisidir. Ancak sağ ve sol koroner arterin aortadaki tek ostiyumdan çıktığı durumları ifade eden tek koroner arter ise daha az oranda görülmektedir ve sıklığı çeşitli anjiyografik serilerde %0.02-0.04 oranında bildirilmektedir. Bizim vakamız Sharini ve arkadaşlarının tek ostiyumun çıktığı sinüse göre yaptığı sınıflamada tek koroner arter sol koroner sinüsten çıktığı için Tek ostiyum Sol Sinüs Valsava'da şeklinde değerlendirilmiştir. Ayrıca tek koroner arter olguları Lipton ve arkadaşlarının yaptığı sınıflamaya göre anjiyografik olarak 3 gruba ayrılmaktadır. Bizim vakamız bu sınıflamaya göre grup 2 (L II) olarak değerlendirilmiştir ve bu grupta tek koroner arter sol koroner sinüsten çıktıktan sonra sağ koroner arteri verebilmek için geniş bir transvers trunkus halinde kalbin bazalini çaprazlamaktadır. Alt grup sınıflamada ise grup 2-B (L II-B) olarak değerlendirilmiştir ve bu alt grupta tek koroner arter sol koroner sinüsten çıkar ve sağ koroner arter başlangıcı yani transvers trunkus aorta ve pulmoner arter arasında seyreder. (Şekil 1, 2). Tek koroner arter bulunan olgularda klinik özellikler koroner arterin seyri ile ilişkilidir ve literatürde sağ koroner arterin ana pulmoner arter ve aorta arasında seyrettiği L II-B olgularında özellikle egzersizle ilişkili akut miyokart enfarktüsü ve ani ölümler bildirilmiştir. Ayrıca tek koroner arterli olguya anjiyoplasti uygulaması esnasında dikkat edilmesi gereken önemli noktalar bulunmaktadır. Geniş çaplı kateterlerin yarattığı geçici osteal obstrüksiyon, göğüs ağrısı, dispne gibi semptomlara ve hipotansiyon gibi hemodinamik bozulmaya yol açar. Tek koroner arterin ortak kök bölümüne anjiyoplasti ve stent uygulaması kontrendike olarak kabul edilmektedir. Tek koroner arterli olgularda klinisyenlerin bu durumları göz önünde bulundurması gerektiğini düşünmekteyiz.



Şekil 1. Sol anterior oblik 40 görüntülemede sağ koroner arterin ve sol ana koronerin birlikte tek ostiumdan çıktığı izlenmektedir.

Şekil 2. LİPTON sınıflamasına göre grup 2 (L II -B)'nin şematize gösterimi.

Girişimsel kardiyoloji / Koroner

OPS3-42

Anterior MI due to colleteral occlusion

Mustafa Ahmet Huyut, Asım Enhoş, Erdem Karaçöp, Aydın Nadir, Muharrem Nasifov, Nuray Kahraman Ay, Abdülkadir Yıldız, Mehmet Ergelen, Ahmet Bacaksız, Özge Özden Tok, Ömer Göktekin, Hüseyin Uyarel

Department of Cardiology, Bezmialem University Faculty of Medicine, İstanbul

A 65-year-old male admitted to our emergency service with complaint of new onset chest pain and cardiogenic shock. The patient's electrocardiography (ecg) was consistent with acute ST-segment elevated anterior myocardial infarction. Coronary angiography was made immediately. We found the right coronary artery (RCA) mid 80% lesion, circumflex artery(CFX) OM3 100% occlusion and left anterior descending artery (LAD) mid 100% occlusion on the coronary angiography (Figure 1). We decided to open all occluded lesions cause of cardiogenic shock. A 7F EBU 3.5 left guiding catheter was inserted using the transfemoral approach. At first we sent 0.014" BMW guidewire to LAD (Movie 1, 2). We could not passed the LAD lesion. After using Pilot 50 guidewire, we failed to cross the lesion. Then we thought that the lesion was chronic occluded. After then we crossed the CFX OM3 lesion (Video 3-5) and we showed that LAD was filling retrogradely from OM3 branch so patient had an anterior MI due to collateral occlusion. After reconstruction the filling retrogradely to LAD, patient's chest pain was dissolved and ST segment elevations were decreased. During the ten months follow up period, patient has no complaint.

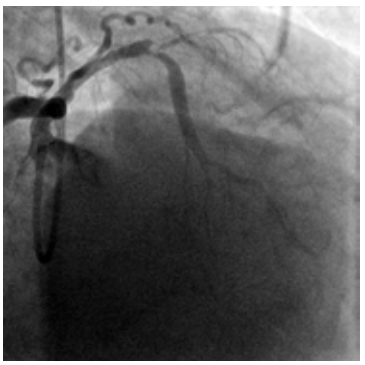


Figure 1.

OPS3-43

Can we use a balloon instead of microcatheter in emergency situation?

Mustafa Ahmet Huyut, Asım Enhoş, Erdem Karaçöp, Aydın Nadir, Maharram Nasifov, Ahmet Bacaksız, Abdülkadir Yıldız, Mehmet Ergelen, Hüseyin Uyarel, Nuray Kahraman Ay, Özge Özden Tok

Department of Cardiology, Bezmialem University Faculty of Medicine, İstanbul

A 55-year-old male admitted to our emergency service with complaint of new onset chest pain and cardiogenic shock. The patient's electrocardiography (ecg) was consistent with acute ST-segment elevated anterior myocardial infarction. Coronary angiography was made immediately. We found the left anterior desending artery 100% occlusion on the coronary angiography. A 7F Judkins left 4 guiding catheter was inserted using the transfemoral approach. At first we sent 0.014" floppy guidewire to LAD (Figure 1, Movie 1). We passed the LAD lesion but it looked like to go subintimal course (Movie 2). Then we tried to pass the lesion with intermediate guidewire for a different path but we failed to cross the lesion (Movie 3). Then we thought to give contrast with a microcatheter but at that moment we could not reach a microcatheter. So we use a balloon instead of microcatheter in emergency situation. We strached the balloon and gave contrast via balloon's second lumen to visualise coronary anatomy and to be sure about being in the true lumen. And balloon showed us that we were in the true lumen (Movie 4). After then we crossed the lesion with 2.0x12 mm balloon for predilatation then insert 3.0x38 mm drug eluting stent to the lesion (Movie 5). Then we used 3.5x15 mm non-compliant balloon for post dilatation and restored TIMI III flow (Movie 6). After the operation ST segment elevations were decreased and we weaned the patient from ventilator 24 hours later. During the one year follow up period, patient has no complaint. In that case we would like to show a way for useing a balloon instead of microcatheter in emergency situation.

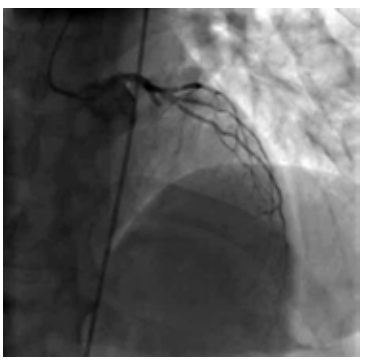


Figure 1.

OPS4-01

An unusual handicap of percutaneous mitral balloon valvuloplasty: Iatrogenic mitral cleft

Edibe Betül Börklü, Muhammed Keskin, Şennur Ünal Dayı, Ahmet Ekmekçi

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Percutaneous mitral balloon valvuloplasty (PMBV) is the primary treatment in rheumatic mitral stenosis (MS) with favorable mitral valve morphology which means echocardiographic score of 8. A 50-year-old female patient came to our clinics with progressive dyspnea for last months. She had been following with the diagnosis of rheumatic MS for 30 years and PMBV had been performed 20 years ago. She had atrial fibrillation on admission. Transthoracic echocardiography (TTE) revealed ejection fruction of 60% by Simpson's method. Mild mitral regurgitation and severe MS with favourable characteristics for PMBV were seen. Mitral valve area (MVA) was calculated 1.1 cm^2 by planimetry and echocardiographic score (Wilkin's score) was found as 4. Left atrial trombus was excluded by performing transesophageal echocardiography (TEE). PMBV was performed by using transseptal technique with Inoue balloon. MVA was calculated 1.68 cm^2 and moderate mitral regurgitation was detected on control TTE after procedure. On the other hand a cleft on basal part of anterior leaflet associated with the procedure was noticed. Images of the cleft that was not seen on pre-procedural echocardiogrphic examinations were obtained by TEE with 3D technique. Although PMBV is a safe procedure with high success rate, there are some complications associated to PMBV. When looking to literature, complications about leaflet are leaflet laceration or rupture of chordae. On the other hand iatrogenic mitral cleft has not been reported in literature till now. Therefore we want to report this unusual complication. PMBV should be performed in qualified cardiac catheterisation labs with a backup facility of cardiac surgery to deal with any potential life-threatening complications. However all complications do not require surgery and can be simply followed up. Because of the cleft mentioned in this case didn't cause any symptom and hemodinamic instability, we agreed with conservative strategy for patient who is still asymptomatic after a follow-up period of 2 years.

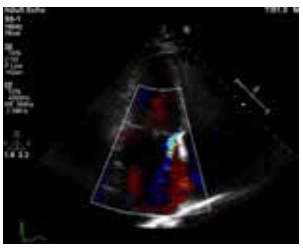

Figure 1. 2D transthoracic echocardiogram shows iatrogenic mitral cleft at the basal part of anterior leaflet.

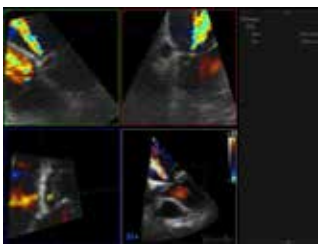

Figure 2. 2D and 3D Transesophegeal echocardiograms show iatrogenic mitral cleft and regurgitant flow.

OPS4-02

Early LOTUS transcatheter aortic valve thrombosis

Gökhan Ertaş, Hatice Betül Erer, Mehmet Eren

Department of Cardiology, Dr. Siyami Ersek Chest, Cardiovascular Surgery Training and Research Hospital, İstanbul

Little is known about the valve thrombosis following transcatheter aortic valve replacement. In this report, we presented the first Lotus valve thrombosis case in the literature.A 86 years old female patient was referred to our hospital due to severe aortic stenosis. Patient was suffering from dyspnea and chest pain. Transthoracic echocardiography (TTE) revealed a concentric hypertrophic left ventricle with preserved left ventricular ejection fraction (EF 55%) and a severely calcified aortic valve with a maximum pressure gradient (PG) of 77 mm Hg and a mean PG of 47 mm Hg. Aortic valve area (AVA) indexed by body surface area was 0.6 cm^2/m^2. Preprocedural coronary angiography revealed nonstenotic coronary atherosclerotic plaque. The EuroSCORE II was 2.15% and the STS score for mortality was 1.11%. The heart team decided to perform transcatheter aortic valve implantation (TAVI). Patient had successful implantation of a 23 mm Lotus valve bioprosthesis (Boston Scientific, Natick, Massachusetts). After an uneventful postoperative period, the patient was discharged on day 3 on aspirin and clopidogrel and advised to stop the clopidogrel after 3 months. Discharge TTE showed normal function of the aortic valve prosthesis with a mean PG of 15 mm Hg and no paravalvular regurgitation. Two months after TAVI the patient suddenly became symptomatic, with dyspnoea (New York Heart Association [NYHA] functional class III). TTE revealed reduced aortic-valve leaflet motion, a mean PG of 56 mm Hg, and an AVA (2D planimetry) of 0.7 cm^2 These findings were confirmed by a transoesophageal echocardiography (TEE). 3D TEE revealed huge obstructive thrombus attached to the valve (Figure 1). Warfarin therapy was started to achieve a target INR of 2.0 to 3.0. After 40 days, patient was totally asymptomatic and control TTE revealed normal function of the aortic valve prosthesis with a mean PG of 13 mm Hg. The optimal duration of anticoagulant therapy after valve thrombosis is unknown. Direct visualization of thrombus is a highly specific finding but it is not seen all cases. Valve thrombosis should be suspected if elevated transvalvular gradients are obtained. Also, computed tomography imaging and transesophageal echocardiography may be useful to support the diagnosis of thrombosis, however if direct visualization of thrombus could not be seen, reduced bioprosthetic aortic valve leaflet motion may be a sign of thrombus. In summary, consider valve thrombosis in case of clincial symptoms and high doppler gradient, even if thrombus could not be seen. A trial of a warfarin therapy should be considered before a valve-in-valve procedure.

OPS4-03

Simultaneously successful treatment of severe mitral regurgitation with CARILLON Mitral Contour transcatheter indirect mitral valve annuloplasty system and left anterior descending artery stenting

Abdullah Nabi Aslan[1], Telat Keleş[2], Hüseyin Ayhan[2], Hacı Ahmet Kasapkara[2], Serdal Baştuğ[1], Esma Arslan[2], Hakan Süygün[2], Murat Akçay[2], Tahir Durmaz[2], Engin Bozkurt[2]

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A 51 year-old male patient with NYHA class III dyspnea was taken to the catheterisation laboratory to perform mitral valve annuloplasty with CARILLON Mitral Contour transcatheter indirect mitral valve annuloplasty system. At first, to determine the course of coronary sinus and the status of coronary arteries, coronary angiogram was performed. It revealed a severe stenosis just before and within the stent that was present in LAD artery (Fig. 1a), patent stent in Cx artery and normal right coronary artery. After that, venous access is obtained with a 9 Fr sheath in the right internal jugular vein. The coronary sinus is engaged and the 9 Fr curved delivery catheter is inserted up to the anterior interventricular vein branch of the great cardiac vein. A 5F marker pigtail catheter with a hand-made cut edge is inserted into the delivery catheter and venography is performed (Fig. 2a). Coronary arteriography is also performed to assess the relationship between the Cx artery and the coronary sinus/ great cardiac vein. The fluoro screen was marked for the desired placement of the anchors. The marker catheter was removed and 13x20x70 mm device was inserted and advanced without fluoro to near the end of the delivery catheter, using the first control knob. Under fluoro (moving image 1), the distal anchor was unsheathed, while pulling gently back on the delivery catheter, in order to position the distal anchor precisely, based upon the mark on the fluoro screen. Then, the proximal anchor was released by first continuing to unsheath as before with the first control knob, then pushing out and locking the anchor using the second control knob, which was attached to a pusher catheter inside the system (Fig. 2b). Left (Fig. 2c) and right (Fig. 2d) coronary angiography were done to ensure patency of the vessels and the device was released. The total duration of the procedure was 23 minutes. Therefore, the percutaneous coronary intervention of LAD artery was intended. After balloon angioplasty of instent and pre-stent stenosis a 2.75x28 mm Xience stent (drug eluting stent) that was overlapped with distal stent was successfully deployed (Fig. 1b). The total amount of contrast material was 80 mL and there was not any renal impairment (based on the estimated glomerular filtration rate) during the hospitalisation period of the patient. After 2 days of follow-up, the patient was discharged with mild degree of MR Compared to baseline, a significant improvement in MR has been observed.

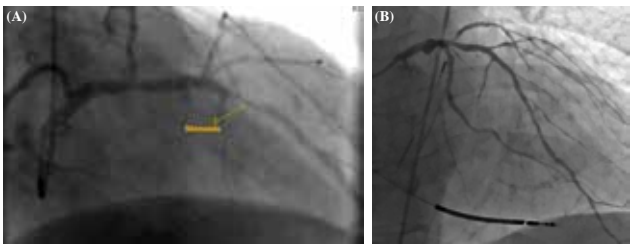

Figure 1. (A) Coronary angiography right cranial view showing pre- and instent severe stenosis at left anterior descending artery. **(B)** Coronary angiography antero-posterior cranial view showing relieved stenosis in the left anterior descending artery.

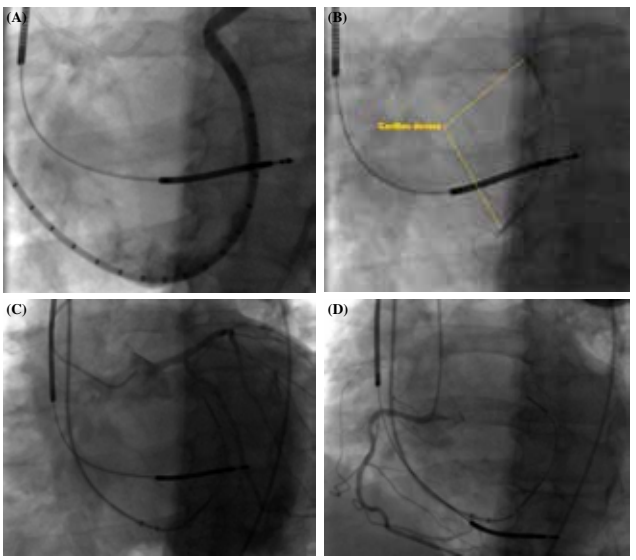

Figure 2. (A) Flouroscopic view of a 5F marker pigtail catheter with a hand-made cut edge that was inserted into the delivery catheter to measure the size of the Carillon device. **(B)** Flouroscopic view of a successfully deployed Carillon device. **(C)** Left coronary angiogram showing patency of circumflex artery after the insertion of Carillon device. **(D)** Right coronary angiogram showing patency of right coronary artery after the insertion of Carillon device.

OPS4-04

Aynı seansta tek balonla mitral ve trikuspit darlığına müdahale

Oğuzhan Yıldırım, Halil İbrahim Erdoğan, Kurtuluş Özdemir

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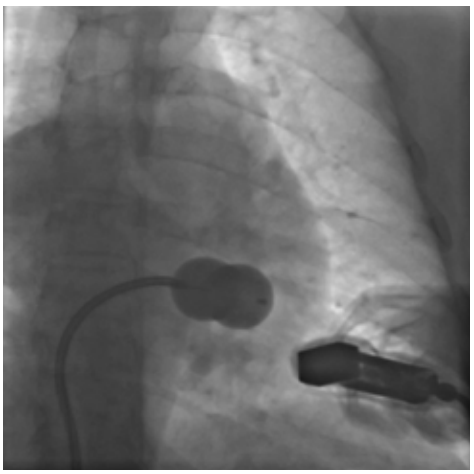
Elli iki yaşında bayan hasta nefes darlığı şikayeti ile değerlendirildi. Hastanın bilinen diyabeti, hipertansiyonu ve astım hikayesi mevcut. Hastanın ekg si sinüs rittmi kalp hızı 100/dk fizik muayene: akciğer: ral+ ronkus-kalp:s1+s2+ 2/6 diyastolik üfürüm mevcut hastanın yapılan ekosunda: mitral kapaklar fibrotik açılımı kısıtlı. Mitral kapağa ait max gr: 40 mmHg mean gr 20 mmHg olarak tespit edildi. Mitral kapak skoru 8-9, kapak alanı 1 cm 2, ciddi mitral darlığı tespit edildi. Triküspit kapak fibrotik açılımı kısıtlı. Triküspit kapağa ait max gr 15 mean mmHg mean gr 9 mmHg olarak tespit edildi. Hastada mevcut bulgularla mitral ve triküspit valvuloplasti yapılması planlandı. Hastanın öncelikle yapılan koroner anjiyosunda önemli patoloji tespit edilmedi.hasta öncelikle mitral kapağa balon valvulaplasti yapıldı. Takibinde mitral kapağa müdahale edilen balon ile triküspit kapağa valvuplasti yapıldı. Mitral kapak maksimum gradienti 40 mmHg'den 11 mmHg'ye, triküspit kapak maksimum gradienti 15 mmHg'den 9 mmHg'ye, sol atriyum basıncı, 24 mmHg'den 10 mmHg'ye, sağ atriyum basıncı 15 mmHg'den 4 mmHg'ye düştü, önemli kapak yetersizlikleri görülmedi, Romatizmal mitral darlığı ve triküspit darlığının beraber görüldüğü vakalar az görülmektedir. Literatürde mitral ve triküspit kapak darlıklarının beraber görüldüğü vakalar mevcut olup her iki kapağa aynı seansta ve aynı balonla müdahale edilmiştir. Kliniğimizde bu özellikte vaka ilk defa yapılmıştır.



Şekil 1. Mitral balon valvuloplasti.

OPS4-05

TAVİ sonrası nadir komplikasyon; Pace leadine bağlı perikardiyal tamponad oluşan sonrasında sağ ventrikül kollaps ve hastanın ölümüne yol açan, perikard içi dev thrombus olgusu

Ömer Şatıroğlu, Murtaza Emre Durakoğlugil, Hakan Duman, Turan Erdoğan, Yüksel Çiçek, Mustafa Çetin

Recep Tayyip Erdoğan Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Rize

Seksen yedi yaşında bayan hasta, nefes darlığı yakınması ile kliniğimize başvurdu, dejeneratif, kalsifik ileri aort darlığı tanısı konuldu. Kliniğimizde kalp takımı tarafından, açık aort kapak operasyonu yüksek riskli kabul edilip TAVİ (Transkateter Yöntemle Aort Kapak Değiştirilmesi) yapılmasına karar verildi. Hastaya 26 mm Edwards SAPIEN XT kapak, transkatater yöntemle, hafif sedasyonla anestezi takibinde, başarılı bir şekilde implante edildi (Video 1, 2). Hastanın bilinci açılmış ve hemodinamik durumu stabil seyrederken, 10 dakika sonra, aniden hipotansiyon, taşikardi ve şuur bulanıklığı gelişti. Acil yatakbaşı ekokardiyografik incelemede, sağ atriyum ve ventrikülde kollaps bulgusu yapan, kalp tamponadına yol açan 1,5-2 cm, perikardiyal efüzyon saptandı (Video 3, 4). Hastada hızlı hıpotansiyon oluşması ve kardiyak kollaps gelişeceği düşünülerek hasta entübe edilip acil perikardiyosentez yapıldı ve 60 cc kanlı mayi çekildi. Hastanın tansiyonunda yükselme ve bilinçte açılma sağlandı, perikard içine kanama devam edeceği düşünülerek ve sağ atriyum lokalizasyonunda efüzyonun devam etmesi sebebiyle kalp damar cerrahi bölümü ile tekrar konsülte edilerek açık cerrahi ve perikardiyal drenaj tüpü takıldı. Açık cerrahi sırasında sağ ventrikül komşuluğunda perikard içinde hematom görüldü ancak aktif kanama izlenmedi. Perikardiyal tamponadın, sağ ventrikül pace leadine bağlı delinme ve perikard içine kanama komplikasyonu olduğu düşünüldü. Hasta operasyon sonrası hemadinamik olarak stabil seyretti, 12. saatte ekstübe edildi, bilinci yerine geldi. Hasta takibinin 5. gününde, hastada aniden hipotansiyon ve kardiyak arrest gelişti. Hasta resüstasyonla döndürülüp entübe edildi ancak hipotansiyonu devam etmekteydi. Yatakbaşı ekokardiyografik incelemede, belirgin bir perikardiyal sıvı görülmemesine rağmen, sağ ventrikül çıkış yolunda, dış (perikarttan) basıya bağlı kollaps görüldü. Kalp damar cerrahi uzmanı ile birlikte hasta değerlendirildi. Hipotansiyonu açıklayacak diğer sebepler ekokardiyografi ve kan tetkikleri ile araştırıldı. Aktif kanama, emboli, protez aort kapak disfonksiyonu, miyokard iskemisi gibi diğer durumlar ekarte edildi. Perikardiyal tüpün tıkandığı düşünülerek tekrar acil cerrahiye alınan hastada, sağ ventrikül komşuluğunda sağ ventriküle bası yapan dev trombüs izlendi. Ancak acil koşullarda ikinci kez cerrahi girişim yapılan hasta kardiyovasküler kollaps sonrası ex oldu. Perikardiyal tamponada yol açan perikard içi büyük trombüsler, perikard içi kanama sonrası gelişebilir, klasik perikardiyal efüzyon-tamponad gibi perikardiyal tüple drenajı ve tedavisi mümkün olmayabilir. Özellikle TAVİ yapılan hastalar, ileri yaş ve yüksek riskli olmaları göz önünde bulundurulursa, bu durum ölüme yol açabilmektedir. Perikard içi kanama ve sonrası trombüs geliştiğinde, ekokardiyografik görüntülemede, trombüs ekojenitesi, miyokard ve perikard ekojenitesine benzemekte, perikardiyal sıvı gibi kesin tanı konulamayabilir.

OPS4-06

A centenarian transcatheter aortic valve implantation case and her four-year follow-up

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A 102-year-old female patient with progressive dyspnea and chronic obstructive pulmonary disease (COPD) was consulted to our department due to development of acute palpitation. Electrocardiogram revealed atrial fibrillation with a rate of 145 beats per minute (bpm). Transthoracic echocardiography (TTE) revealed left ventricular (LV) global hypokinesia with an ejection fraction of 40%, concentric LV hypertrophy, severe AS (mean gradient 43 mmHg, aortic valve area 0.5 cm2) (Fig. 1a), moderate to severe tricuspid regurgitation (TR) and severe pulmonary hypertension (PHT) (systolic pulmonary artery pressure (sPAP): 60 mmHg). The patient was admitted to our coronary intensive care unit. The other co-morbid illnessess at this time included severe COPD (FEV1/FVC: 40%) and chronic renal failure stage 3 with an estimated glomerular filtration rate of 46 ml/min/1.73m^2. Coronary angiography demonstrated diffuse calcification of coronary arteries without significant stenosis. The patient's calculated logistic EuroSCORE was 79% and Society of Thoracic Surgeons (STS) score was 14 %. Her Duke Activity Status Index (DASI) was 1.75 due to the severe respiratory insufficiency. We have learned from the relatives of the patient that the patient had a good cognitive function and living without any care assistant until one month ago. The heart team evaluated the patient and decided to proceed with TAVI based on high surgical risk scores and patient's co-morbid illnesses. For an accurate measurement of annular dimension and evaluation of aortic valve anatomy, transesophageal echocardiography was performed and determined the annulus diameter as 21 mm (Fig. 1b). Then, the patient was taken to the catheterization laboratory and successful deployment of a 23 mm Edwards Sapien XT valve (Edwards Lifesciences, Irvine, CA, USA) was performed under general anesthesia using a transfemoral approach (Fig. 1c). This led to a marked improvement of the transvalvular gradient with only mild paravalvular AR. Thereafter, the patient was taken to the coronary intensive care unit and extubated 12 hours after the procedure. Before the hospital discharge, echocardiography showed functional aortic bioprosthetic valve (mean gradient 18 mmHg and aortic valve area 1.7 cm^2), mild paravalvular AR, moderate tricuspid regurgitation, moderate PHT (sPAP: 50 mmHg) and near normal ejection fraction (50%). Electrocardiogram revealed normal sinus rhythm with a ventricular rate of 80 bpm. Atrial fibrillation was also detected to be resolved after TAVI. She has stayed in the coronary intensive care unit service for three days, then taken to the cardiology service. After follow up for a total of 13 days she was discharged without any complications. She has been followed for 4 years clinically and with TTE (Table 1), and now her DASI is 15.45. She lives with her daughter and need no assistance in her daily life.

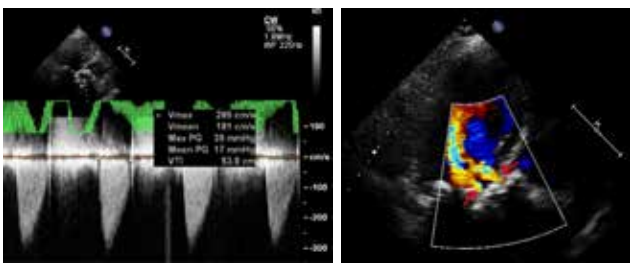

Figure 1. (D) Follow-up transthoracic Doppler echocardiography at 4 years follow-up with 5-chamber continuous wave Doppler analysis showing a mean gradient of 17 mmHg. **(E)** Mild bilateral paravalvular aortic regurgitation (arrows) at parasternal long axis view.

Table 1. The pre-procedural, post-procedural and follow up echocardiographic parameters

Parameters			Time of echocardiography months/years				
	Pre-TAVI	Post-TAVI	10/2012	2013	2014	2015	03/2016
AVA (cm2)	0.5	1.7	1.8	1.7	1.8	1.7	1.7
LV/Ao peak gr (mmHg)	74	35	36	35	36	37	35
LV/Ao mean gr (mmHg)	43	18	18	17	17	18	17
AR (grade)	1	1	1	1	1	1	1
MR (grade)	2	1	1	1	Trivial	Trivial	Trivial
TR (grade)	3-4	2	2	2	1	1	1
sPAP (mmHg)	60	50	50	45	40	40	40
LVEF (%)	40	50	55	60	65	65	65
LVEDD (cm)	4.5	4.4	4.4	4.2	4.2	4.2	4.1
LVESD (cm)	2.8	2.7	2.7	2.4	2.4	2.4	2.5
IVSDD (cm)	1.4	1.4	1.4	1.4	1.3	1.3	1.3
PWDD (cm)	1.3	1.3	1.2	1.2	1.2	1.2	1.2

Ao: aorta; AR: aortic regurgitation; AVA: aortic valve area (calculated from continuity equation); gr: gradient, IVSDD: Interventricular septum diastolic diameter; LV: Left ventricular; LVEDD: Left ventricular end-diastolic diameter; LVEF: Left ventricular ejection fraction (as measured by Biplane Simpson's method); LVESD: Left ventricular end-systolic diameter; MR: Mitral regurgitation; PWDD: Posterior wall diastolic diameter, sPAP: systolic pulmonary artery pressure, TAVI: transcatheter aortic valve implantation, TR: tricuspid regurgitation.

OPS4-07

An incidentally detected acute pulmonary embolism in a nonagenarian severe aortic stenosis case before undergoing transsubclavian aortic valve implantation

Abdullah Nabi Aslan[1], Hüseyin Ayhan[2], Serdal Baştuğ[1], Hacı Ahmet Kasapkara[2], Esma Arslan[2], Murat Can Güney[2], Hasan Biçer[2], Telat Keleş[2], Tahir Durmaz[2], Engin Bozkurt[2]

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A 90-year-old cachectic (body mass index 17 kg/m^2) female presented to our hospital with the complaint of exertional retrosternal pressure sensation, severe dyspnea (NYHA IV) and frequent episodes of presyncope. At physical examination, there was a 3/6 grade systolic ejection murmur spreading to both carotid arteries and maximally heard at the aortic focus. Electrocardiogram was unremarkable with a normal sinus rhythm and 102 beats per minute. Her past history was clear apart from hypertension. The echocardiogram revealed a severe calcified degenerative aortic valve stenosis (AS) with an aortic orifice area of 0.6 mm^2, a mean gradient of 67 mmHg, a moderate mitral stenosis and moderate regurgitation, a left ventricular ejection fraction of 65%, and an estimated systolic pulmonary arterial pressure of 40 mm Hg. The Society of Thoracic Surgeons (STS) score 31.4% and The Euroscore II was calculated to be 24.5% and the patient was consequently deemed a high-risk surgical candidate. Based on a heart team discussion, we decided to proceed with a TAVI. An angiographic study revealed near-normal coronary arteries with an only a 40% stenosis at the proximal of right coronary artery. Multi-detector computed tomography (MDCT) angiography visualized calcifications and severe stenosis of both iliac and femoral arteries precluding a transfemoral or iliac approach. However, a careful examination of the images also showed bilateral subsegmentary pulmonary artery embolism (Fig. 1). Then, the patient was consulted to the department of the chest diseases and transferred to their clinic with the aim of treatment. They scheduled rivaroxaban 20 mg/day and after the two-week of follow-up, the patient again transferred to our clinic for TAVI. Due to the severe ilio-femoral arteriopathy, TAVI with Lotus valve systems was successfully performed via the left subclavian artery under general anesthesia (Fig. 2). Post-procedural echocardiography revealed functional aortic bioprosthesis with a mean gradient of 8 mmHg and aortic valve area of 2.4 cm^2. After 10-day follow-up, the patient was discharged from the hospital without any complication.

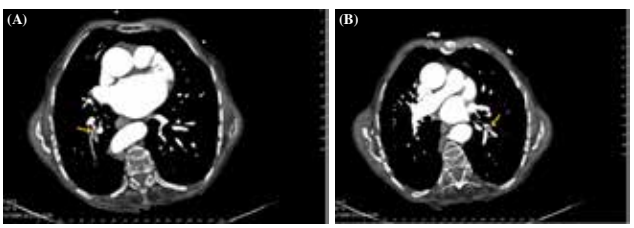

Figure 1. (A) Multi-detector computed tomography image showing subsegmentary embolism at the right pulmonary artery (arrow). **(B)** Multi-detector computed tomography image showing subsegmentary embolism at the left pulmonary artery (arrow).

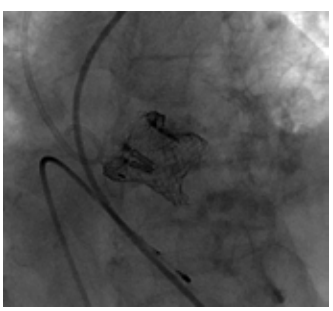

Figure 2. Flouroscopic image of a successfully implanted Lotus valve through the left subclavian artery.

OPS4-08

Treatment of brachiocephalic vein stenosis

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65 years old man was admitted our clinic because of ineffective hemodialysis. From his past history we learned that he was taking hemodialysis for two years but last two months hemodialysis could not be effective because of the low flow. After patients examination due to the weak fistule improvement, we decided to make venography and found 90 percent brachiocephalic vein stenosis (Figure 1). After diagnose we determined to put stent to the lesion. At first we prepared the lesion with 6x40 mm balloon expansion (Figure 2) then 8x60 mm balloon expansion (Figure 3). For final treatment we insert 10x60 mm Assurant cobalt stent to the lesion (Figure 4, 5). After six month follow up patient has no complaint and hemodialysis treatment goes on without any problem. We would like to demonstrate a brachiocephalic vein stenosis case and its treatment.

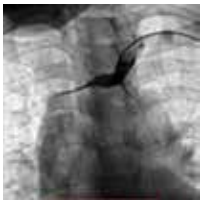

Figure 1.

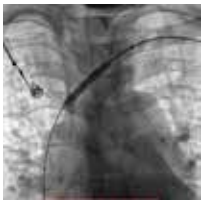

Figure 2.

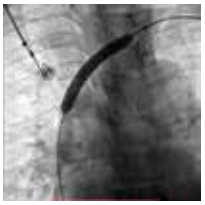

Figure 3.

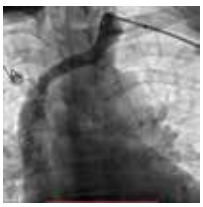

Figure 4.

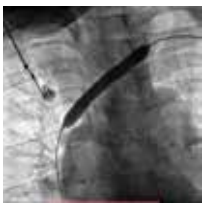

Figure 5.

OPS4-09

An alternative endovascular approach for occluded arteriovenous fistula

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[2]Department of Cardiovascular Surgery, Bezmialem University Faculty of Medicine, İstanbul

Introduction: Maintaining sufficient blood flow through the vascular access is necessary for adequate dialysis. Dysfunction of arteriovenous fistulas and grafts contribute significantly to morbidity and hospitalization in dialysis population. When access flow compromised, angiographic evaluation is electively done to detect access stenosis that can be remediated with percutaneous transluminal angioplasty and/or intravascular stenting.. This report describes a percutaneous transluminal angioplasty in a hemodialysis patient with vascular access failure.

Case Report: A 71 year old woman who had hemodialysis for chronic renal failure for 14 years was consultated to interventional cardiology department for occluded hemodialysis fistula.Her medical history included chronic renal failure and coronary artery disease. Electrocardiography showed normal sinus rhytm, heart rate:60/minute, negative T waves on precordial leads, increased magnitude of QRS on V4-V6. Echocardiography revealed left ventricular hypertrophy, ef: 64%, diastolic dysfunction, moderate mitral regurgitation, mild aortic and tricuspid regurgitation. There was stenosis of the juxta-arterial anastomosis segment in upper extremity angiography. (Figure 1) Predilatation with 2.0x20 mm and 4.0x20 mm balloons were performed. 4.5x18 mm bare metal coronary stent was successfully implanted (Figure 2). Finally improved blood flow through occlusion was observed (Figure 3).

Discussion: Hemodialysis is a life saving procedure in end stage renal failure patients. Surgically created hemodialysis fistulas have high rates of primary failure (23-46%). Sustainable vascular acces with high blood flow is very essential. Acces dysfunction should be promptly identified before the acces and life threatening complications. Endovascular treatment gives promising results for salvage of fistulas. It does not eliminate the need for additional vascular surgery, succes rate is high, complication rate is low. Longterm patency is promising.

Conclusion: Endovascular treatment for occluded arteriovenous fistula is an alternative treatment option for occluded arteriovenous fistula.

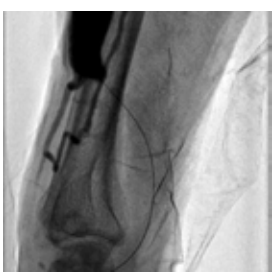

Figure 1. Arteriovenous fistula occlusion was noted.

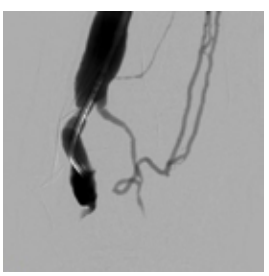

Figure 2. Improved blood flow.

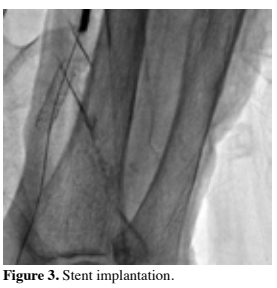

Figure 3. Stent implantation.

OPS4-10

Kronik total tıkalı sol subklavian arterin retrograde teknikle perkütan balon ve stentle açılması

Ömer Şatıroğlu, Murtaza Emre Durakoğlugil, Hakan Duman, Turan Erdoğan, Yüksel Çiçek, Mustafa Çetin

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Elli sekiz yaşında erkek hasta, daha önceden aterosklerotik koroner arter hastalığı, koroner stent ve sigara içme öyküsü mevcuttu. Aynı zamanda geçmeyen sol kolda ağrı, güçsüzlük şikayetleri olması üzerine kliniğimize başvurdu. Yapılan fizik muayenede, sol brakiyal ve radiyal nabızları alınmıyor ve sağ kol kan basıncı, sol kol kan basıncından yüksek saptanmış. Sol kol arteryel tıkanıklık şüphesiyle, üst ekstremite arteryel dopler ultrasonografik tetkik yapıldı. Tetkik sonucunda sol subklaviyan arter düzeyinde tıkanıklık bulguları saptandı. Üst ekstremite arteryel anjiyografi yapıldı ve sol subklavian arter proksimalde tam tıkanıklık görüldü aynı seansta koroner anjiyografi ile koroner arterlerde ve stentlerde herhangibir ciddi darlık saptanmadı. Ayrıca brakiyal arter ponksiyonu ile retrograd olarak subklaviyan arter tıkanıklık düzeyi ve mesafesi görüntülendi (Video 1). Hastanın şikayetlerinin mevcudiyeti ve cerrahi risk yüksek saptanması sebebiyle, kronik tam tıkalı sol subklavian arter perkütan yolla, balon ve stentle açılmasına karar verildi. Tam tıkalı segmentin geçmenin daha kolay olacağı ve işlem sırasında antegrad yoldan kronik tam tıkalı segmenti geçmenin zorluğu, gerçek lümene düşme, ilerleme güçlüğü, diseksiyon gelişmesi ve yayılması gibi risklerin fazla olacağı düşünülerek, retrograd yoldan total okluzyonu açma kararı alındı. Sol brakiyal arterden ponksiyon yapıldı ve arteryel kılıf takıldı, sağ judgins kılavuz kateterle sol subklaviyan arter tıkanıklığın distaline ilerletildi. Asahi fielder xt, 0.14 guide wire ile total okluzyon geçilemedi, diğer total okluzyon tel (Conquest pro 12, 0.14 guide wire) ile, 1.25-15 total okluzyon balon desteğiyle tam tıkalı segment distalden proksimale doğru geçildi ve sol subklavian arter proksimalinde gerçek lümene düşüldü. Lezyon aşamalı olarak önce, 1.25-15, ardından 2.5-20 ve 4.0-20 balonlar ile dilate edildi (Video 2, 3). Guide wire, 0,35 hidrofilik guide wire ile değiştirildi (Video 4). Lezyona 8.0-59 balon ekspandabıl periferal stent (Abott vascular Omnilink Elite stent) implante edildi (Video 5), stentin proksimali, 10.0-40 balon ile postdilate edildi (Video 6). Kronik tam tıkalı sol subklaviyan arterde, tam açıklık ve akım sağlandı, komplikasyon olmadı (Video 7). Hastanın sol kol ile ilgili şikayetleri kayboldu, sağ kol ile sol kol arasındaki kan basıncı farkı ortadan kayboldu. Sol brakiyal ve radiyal nabız palpasyonları normal saptandı. Bir gün sonra hasta, aterosklerotik subklaviyan daralma etyolojisine yönelik, risk faktörleri modifikasyonu önerileri, ikili antiagregan ve antihiperlipidemik tedaviler verilerek şifa ile taburcu edildi.

OPS4-11

İleri derecede tortiyöz sol internal karotid arter stenozunun başarılı revaskülarizasyonu

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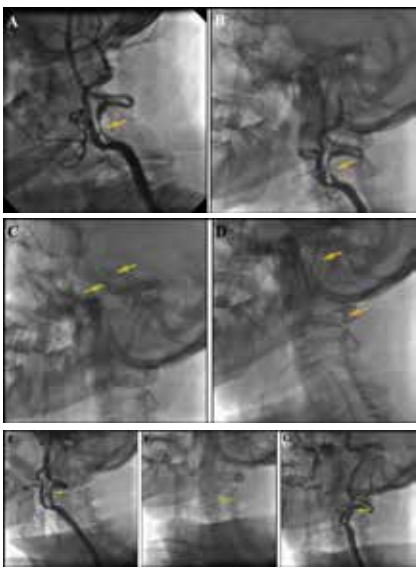
Seksen iki yaşında erkek hasta, son bir ay içerisinde iki kere geçici iskemik atak öyküsü mevcut olup yapılan karotis dopplerde sol internal karotid arter (IKA) darlığı saptanması ile karotis anjiografi yapılmasına karar verildi. Karotis anjiografide sol IKA'da ileri derecede tortiyözite ve %90 darlık izlendi. Karotis stent implantasyonuna karar verildi. Tortiyöz sol IKA %90 lezyon distaline filtre yerleştirildi. Distal filtre telinin yanından, bodywire oluşturmak amacıyla extra-support tel geçildi. 4.0x20 mm balon ile predilatasyon yapıldı. 7-9x30 mm self-expandable, açık hücreli stent yerleştirildi. Stentin lezyonu tamamen kapsamadığı görüldü. İkinci stentin yerleştirilmesine karar verildi. İlk denemede tortiyözite nedeniyle geçilemedi. İkinci extrasupport tel yerleştirildi. Tüm bunlara rağmen lezyonun geçilmesinde zorlukla karşılaşılması nedeniyle stenti kayganlaştırılması amacıyla, stent propofole batırıldı. Destek teller ve propofolün kayganlaştırıcı etkisi ile 6-8x40 mm self-expandable, açık hücreli ikinci stent başarı ile implante edildi. 5.0x20 mm balon ile postdilatasyon yapıldı ve optimal açıklık sağlandı. Literatüede KKA (Kommen Kartons arter) ve IKA lezyonları; <30° hafif derece, 30°-60° arası orta derece ve >60° ileri derecede tortiyöz olarak sınıflandırılır. Orta ve ileri derecede tortiyözite genellikle 80 yaş üzeri hastalarda saptanmaktadır. Biz, ileri derecede tortiyöz zorlu İKA lezyonuna başarılı perkütan girişim vakamızı sunduk.



OPS4-12

Periferik perkütan girişim sonrasında gelişen nadir komplikasyon; Renal subkapsüler hematom

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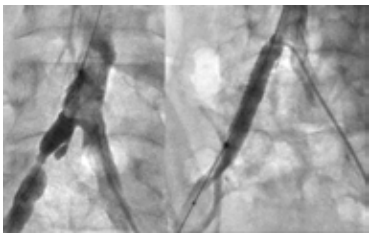
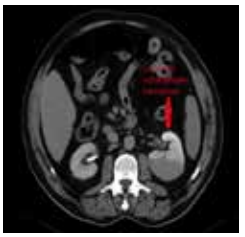
Giriş: Periferik arter hastalığı (PAH) tedavisinde endovasküler girişim yöntemleri son on yılda cerrahi tedavi yerini almıştır. Bununla beraber perkütan girişim sonrası çeşitli komplikasyonlar izlenebilmekte ve hayatı tehdit edebilmektedir. Yazımızda endovasküler girişim sonrası gelişen ve nadir olarak izlenen renal subkapsüler hematom (RSH) vakasını bildirmeyi amaçladık.

Olgu Sunumu: Altmış üç yaşında erkek hasta, diabetes mellitus ve hipertansiyon(HT) hikayesi mevcut. 40 gün önce farklı seanslarda sirkümfleks ve sağ koroner artere başarılı perkütan girişim uygulandı. Hastanın fontaine 2b kladikasyo şikayeti nedeni ile elektif şartlarda sağ common iliak artere başarılı periferik perkütan girişim yapıldı (Şekil 1). Hasta klopidogrel 75 mg 1x1, asetilsalisik asit 100 mg 1x1, rosuvastatin 20 mg 1x1, valstartan + hidroklorotizid 320/25 mg 1x1, metformin 1000 mg 1x1 ve metoprolol 50 mg 1x1 kullanmaktaydı. Mevcut medikal tedavisinin ek olarak işlem sırasında hastaya 60 ü/kg unfraksiyone heparin uygulandı. Başarılı sonlandırılan işlemden 4 saat sonra hastanın şiddetli sol yan ağrısı başladı. Vücut sıcaklığı 36.4 0C, arter kan basıncı 150/80 mmHg, nabzı 103/dk düzenli izlendi. Fizik muyanede akut batın bulgusu izlenmeyen hastada sol kostovertebral açı hassasiyeti mevcuttu. Elektrokardiyografi sinüs ritminde ve iskemik değişikliği yoktu. Laboratuarda; wbc 12.300/mm^3, hemoglobin 12.2 g/dl, platelet 228/mm^3, bun 60 mg/dl, kreatinin 1.92 mg /dl, ck-mb 15 u/dl, ck 73 u/dl, sodyum 132 mmol/l, potasyum 4.47 mmol/l izlendi. acil batın ultrason incelemede sol renal bölgede 93x45 mm boyutlarında hematom izlendi. Takibinde kontrastlı abdominal tomografi tetkiki sonucunda sol renal subkapsüler bölgede hemotom olduğu izlendi (Şekil 2). Üroloji kliniği konsültasyonu sonucunda hastanın dual antiagregan tedavisi stoplandı. Hasta klinik, hemogram ve böbrek fonksiyonu açısından yakın takibe alındı. Hemoglabin değerinde 3 birim düşüş olan hastaya 2 ünite eritrosit süspansiyonu replase edildi. Takiplerde hastanın kreatinin değerinde yükselme (2.5 mg/dl) izlenmiş olup sonrasında 1.56 mg/dl değerine gerileldi. Hastanın hematokrit takiplerinde ek düşme izlenmedi. 5 gün sonra çekilen kontrol tomografide hematom 65x50 mm olarak izlendi. Takibinin 18. gününde kliniği stabil olan hastanın ikili antiagregan tedavisi tekrar başlanarak taburcu edildi.

Sonuç: Perkütan girişimler sonrası yaklaşık %1 oranında vasküler komplikasyon izlenebilmektedir. Hematom, femoral girişimler sonrası en sık işlem bölgesinde oluşmakla beraber seyrek olarak retroperitoneal ve intraabdominal alanda oluşabilmektedir. RSH ise çok nadir izlenmekle beraber gelişmesinde klasik risk faktörleri yanında aksesuar renal arter varlığı ve guide-wire nedenli hasar en çok şuçlanan faktör olmaktadır. Literatür incelendiğinde RSH gelişen olgular benzer şekilde invaziv müdahale olmadan başarı ile takip edilmiştir. Bunun yanında olgumuzun alt ekstremite endovasküler tedavi sonrasında RSH gelişen ilk vaka olduğunu düşünmekteyiz.



Şekil 1. Sağ iliak arter lezyonu ve stent sonrası görünüm.



Şekil 2. Sol renal subkapsüler hematom.

OPS4-13

Successful endovascular treatment of severe post-thrombotic syndrome in a patient with venous leg ulcers

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A 50-year-old male who had a history of left lower extremity deep venous thrombosis presented with complaints of leg pain, erythema, persistent edema and venous ulcers in his left lower extremity. Compression stocking therapy was not combating the edema and venous ulcerations associated with the severe post-thrombotic syndrome (PTS). In this case, we decided to perform percutaneous recanalization of chronic deep venous thrombosis. By using the retrograde popliteal venous approach with a 7-Fr sheath, left lower extremity venography revealed chronic post thrombotic occlusion of the deep femoral vein (A). Crossing the lesion was accomplished by combining a directional catheter and a stiff body guide wire. After the stiff guide wire was passed through the lesion, balloon angioplasty was repeatedly performed with a 8.0×100-mm balloon (B) and complete revascularization was provided (C,D). At the 4-week follow up patient's symptoms nearly completely resolved and venous leg ulcers were improved. At the 12-week follow up venous leg ulcers almost completely healed. The patient's ambulatory status and ability to perform activities of daily living were improved. The PTS is a frequent, potentially disabling complication that develops in 20–50% of patients after deep venous thrombosis (DVT). Clinical manifestations include symptoms and signs such as leg pain, edema, redness, telangiectasia, new varicose veins, hyperpigmentation, skin thickening and in severe cases leg ulcers. Post-thrombotic venous ulcers are treated with compression therapy, leg elevation, topical dressings and sometimes hemorheological agents like pentoxifylline but can be refractory to all therapy and tend to recur. Surgical or endovascular procedures should be considered to treat appropriately selected PTS patients. We report a case of severe post-thrombotic syndrome with venous ulcers successfully treated with endovascular therapy.

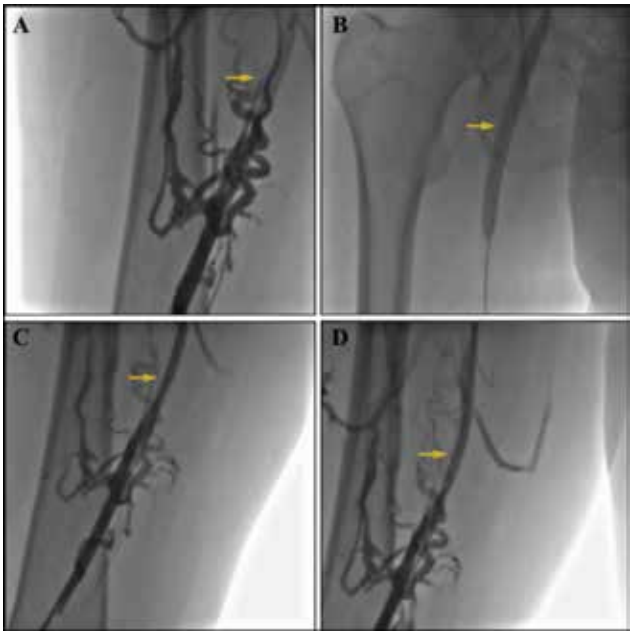

Figure 1.

OPS4-14

Takayasu arteritine bağlı gelişen torakoabdominal anevrizma ve koarktasyonlar

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Yaklaşık 8 yıldır Takayasu arteriti tanısıyla takipli 26 yaşında kadın hastada ciddi aort yetersizliği ve asendan aort anevrizması 7 yıl önce tespit edilmiştir. O dönemde dirençli hipertansiyon gelişmesi üzerine renal arteryel Doppler ultrasonografide her iki renal arterlerde ciddi darlık saptanmıştır. Preoperatif aortografi ve renal arteryel anjiyografi yapılmıştır (Video 1-4). Bentall ameliyatı ve bilateral renal stent implantasyonu uygulanmıştır. Yedi yıl sonraki çok kesitli bilgisayarlı tomografik anjiyografide desendan aortta proksimal çapı 45 mm, en dar çapı 16 mm olan koarktasyon görünümü, ve abdominal aortta proksimal çapı 39 mm olup en dar çapı 17 mm olan koarktasyon görünümü izlenmiştir (Video 5, 6). Aortik kateterizasyon uygulandı (Video 7). Koarktasyon öncesi ve sonrası basınçlar sırasıyla 159/75 mmHg ve 140/73 mmHg olup, Darlık öncesi ve sonrası arasında 20 mmHg'nin altında gradient tespit edildiğinden medikal tedavi kararı verildi. Torakoabdominal aort koaktasyonları konjenital veya fibromuskuler displazi, mukopolisakkaridoz ve Takayasu arteriti gibi edinsel nedenlere bağlı gelişebilir. Takayasu arteriti aort ve dallarını tutan progresif enflamatuvar bir hastalıktır. Klinik gereklilik halinde açık cerrahi veya perkutan girişimler aortik koarktasyonunda altın standarttır.

OPS4-15

Successfull stenting of multiple renal arteries in a patient with renovascular hypertension

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A 58-year-old male was referred for coronary angiography because of resistant stable angina pectoris. This patient had a long history of arterial hypertension and had been treated with beta-blockers, diuretics and angiotensin receptor-blockers. Despite this triple therapy, his blood pressure was not controlled properly and remained in the range of 170/110 mmHg. He had also history of diabetes. His serum creatine was 1.5 mg/dl and creatine clearance was 75 ml/min. In his coronary angiogram a chronic total occlusion of left ascending artery (LAD) was observed. His renal angiogram showed 80% stenosis of the left renal artery, 90% stenosis of the right superior renal artery, and 90% stenosis of the inferior right renal artery (Figure 1a-c, respectively).Firstly we successfully treated the LAD CTO lesion with an antegrade CTO procedure.At an apart single session, percutaneous transluminal angioplasty and stenting of the 3 significant lesions were performed and a bolus of 10.000 IU heparin was administered. The left renal artery was engaged with a 7 Fr JR 4 guiding catheter and the lesion was crossed with a 0.014 inch guidewire. A 5 mm x 19 mm bare metal stent was directly deployed at 18 atm without residual stenosis (Figures 2a). Next, the right inferior polar artery was engaged using the same guiding catheter, and the lesion was crossed with the same guidewire. A 6 mm x 14 mm bare metal stent was deployed at 18 atm without residual stenosis (Figures 2b). Using the same guiding catheter and guidewire, stenting of the right superior renal artery was also performed with implantation of a 5 mm x 14 mm bare metal stent (Figure 2c). After that we performed a digital substraction angiography (DSA) procedure (Video 1 and 2). The patient recovered uneventfully and was discharged after the procedure on 100 mg of acetyl salicylic acid lifelong and 75 mg of clopidogrel for one year. At 3-month follow up, the patient's blood pressure control was adequate at around 110/70 mmHg on the same antihypertensive regimen. Accessory renal arteries are aberrant arterial branches originating directly from the aorta, usually serving a small portion of the kidney. The anecdotally reported observations that portions of the kidney served by accessory renal arteries tend to exhibit delayed parenchymal enhancement on angiographic studies have appeared in the literatüre. These reports have led to the suggestion that the relative lack of perfusion in the renal parenchyma served by the slower flow and lower pressure of accessory renal arteries results in increased renin secretion and subsequent development of hypertension.

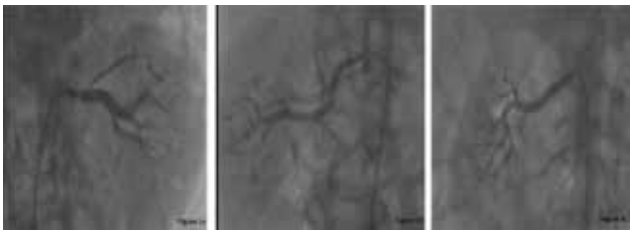

Figure 1. (A) Renal angiogram showed 80% stenosis of the left renal artery. **(B)** Renal angiogram showed 90% stenosis of the right superior renal artery. **(C)** Renal angiogram showed 90% stenosis of the inferior right renal artery.

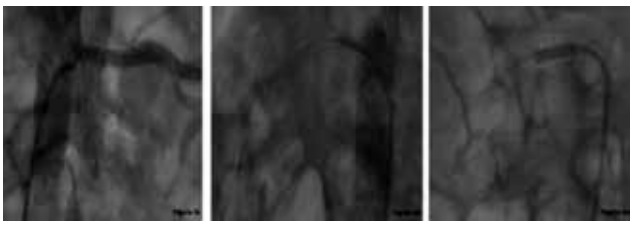

Figure 2. (A) Bare metal stent deployed on the ostium of the left renal artery. **(B)** Bare metal stent deployed at 18 atm without residual stenosis. **(C)** Stenting of the right superior renal artery with a bare metal stent.

OPS4-16

Carotid artery stent may provide ipsilateral antegrade re-flow of intracranial arteries: a case report

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Stroke remains to cause the permanent disability and death in the world in the last decade. Carotid artery stenosis is one of the major causes of ischemic stroke. Thanks to advances in catheter, vascular device, and stent etc. technologies, carotid artery stenting (CAS) has become alternative treatment for CAS. Recent published study regarding carotid revascularization indicated that CAS was not inferior to Carotid endarterectomy in improving clinical outcomes in symptomatic carotid artery disease. The aim of CAS is that providing stabilization atherosclerotic plaque and prevent embolism to cerebral arteries by deploying stent to the lesion. Because ipsilateral intracranial antegrade flow and pressure are reduced in to tight lesions in internal carotid artery (ICA) or common carotid artery (CCA), those arteries' flow is generally supplying from other cerebral arteries via anterior communicant artery or posterior communicant artery. CAS may provide ipsilateral antegrade re-flow of intracranial arteries. We reported that providing antegrade reflow in anterior cerebral artery (ACA) after stenting left internal cerebral artery. A 65 years old male with hypertension had ischemia stroke 1 month ago presented to our clinic. After noninvasive assessment, Carotid angiography revealed that a tight 95% lesion on left ICA and absence of ACA flow (Video 1, 2 and Figure 1, 2). The lesion was crossed 0.014" guide wire. And then a 7x30 mm (The Cordis PRECISE PRO Rx Nitinol Stent) self-expandable stent was implanted accompanied distal protection. The stent was post dilated with 5x20 mm balloon (the Cordis AVIATOR® Plus) due to more than 50% residual lesion. Final result was satisfactory (Video 3 and Figure 3). In addition, cerebral angiography shown that antegrade flow of ACA was provided again from ipsilateral carotid artery (Video 4 and Figure 4). More than ten millions of patients every year worldwide are suffering ischemic stroke which lead to permanent disability and death. CAS is increasingly used to prevent embolization from carotid artery. CAS crushes atherosclerotic plaque to the vessel wall. In this way, plaque is stuck between stent striate and vessel wall. Reducing in Intracranial antegrade flow and pressure result to interrupt of intracranial cerebral artery in ipsilateral to tight lesions on ICA or CCA. Those arteries' flow is generally supplying from other cerebral arteries via anterior communicant artery or posterior communicant artery. CAS may provide ipsilateral antegrade re-flow of intracranial arteries by increasing pressure and flow. At our patients after left ICA stenting, left ACA flow restored.

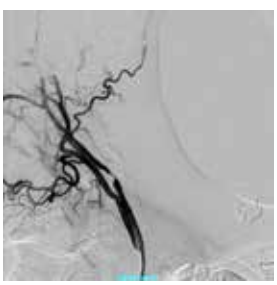

Figure 1. A tight 95% lesion on left ICA.

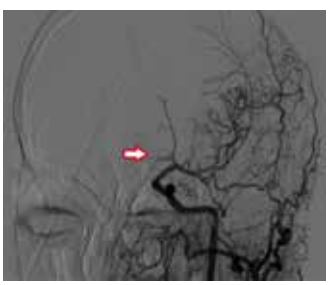

Figure 2. Absence of ACA flow.

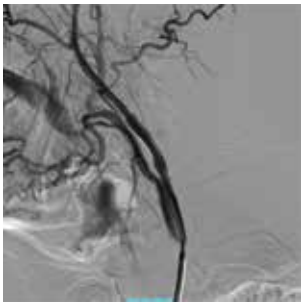

Figure 3. Final result was satisfactory.

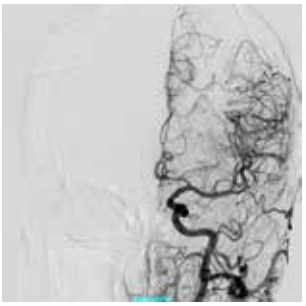

Figure 4. Antegrade flow of ACA was provided again from ipsilateral carotid artery.

OPS4-17

Subklavyen çalma sendromu: Ateroskleroz ve king

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Yetmiş bir yaşında bayan hasta baş ağrısı, omuz ağrısı, baş dönmesi ve vücut postüründen bağımsız olan sık bayılma şikayetleri ile başvurdu. Hipertansiyon ve Diyabetes Mellitus için oral tedavi alan hastanın 5-6 yıldır bu şikayetlerinin olduğu 4 yıl önce sol elde uyuşma sebebiyle karpal tünel sendromu tanısı aldığı ve sol el bileğine cerrahi işlem yapıldığı öğrenildi. Hastanın sol radyal, ulnar ve brakial nabızları mevcut değildi. Yapılan renkli Doppler ultrasonografide karotis sistemde akımın kraniale doğru olduğu, sol vertebral arterde ters yönde akım bulunduğu saptandı. BT anjiyografide sol subklavyen arter çıkışından 1 cm sonra tıkalı olduğu izlendi. Hastanın sol subklavyen arterdeki darlığına perkütan transluminal anjiyoplasti ve stent işlemi planlandı. Sağ femoral arterden periferik görüntüleme yapıldı. Sol subklavyen arterin aortadan çıkışının 1 cm sonrası tam tıkalı olduğu görüldü (Şekil 1). Sağ guiding kateter taşıma destekli kateterler vasıtası ile uygun teller kullanılmasına rağmen lümen içine düşülemedi. Sol brakial arterden doppler usg vasıtası ile retrograd girişim yapıldı. subklanyen arterin tıklalı alan distaline opak verildiğinde lezyon bölgesinde darlıkla beraber king izlendi. Brakial arterden gönderilen retrograd ilerletilen telin kılavuzluğunda lezyon geçildi (Şekil 3) 7x29 mm stent implante edildi (Şekil 4). İşlem sonrası sol kolda nabızlar alınır hale geldi. Hastanın şikayetleri düzeldi. Proksimal subklavyan arterin tıkanıklığı nedeniyle vertebral arterden subklavyan artere olan geri akım, subklavyan steal sendromu olarak tanımlanmıştır. Steal sendromu Perkütan anjioplasti ve cerrahi yöntem ile tedavi edilebilmektedir. Aterosklerozun fizyopatolojisinde ana etken olduğu bu durum vakamızda subkavyen arterin king yapması ile birlikte işlemi zorlaştıran bir hal almıştır. Brakial arterden gönderilen telin kılavuzluğu ile doğru lümen saptanabilmiş ve işlem başarılı olmuştur. retrograd kılavuz tel kullanmak bu tür vakalarda işlemin başarısı açısından önemli olduğunu hatırlatmak istedik.

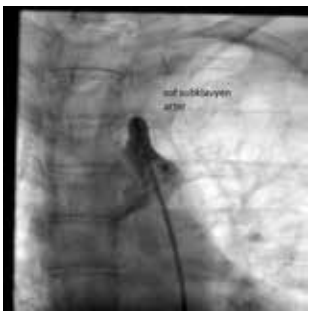

Figure 1.

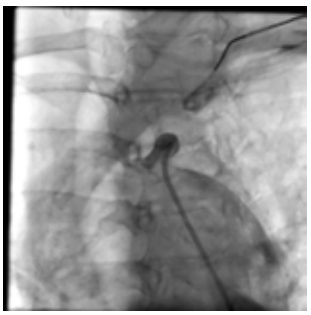

Figure 2.

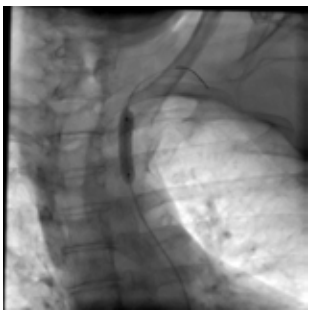

Figure 3.

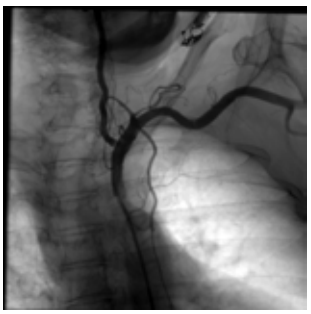

Figure 4.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

OPS4-18

Superiyor mezenterik artere perkütan yolla başarılı stent implantasyonu

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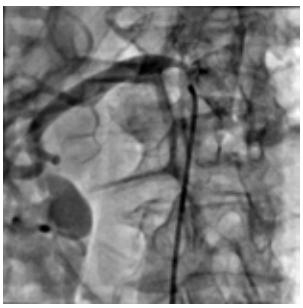
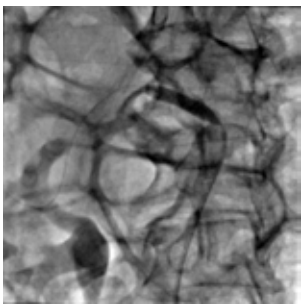
Giriş: Kronik mezenter iskemisi intestinal bölgedeki kanlanma bozukluğuna bağlı olarak gelişen klinik bir durumdur. Etyolojide en sık neden ateroskleroza ikincil intestinal bölge damarlarının tıkanmasıdır. Kronik mezenter iskeminin tedavisinde koruyucu tedaviye ek olarak açık cerrahi ve endovasküler girişim yer almaktadır. Endovasküler girişim cerrahi mortalite ve morbiditesi yüksek hastalarda tercih edilen tedavi yöntemidir.

Olgu Sunumu: Seksen bir yaşında erkek hasta kliniğimizde hipertansiyon diyabet ve koroner arter hastalıkları nedeniyle takip edilirken son 1 yıldır var olan yemek sonrası karın ağrısı ve iştahsızlık şikayeti nedeniyle mezenter iskemisi ön tanısıyla aorta-iliak BT anijyo planlandı. Hastaya merkezimizde çekilen aorta iliak BT anjiografide Çölyak arter çıkışında %80 darlık, Superior Mezenterik Arter (SMA) proximalinde %80-90 darlık saptandı. Hastanın mevcut komorbid durumları nedeniyle cerrahi girişimin yüksek riskli olacağının belirtilmesi üzerine perkutan yolla SMA'ya girişim planlandı. Hasta anjiyografi laboratuvarına alındı ve sağ femoral artere seldinger yöntemi ile 6 F kılıf yerleştirildi. Sağ guiding kateter ile Superior Mezenter Arter (SMA) görüntülendi (Şekil 1). SMA proximalinde %80-90 darlığa direkt stent implante edildi (Şekil 2, 3). Hastanın klinik takiplerinde karın ağrısı şikayeti olmaması üzerine taburcu edildi.

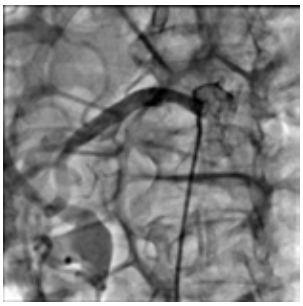
Tartışma: Perkütan yolla mezenterik artere stent implantasyon yapılan vakalarda seçilen hasta profili genel durumu bozuk malnütre hastalardır. Bu hastalar preoperatif yüksek riske sahip olmakla birlikte postoperatif komplikasyon riski de artmaktadır. Minimal invaziv girişimler gelişebilecek komplikasyonları minimalize etmektedir. Birden fazla mezenter arterde ciddi darlık olması halinde tek damara girişim planlanmakta olup öncelikli arter SMA olarak seçilmektedir. Hastalar stent implantasyonu sonrası sık doppler görüntülemeleri yada semptom tekrarı değerlendirilerek yakın takip edilmelidirler.



Şekil 1.



Şekil 2.



Şekil 3.

OPS4-19

Treatment of brachial fistula stenosis

Mustafa Ahmet Huyut[1], Bekir İnan[2], Mehmet Ergelen[1], Asım Enhoş[1], Erdem Karaçöp[1], Ahmet Bacaksız[1], Abdülkadir Yıldız[1], Aydın Nadir[1], Maharram Nasifov[1], Nuray Kahraman Ay[1], Özge Özden Tok[1], Hüseyin Uyarel[1], Ömer Göktekin[1]

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58 years old female was admitted our clinic because of ineffective hemodialysis. From his past history we learned that he was taking hemodialysis for two months but during last month hemodialysis could not be effective because of the low flow. After patients examination due to the weak fistule improvement we decided to make venography and found 90 percent brachial fistula stenosis (Figure 1). After diagnose we determined to put stent to the lesion. At first we prepared the lesion with 4x20 mm balloon expansion (Figure 2). For final treatment we insert 5x11 mm Biomatrix flex drug eluting stent to the lesion (Figure 3, 4). After four months follow up patient has no complaint and hemodialysis treatment goes on without any problem. We would like to demonstrate a brachial fistula stenosis case and its treatment.

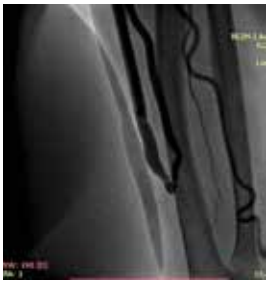
Figure 1.


Figure 2.


Figure 3.

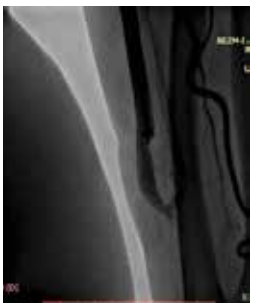
Figure 4.

OPS4-20

Percutaneous treatment of a coronary subclavian steal syndrome

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Coronary subclavian steal syndrome refers to a decreased or reversed internal mammary artery flow, which causes angina in patients with in situ internal mammary to coronary artery graft. The primary cause is the subclavian artery stenosis or compression proximal to the internal mammary artery graft. We present a clinical case of a 57 years old man patient with a history of single coronary bypass graft surgery admitting with efor angina and claudication of his left arm. We referred the patient for nuclear stress testing which demonstrated a large size area of lateral wall and apikal ischemia on imaging. The patient exercised to exercise capacity of 9 METS with moderate chest pain but no ECG changes. Coronary angiography revealed a severe stenosis of the left subclavian artery, proximal to the left internal mammary artery graft and retrograde filling of the iternal mammary artery from the left anterior descending artery. Percutaneous transluminal subclavian artery stenting was succesfully performed. The reduction of efor angina and claudication of arm was provided.

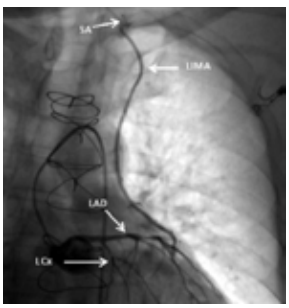

Figure 1. Caudal anteroposterior view. Retrograde filling of the LIMA from the LAD into the left SA distal to the occlusion.

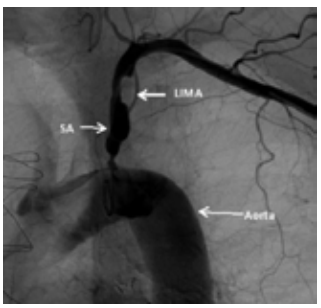

Figure 2. Subclavian angiography showes a proximal severe stenosis with slow distal flow.

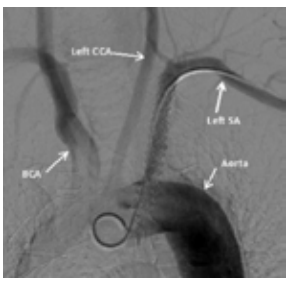

Figure 3. View of arcus aorta and its branches after percutaneus transluminal subclavian.

OPS4-21

An overlooked cause of lower extremity arterial occlusion: Popliteal entrapment syndrome

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Case Report: 46 years old male patient who had medical history of hypertension, diabettus mellitus and right big toe amputation was admitted to outpatient cardiology clinic with an exertional leg pain which occurs at the begining of walking. Baseline popliteal artery blood flow was triphasic at lower extremity arterial doppler ultrasonography but it changes to biphasic when patient makes planter flexion. Upon this, MRI imaging was requested on suspicion of PAES. We observed the extension of upper medial gastrocnemius muscle between popliteal artery and popliteal vein at MRI. 30% stenosis of current popliteal artery (Figure 1) was increased to 95% when patient makes plantar flexion (Figure 1b) at conventional coronary angiography. After Heart Surgery and Orthopedics consultation, medial head of the gastrocnemius and popliteal muscle was excised and the pressure on the popliteal artery was removed. Patient had no calf pain after operation in the outpatient controls.

Discussion: Popliteal entrapment syndrome (PAES) occurs by congenital discordance of the between popliteal artery and muscle-tendinous tissue at popliteal area which frequently encountered by incorrect or delayed diagnosis. PAES should be kept in mind, especially in young person who doesn't have any risk factor and have claudication in first step. Recurrent compression of the popliteal artery is able to trigger endotelial dysfunction and plaque formation. Acute arterial oclusion is often as chronic critical crural ischemia which can be developed as a result of thrombus formation after plaque erosion or rupture and distal embolization. Thus our patient had amputation story which was caused by ischaemia. In the diagnosis step; changes in flow patterns with plantar or dorsal flexion during the doppler ultrasonography can occur suspicion. For definitive diagnosis; conventional angiography and MRI is essential for demonstrating relationship of arteries and muscle or tendon. Two different surgical strategy can be used in PAES. One of them is removing the pressure from that region and the other option is to bypass the area of the occlusion. Anatomical abnormalities and damage of popliteal arteries determines the surgical approach. If there is no damage in popliteal artery, the preferred method could be muscle resection for elimination the pressure whereas if there is a damage in popliteal artery, vascular graft can applied.

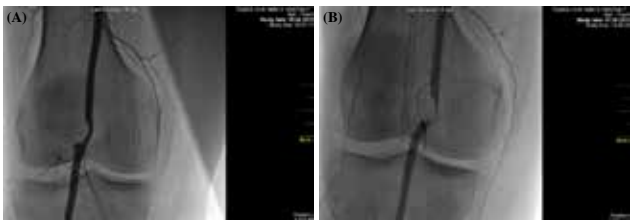

Figure 1. (A) Previous plantar flexion. **(B)** After plantar flexion.

OPS4-22

Koroner anjiografi sırasında insidental saptanan psödokoarktasyon olgusu

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[1]*Bakırköy Dr. Sadi Konuk Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, İstanbul*
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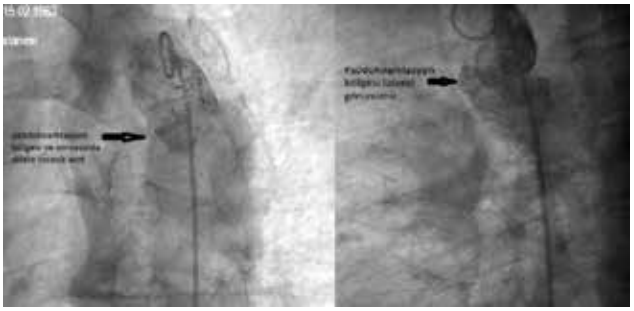
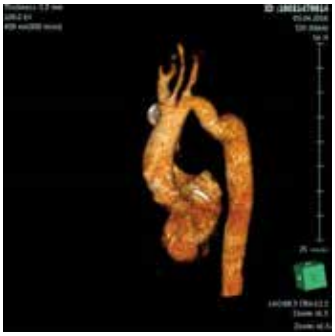
Giriş: Psödokoarktasyon aortik arkın nadir karşılaşılan konjenital anomalisi olup, ligamentum arteriosum bölgesinde obstrüksiyon olmaksızın elonge ve büklüntülü olması şeklinde tanımlanmaktadır. Genellikle bening karakterli olup asemptomatik seyretmektedir. Biz bu yazımızda koroner anjiografi sırasında insidental olarak saptanan psödokoarktasyon vakasını sunmayı amaçladık.

Olgu Sunumu: Elli üç yaşında erkek hasta, hipertansiyon, diabetes mellitus ve koroner arter hastalığı mevcut (circumflex (CX) koroner artere 2 defa girişim yapılmış). Stabil anjina nedeniyle yapılan eforlu elektrokardiyografik stres testi pozitif olması ve transtorasik ekokardiyografide (TTE) inferolateral duvarda hipokinezi saptanması üzerine hastaya koroner anjiografi planlandı. Sağ femoral arter girişim yolu kullanıldı. Standart klavuz tel aortada ilerletilirken desenden aorta proksimalinde telin takıldığı izlendi. Tel geri alınıp opak verildiğinde proksimale doğru dolum olduğu izlendi. Koarktasyon düşünülen bölgeden hidrofilik guide-wire yardımı ile geçilerek selektif koroner anjiografi ve CX mid lezyona stent işlemi başarıyla yapıldı (Şekil 1). Ardından pigtail katater alınarak desenden aort bölgesinden anteroposterior (AP) ve lateral görüntü alındı. AP pozda darlık gerçek koarktasyon gibi izlense de lateral pozda olgunun psödokoarktasyon olduğu düşünüldü (Şekil 2). Pull back esnasında darlık proksimal-distal arasında 13 mmHg gradyent alınması ön tanımızı destekledi. Hastanın takibi sırasında TTE tekrarlandı. Ejeksiyon fraksiyonu %45, hafif mitral yetmezlik ve hafif sol ventrikül hipertrofisi izlenmiş olup eşlik eden konjenital kardiyak anomali izlenmedi. Aortaya dıştan bası ihtimalini dışlamak ve kontrast madde nefropati gelişimini engellemek amacıyla hastaya 48 saat sonra bilgisayarlı tomografi (BT) yöntemi ile aortagrafi yapıldı. Aort duvarına dıştan bası olmadığı, aortun o bölgede elonge ve tortüyöz olduğu izlendi (Şekil 3). Hastada kladikasyo, tekrarlayan karın ağrısı, renal disfonksiyon vb. distalde hipoperfüzyon bulgularının olmaması ve takip eden torasik aortun cerrahi sınırda anevrizmatik genişlemesi izlenmemesi üzerine hastaya medikal tedavi kararı verilerek hali ile taburcu edildi.

Tartışma: Psödokoarktasyon, nadir karşılaşılan konjenital anomali olup proksimal-distal ekstremite basınç gradyentinin <25 mmHg olması ve artmış kollateral damarların olmayışı tanısının konulması ve gerçek koarktasyondan ayırt edilmesinde önemlidir. Tanısı çoğu zaman çekilen göğüs x-ray grafide sol üst mediastende dansite artışı şüphesi sonucu konulur. Genellikle bening karakterli olup spesifik tedavi gerekmemektedir. Bazı vakalarda gerçek koarktasyonda olduğu gibi konjenital kardiyak anomalilerin (biküspis aort kapak, patent duktus arteriosus, ventriküler septal defekt vb.) eşlik etmesi, tortüyosite sonrası torasik aortada anevrizma, diseksiyon veya rüptür riski olması nedeniyle tanısının konulması ve bu yönde irdelenmesi önem kazanmaktadır.



Şekil 1. Psödokoarktasyon anjiografik görünümü.



Şekil 2. Psödokoarktasyon üç boyutlu BT anjiografik görünümü.

OPS4-23

Uzun süre önce vena cava inferiorda bırakılan kılavuz telin snare yardımı ve cerrahi ile çıkarılma girişimi

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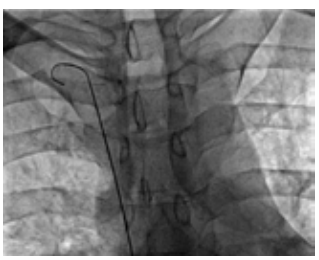
Adana Numune Eğitim ve Araştırma Hastanesi Seyhan Uygulama Merkezi, Kardiyoloji Kliniği, Adana

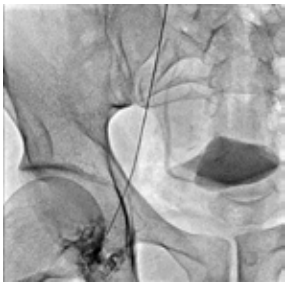
Günümüzde artan endovasküler girişim oranlarıyla birlikte, bu işlemlere sekonder gelişen komplikasyonlar da artmaktadır. Bu girişimler parenteral nutrisyon, ilaç uygulamaları, kemoterapi, pacemaker yerleştirilmesi ve hemodinamik monitorizasyon amacıyla yaygın şekilde kullanılmaktadır. Nadir olmakla birlikte periferik venöz kateterizasyon sırasında damar içerisinde kılavuz tel kalabilmektedir. Bu olgumuzda uzun süre önce damar içerisinde kılavuz tel kalan bir hastada, telin çıkarılması esnasında karşılaşılabilecek zorlukları tartışıyoruz. Yaklaşık 4 yıl önce dış merkezde femoral venden damar yolu için kateter takıldığı sırada kılavuz telin vena cava inferiorda bırakılma öyküsü olan hasta kliniğimize atipik göğüs ağrısı ile başvurdu. Çekilen akciğer grafisinde Vena kava inferior trasesinden sağ subklavian ven çıkımına kadar uzanan ve kıvrık ucu truncus brachiocephalica içerisinde katlanan muhtemel 0.38 inch kalınlığında 90 cm uzunluğunda J kılavuz teli olduğu görüldü. Çekilen ayakta direkt batın ve pelvis grafilerinde distal tel ucunun vena cava inferiordan sağ femoral vene ilerlediği görüldü. Hastaya bu J kılavuz telin çıkarılması amacı ile girişim kararı alındı. Hastaya sol femoral venden 8F sheat yerleştirildi. Telin distal ucunun sert olması ve olası vasküler zedelenme riski nedeniyle proximaldeki J ucundan yakalanması planlandı. İki farklı snare sistemi kullanılarak (En-snare, Andrea-snare) telin proximaldeki J ucu yakalandı ve skopi altında sol femoral vene doğru çekildi. Tel dışarı alınırken vena cava düzeyinde dirençle karşılaşıldı, telin sarmal şeklinde yaylanan parçası dışarı alındı. Telin iç kısmı ve sarmal haline gelen parçasının çıkarılması için pacemaker lead ekstraksiyon cihazı kullanılması düşünüldü. Ancak telin dışarıdaki sarmal parçası çekilirken koptu. Bunun üzerine hasta cerrahiye verildi. Cerrahi sırasında kılavuz telin ven intimasına ciddi fibröz doku ile ven boyunca yapışık olduğu görüldü. Telin intimadan sıyrılarak çekilmesine rağmen hareket ettirilememesi üzerine rüptür riski nedeniyle tel içeride bırakılarak işleme son verildi. İntrakaval yerleşimli yabancı cisimlerin neden olabileceği komplikasyonlar arasında sepsis, aritmi, perforasyon, miyokard hasarı, endokardit, venöz trombüs gelişimi ve pulmoner emboli bulunmaktadır. Bu nedenle özellikle intravasküler serbest yabancı cisimlerin erken dönemde perkütan veya cerrahi yolla çıkarılması gerekmektedir. Geç dönemde yaygın fibrozis nedeniyle bu cisimlerin çıkarılması teknik olarak oldukça zordur. Belirtilen komplikasyonlar açısından asemptomatik olan hastalar invaziv girişim düşünülmeden medikal tedavi ile takip edilebilir.

Şekil 1. Dışarı çekilen telin bütünlüğünün bozularak sarmal şekle dönüşmesi.

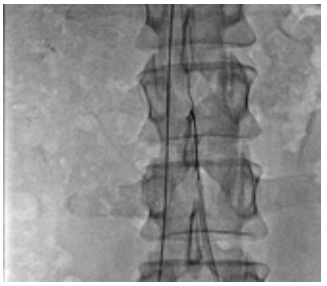


Şekil 2. Sağ brakiosefalik vene uzanan guide wire'ın proksimal kısmı.

Girişimsel kardiyoloji / Karotis ve periferik vasküler



Şekil 3. Sağ ana femoral ven içinde bulunan guide wire'ın distal kısmı.



Şekil 4. Sol ana femoral ven'den telin snair ile damar dışına alınırken deforme olması.

OPS4-24

Symptomatic nodal rhythm after successful right carotid artery stenting

Ahmet Hakan Ateş, Uğur Arslan, Aytekin Aksakal, Huriye Yücel, İlsen Atasoy Günaydın, Osman Can Yontar

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Introduction: Carotid stenosis is a major risk factor for stroke and can lead to serious complications. Treatment aims to reduce the risk of stroke by controlling or removing plaque build up and preventing blood clots. The main treatment way is carotid endarterectomy but carotid artery stenting is a less invasive alternative to endarterectomy to treat symptomatic carotid stenosis.

Case Report: A 80 year old female was admitted to our department with transient ischemic attack (TIA). The doppler ultrosonography of carotid artery showed serious narrowed lesion of right internal carotid artery. After that carotis angiography was performed and percutenous angioplasty was planned for treatment of carotid stenosis (Figure 1). After neurology consultation the patient was taken to angiography laboratory. 7F sheath was fixed to right femoral artery and 7F Terumo Destination sheath was placed to right common carotid artery. Later, the emboshield distal protection system (4.0-7.0 mm) was placed to distal region of lesion.). Afterwards tapered self expandable carotid stent was implanted (6-8 x 30 mm, Abbott) and the post dilatation was done with 5x15 mm balon expandable balloon (Figure 2). One day later,during the follow up in coronary care unit symptomatic nodal rhythm was detected (Figure 3). There weren't any beta blockers, calcium channel blockers or other negative chronotropic drugs in patient medical list and the potassium level was in normal limits. The temporary pacing was placed to the right ventricular apex through femoral venous way. After follow up permament VVIR pace maker was performed to patient.

Discussion: Carotid angioplasty / stenting is a minimally invasive endovascular procedure that compresses the plaque and widens the lumen of the artery.this procedure has lotd of complications like distal embolism, dissection or rhythm problems.

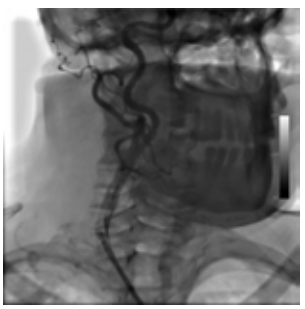

Figure 1.

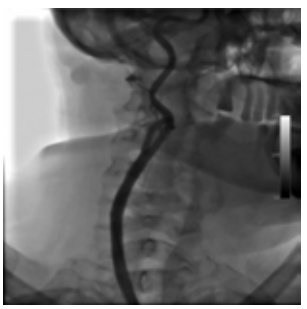

Figure 2.

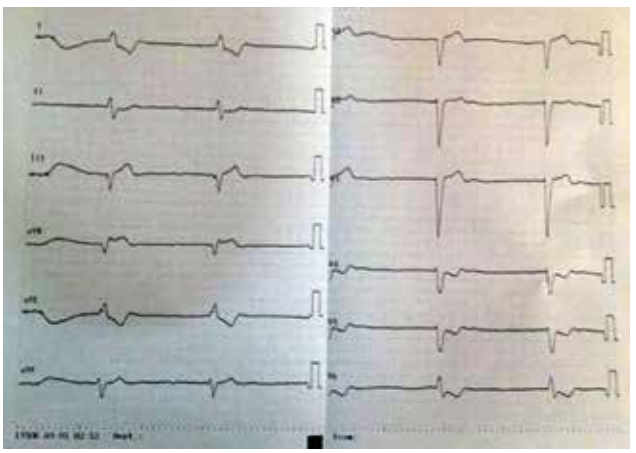

Figure 3.

OPS4-26

Kompleks çölyak trunkus artere ve SMA ya başarılı PTA stent uygulaması

Beytullah Kulaç, Şeref Alpsoy, Demet Özkahramanlı Gür, Aydın Akyüz, Dursun Çayan Akkoyun, Niyazi Güler, Kubilay Erselcan, Şahin Topuz

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Abdomen angina ile gastroentoroloji polk başvuran hastaya endoskopi yapılmış. Patoloji saptanamaması üzerine Kalp Damar Cerrahisi Polk yönderilmiş. Kalp Damar Cerrahisi tarafından Abdominal Renkli Doppler US tetkiki istenmiş. Trunkus Çölyakus normal,orifis düzeylerinde aterosklerotik kalsifikasyonların izlenmesi üzerine BT anjiogram önerilmiş.Hastanın şikayetlerinin devam etmesi üzerine yapılan Torakoabdominal Aorta Bt de Çölyal Trunkusun abdominal aort çıkışında %790 darlığa neden olan fibro fatty plak formasyonu, SMA çıkımında lümende yaklaşık olarak 2 cm'lik segment boyunca oklüyon tesbit edilmesi üzeriene tanısal diognastik anjiogram için kardiyoloji kliniğine yönderildi. Hastaya femoral axesten vistarel anjiogram yapıldı. Çölyak Trunskusun ostealinde %95 ciddi darlık, SMA osteal de %99 darlık tesbit edildi. 7F Destination Sheatle Çölyak hizasına gelindi. 0.014 guide wire ile lezyon geçildi. 0.8x135 cm crosssing support katater splenic artere geçildi. 0.18x300 cm extrasupport guide wire gönderilerek 5.5F guide liner ile exchange yapıldı. 0.35x260 cm Amplatz Süper stif guide wire guide liner içerisinden exchange yapılarak guide liner dışarı alındı. 8 mm x 26 mm greft kaplı stentv implante edildi. Sonraki seansta SMA ya işlem için 7F Destination sheat ve 0.35x260 cm hidroflic guide wire ile SMA osteale oturuldu. %99 lezyon 0.18x135 cm Crossing Support Katater ve 0.18x300 cm guide wire ile geçildi. 3.0x30 mm OTW PTA balonu ile predilatasyon pta yapıldı.Ardından exchange tekniği ile 0.18 guide wire ile 0.35x260 cm amplatz süper stift guide wire değiştirildi.5.0x26 mm greft stent lezonda açıldı. Bir hafta sonraki kontrolünde abdomen anginasının geçmesi üzerine yemek yemeğe başlayan hastanın 1 aylık polk kontrolünde karın ağrısının tamamen ortadan kaybolduğu ve kilo aldığı gözlendi.

OPS4-27

Sağ koroner artere perkütan koroner girişim sonrası belirginleşen anormal koronerler arası bağlantısı

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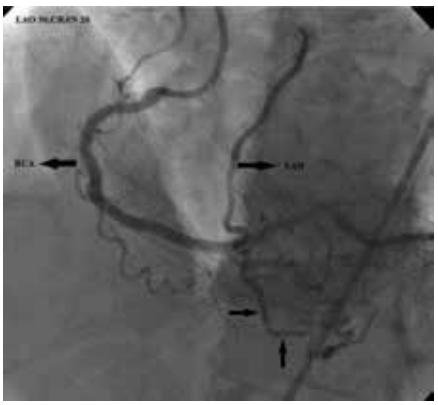
Yetmiş altı yaşında kadın hasta 2 aydır mevcut olan göğüs ağrısı ile kardiyoloji polikliniğinde değerlendirildi. Hastanın koroner arter hastalığı nedeni ile daha önce iki kez perkütan koroner girişim öyküsü mevcuttu. Hastaya gerekli hazırlıkların ardından koroner anjiyografi yapıldı. Koroner görüntülemede sağ koroner arter (RCA) proksimal stent içi %30, sağ ventrikül dalı sonrasında ciddi darlık saptandı ve RCA'ya perkütan koroner girişim kararı verildi (Video 1). Sol sistemin sağ kaudal açıdan görüntülenmesinde ise LAD gövde stenti açık ve devamında ise RCA bölgesine doğru anormal koroner uzanım gösterdiği görüldü (Video 2). Ayrıca hastanın sağ koroner arter görüntülenmesinde ciddi darlık sonrasında PDA'nın retrograd gelen kan akımı ile yıkandığı görüldü (Video 1). RCA ciddi darlık bölgesine başarılı balon ve ilaç kaplı stent uygulandı ve farklı açılardan alınan kontrol görüntülerde RCA'nın PDA'nın devamı şeklinde anormal koronerler arası bağlantı ile LAD'yi retrograd doldurduğu izlendi (Resim 1, Video 3, 4). Koronerler arası bağlantı çok nadir görülen bir anomalidir ve kollateralden farklı bir durumdur. Arterler açık uçlu bir devamlılık göstermekte ve ikinci arterin doluşu kollateraller yoluyla olmamaktadır. Koronerler arası bağlantı genellikle sağ koroner arter ve sirkumfleks arter arasında izlenmektedir. Bizim vakamızda ise sağ koroner arter ile LAD arasında izlenmiştir. Sağ koroner artere radyoopak madde verildiğinde LAD sağ koroner arterin devamı gibi dolmakta, LAD'ye radyoopak madde verildiğinde RCA dolmaktadır. Bu tip iki arterin birbirini doldurduğu durumlar iki yönlü akım olarak isimlendirilir, bazen tek yönlü akımda görülebilir. Koronerler arası bağlantının benign bir anomali olduğu düşünülmektedir ve genellikle koronerlerde tıkayıcı bir lezyon yoktur. Bizim olgumuzda ise koroner arter hastalığı gelişmeden önce koronerler arası bağlantının mevcut olduğunu düşünmekteyiz. Bu olguda olduğu gibi koronerlerden birinde tıkanma meydana geldiğinde yeterli olmasada koronerler arası bağlantı ile akımın hiç kesintiye uğramadan devam edeceği ve bu anomalinin bulunmasının koruyucu olduğu düşünülmektedir. Biz bu olguda koroner girişim sonrası belirginleşen farklı bir koronerler arası bağlantı olgusunu sunmak istedik.



Şekil 1. Sağ koroner arterin girişim sonrasında görüntülenmesinde RCA'nın anormal koroner bağlantı ile LAD'yi retrograd doldurduğu gözlenmektedir. Anormal koronerler arası bağlantı oklarla gösterilmiştir.

OPS4-28

Akut koroner sendromlu bir hastada sirkumfleks koroner arterde dev sakküler anevrizma ve ciddi darlık olgusu

Ömer Şatıroğlu, Ramazan Gengörü, Hüseyin Durak, Oğuzhan Bodur, Hakan Duman, Mehmet Bostan, Zakir Karadağ

Recep Tayyip Erdoğan Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Rize

Altmış bir yaşında erkek hasta, istirhatle gelen göğüs ağrısı yakınması ile kliniğimize başvurdu. Daha önceden bilinen koroner arter hastalığı öyküsü yoktu. Aterosklerotik risk faktörlerinden sigara içme ve hiperlipidemisi mevcuttu. Tetkiklerinde; Elektrokardiyografisinde, miyokard iskemi bulguları mevcuttu, Labaratuvar tetkiklerinde, kardiyak enzimleri normal bulundu. Hastaya, stabil olmayan angina pektoris (akut koroner sendrom) tanısı konularak, tedavisi başlandı ve koroner anjiyografi yapıldı. Sirkumfleks koroner arter (Cx) ortasında, ciddi darlık ve darlık sonrası dev sakküler anevrizma saptandı (Video 1, 2). Lezyona, standard koroner ilaç kaplı veya çıplak metal stent takılması darlığı açacak ancak uzun vadeli sakküler koroner anevrizma rüptür riskini ortadan kaldırmayacaktır. Bu sebeple, lezyon önce balonla predilate edildi ardından Cx ortasındaki lezyonu ve anevrizmatik segmenti kaplayacak şekilde, 3.0-24 mm boyutunda, 12 atmosfer basınçla, greft kaplı stent takıldı (Video 3). Lezyon tam açıldı, dev sakküler anevrizma görünümü kayboldu. (Video 4). Şikayetleri geçen ve klinik olarak stabil seyreden hasta ikili antiagregan, antiiskemik ve antihiperlipidemik tedaviyle taburcu edildi.

OPS4-29

Successful management of coronary artery rupture during PCI

Veysel Oktay, Onur Kılıcarslan, Cüneyt Kocas, Ahmet Yıldız, Murat Kazım Ersanlı

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A 58 year old male patient presented to our emergency department because of chest pain started two days ago. His pyhsical examination was unremarkable. ECG showed negative T waves and 2 mm ST segment depression at precordial derivations and troponin level was high so he was diagnosed as non ST-segment elevation acute myocardial infarction. He was hospitalized in intensive coronary care unit (ICCU) and medical treatment was started. Diffuse critical stenosis of proximal LAD was seen by coronary angiography. Ad hoc PCI was decided by our heart team. A soft guidewire was passed through the lesion and predilatation was performed using a 2.5x12 mm (Invader) balloon. After predilatation 2.5x30 mm and 2.75x25 mm drug eluting stents (DES) were deployed as overlapping. Post-dilatation was also performed with a 3.0x12 mm non-compliant balloon at 18 atmospheres. After postdilatation, the patient complained of severe chest pain and hypotension was developed. Immediate angiographic images were taken which showed type 3 coronary artery perforation according to Ellis classification with massive and pulsatile extravasation from the LAD into the pericardial space (Figure 1). Bedside echocardiography was revealed cardiac tamponade. Prolonged balloon inflation over perforation site was repeated for 10-15 minutes and emergent pericardiocentesis was performed. Because of persistent perforation, a 3.0x16 mm PTFE covered stent was implanted in the site of the rupture, with subsequent complete restitution of coronary blood flow in the LAD and termination of extravasation into the pericardial space (Figure 2). The patient was followed up for 2 days in ICCU and repeated transthoracic echocardiography showed minimal intrapericardial fluid but no progression was observed. The patient was discharged from our hospital 1 week later under medical treatment without any wall motion abnormality. Coronary perforation is a rare but fatal PCI complication that can result in life threatening cardiac tamponade. The incidence of coronary perforation ranges from 0.19% to 0.59%.The possible factors predicting its development are clinical factors: advanced age, female sex, renal impairment, non-ST-elevation myocardial infarction patients; angiographic factors: chronic total occlusion, coronary artery calcification, type-C lesions, tortuous vessels, target lesions in the circumflex and right coronary arteries, long target lesions (>10 mm), and eccentric lesions; technique-associated factors: use of hydrophilic/ extra stiff wires, atherectomy devices, increased balloon to artery ratio, high-pressure stent post-dilatation, and cutting balloons. Prolonged balloon inflation, reversal of the anticoagulation with protamine, covered stent implantation and surgery are the management options of coronary artery perforation. Interventional cardiologists have to be well- experienced about PCI related complications to prevent morbidity and mortality.

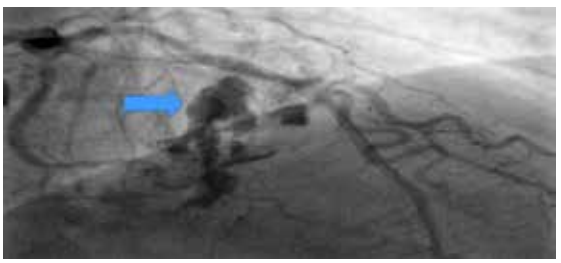

Figure 1. Coronary artery perforation after postdilatation (Blue Arrow).

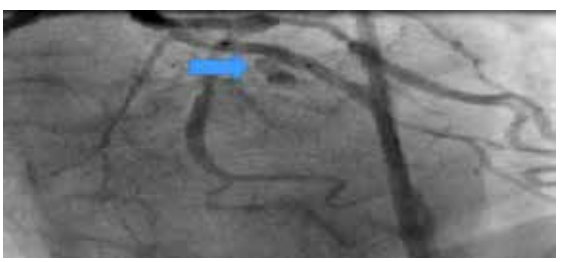

Figure 2. Covered stent implantation and limitation of coronary perforation (Blue Arrow).

OPS4-30

Stent embolization into the left ventricle during primary percutaneous coronary intervention

Özgül Uçar Elalmış, Ümit Güray, Mehmet İleri, Pınar Duyuler, Deniz Şahin, Havva Tuğba Gürsoy

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Introduction: Stents have become an important component of percutaneous coronary intervention (PCI), with up to 80% of all treated vessels being stented. In clinical trials, successful stent delivery rate is about 99%, but despite improvements in stent technology, stent deployment failure is not an unusual event in daily clinical practise.

Equipment and Methods: A sixtyfour-year-old man presented to the emergency room with new onset chest pain. ECG revealed ST segment elevation in inferior leads and he was taken to catheter laboratory for PCI. Coronary angiography revealed 50% stenosis in LAD at the first diagonal branch level and 98% stenosis in proximal RCA. Our decision was primary PCI for RCA and fractional flow reserve measurement for LAD. First we placed a 6F right Judkins guiding catheter (Launcher®, Medtronic AVE, USA). After wire crossing of the stenosis, we intended to deploy a 4x15 mm stent (Simchrome® CoCr, Simeks, Turkey). However, we were unable to pass the stenosis with the stent and the complete guiding catheter system dislodged in the ascending aorta. We decided to use an AL1 (Amplatzer) guiding catheter. However, we noticed that the stent slipped from the the balloon and it was seen freely floating in the anterior mitral annulus in the fluoroscopy. After completing the primary PCI, we attempted to retrieve the stent from the left ventricle (LV) by a snare catheter (AndraSnare®, Andramed, Reutlingen, Germany) and a multicurve ablation catheter. We tried to dislodge the stent, which was thought to be between the mitral chords. We reached the stent with multicurve ablation catheter, but the shape of the stent was distorted.

Results: Despite many efforts, the stent could not be retrieved. Since, the stent seemed stable at the posterior mitral annulus, we decided to leave it there. We performed a transosephageal echocardiography (TEE) to determine the location of the stent and to see whether the mitral valve was damaged or not. TEE revealed a small tear in the posterior mitral valve and associated mild mitral regurgitation. Patient was discharged uneventfully and periodic echocardiographic examinations revealed no progression in two years.

Discussion: Stent losses are commonly due to stripping off the stent from the delivery baloon. In a series, peripheral stent embolization occured in 10 of 3537 patients (0.3%). Embolization can be into the coronary arteries, LV or extremities. Since, this is a potentially serious condition, every effort should be made to retrieve the stent. However, a stable stent may become unstable and adjacent tissues can be damaged during prolonged extraction procedures. The management of stent embolization into the LV is not clear. In a case report, the stent was stuck at the basal anterior wall and in another, the stent was stuck under the posterior mitral valve leaflet. No complications occured in both patients during the follow-up. We also decided to follow our medically with echocardiographic exams.

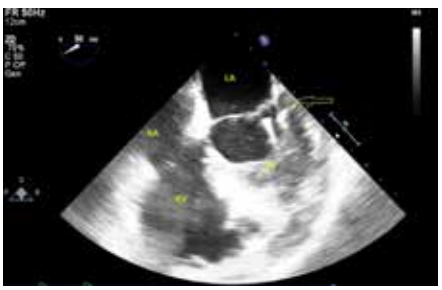

Figure 1. TEE examination, arrow points acoustic shadowing due to the stent under the posterior mitral valve. LA: Left atrium, LV: Left ventricle, RA: Right atrium, RV: Right ventricle.

OPS4-31

Zor bir işlem: Aort-safen distal anostomozundan RCA proximaline balon uygulama

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Giriş: Koroner arter hastalığı ön tanısı ile yapılan koroner anjiyografilerin %20 kadarında kronik total oklüzyon izlenir. Kronik total oklüzyonların %60 kadarı anterograd yolla %40 kadarı ise retrograd yolla revaskülarize edilmektedir. Retrograd yol olarak septal, safen ven ve epikardiyal kollateraller kullanılmaktadır. Bu vakada RCA'ya Ao-safen ile anastomoz yeri tıkanıklığı üzerinden revaskülarizasyon işlemi sonrası retrograd nativ akımın kesilmesi üzerine anastomoz sonrası total oklüzyona retrograd başarılı PTCA işlemi uygulanmıştır.

Olgu Sunumu: Seksen yaşında erkek hasta bilinen koroner arter hastalığı ve kalp yetersizliği olup acile tipik anjina şikayeti ile başvurmuştu. Ekokardiyografide EF %35, 2/4 mitral yetersizliği ve 2/4 aort yetersizliği saptandı. EKG sinde inferior derivasyonlarda ST elevasyonu izlenen hastaya yapılan koroner anjiyografide LMCA plaklı, LAD proksimalde %100 tıkalı, OM2 başında %40 darlık, RCA PDA başında %50 darlık izlendi. LİMA-LAD grefti açık, Ao-safen-RCA grefti açık ve anastomoz yerinde %80 darlık izlendi (Şekil 1). Nativ RCA retrograd RCA safen üzerinden tamamen doluyor idi. Koroner anjiyografiyi takiben Ao-RCA-safen ostiumuna kılavuz kateter ile oturuldu. Ao-RCA-safen anatomoz yerindeki %80 lezyondan kılavuz telle geçildi. Lezyona 3.5x12 mm balon ile PTCA uygulandı. PTCA sonrası lezyona 4.5x12 mm zotarolimus kaplı stent implante edildi (Şekil 2). Tam açıklık sağlandı. Stent sonrası nativ RCA'da anastomoz öncesinde total oklüzyon izlendi (Şekil 3). Nativ RCA'ya retrograd tel ilerletilmeye çalışıldı ancak başarı sağlanamayınca işleme son verildi. İşlem sonrası koroner yoğun bakımda takip edilmekte olan hastada ventriküler fibrilasyon gelişti. Acil defibrilasyon sonrası hasta tekrar koroner anjiyografi laboratuvarına alındı. Ao-safen-RCA grefti anastomoz yerine yerleştirilen stentin açık olduğu saptandı ancak nativ RCA'ya doğru akım olmadığı gözlendi (Şekil 4). Grefte kateter ile oturulduktan sonra 0.14 inç kılavuz tele Simmons kateterin açısına benzer şekilde açı verildi (Şekil 5). Tel nativ RCA distaline ilerletilip geri çekilirken telin ucu bu şekilde nativ RCA'ya retrograd olarak kanüle edilmiş oldu (Şekil 6). Sonrasında tel ilerletildiğinde tel gövdesi RCA distaline doğru yönlendi-

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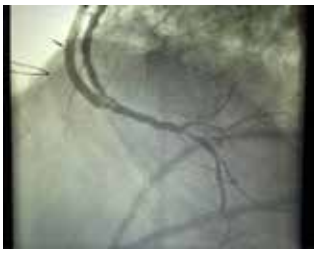
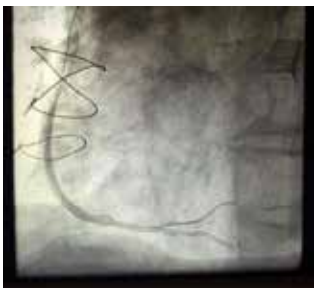
yordu. Telin ucunun nativ RCA'daki lezyonu retrograd geçmesini sağlamak amacıyla stent içine 3.0x12 mm balon şişirilerek tel ilerletildi ve %100 lezyondan retrograd olarak geçildi (Şekil 7). Sonrasında Lezyona 3.0x12 mm balon ve RCA anastomoz hattındaki stent içine 4.0x12 mm balonlar yerleştirilerek kissing PTCA yapıldı (Şekil 8a). PTCA sonrası nativ RCA'ya retrograd TIMI-III akım sağlandı ve rezidü %30 lezyon ile işleme son verildi (Şekil 8b). Başarılı PTCA sonrası hasta yoğun bakım ünitesine alındı. VT/ VF tekrarı olmadı. Hastaya dual antikoagülan tedavi başlandı. Bu vakamızda RCA- safen greftine PTCA+ stent sonrası gelişen nativ RCA akımının ortadan kalkmasını retrograd yolla perkütan koroner işlemle başarılı bir şeklide sonlandırdık.



Şekil 1.

Şekil 2.



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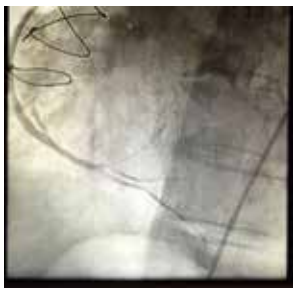

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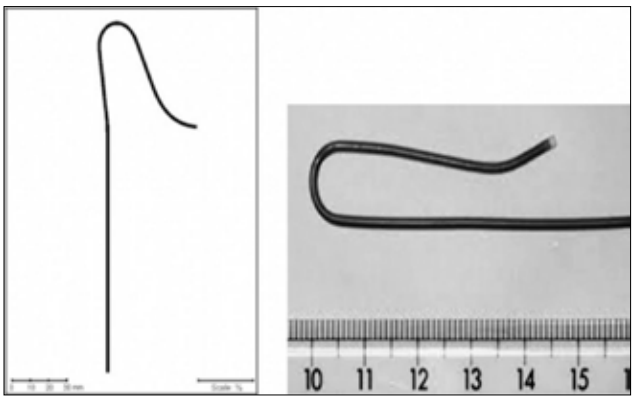
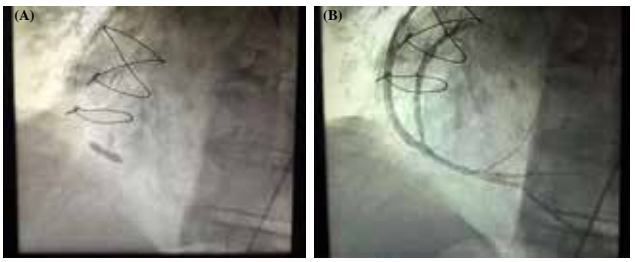


Şekil 3.

Şekil 6.

Şekil 7.



Şekil 8.

OPS4-32

Farklı teknikler ile sağ koroner arter çıkış anomalili STEMI hastalarına başarılı perkütan koroner girişim

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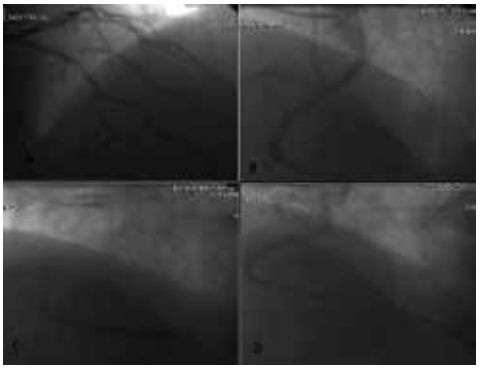
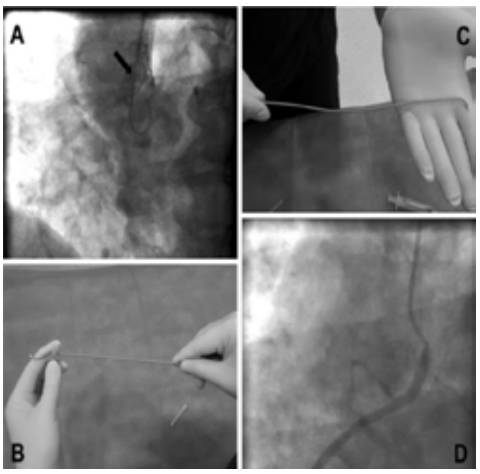
Sağ koroner arter(RCA) çıkış anomalisi (0.06% to 0.5 %) çok nair görülen bir anomalidir. Tüm koroner çıkış anomalileri arasında ise RCA'nın sol valsalvadan çıkması %10 civarındadır. Bu hastalara sol ve sağ amplatz, Barbeau veya yeniden şekil verilmiş sol Judkins kateterler kullanılmıştır. Yazımızda iki farklı RCA çıkış anomalili STEMI hastasına farklı teknikle kateterler kanule edilip PCI yapılmıştır. Elli sekiz ve 62 yaşındaki iki inferior MI'lı hastanın yapılan koroner anjiografisinde RCA sol valsalvadan çıktığı görüldü. Bu iki vakayada farklı teknikler ile RCA'ya kateterler entübe edildi. İlk vaka da yapılan KAG sonucu LAD osteal plaklı, D1 dalı sonrası %40, 2. diyagonal (D2) dalı sonrası %80 darlık, CX plaklı, RCA'nın sağ koroner sinüsten çıkmadığı ve yüksek çıkışlı olduğu proksimalinde %80 darlık, crux öncesi %60 darlık izlendi. Hastaya birçok kateter denendikten sonra EBU 3 kateter ile yaklaşılıp PCI yapıldı (Şekil 1). Daha sonra LAD için elektif PCI alınan hastanın anjiosu sağ radialden yapıldı. Sağ Judkins kateter önce normal denendi ama entübe olması zorlanınca, kateterin uç kavisi düzleştirilip yeniden şekillendirilerek kolayca entübe olduğu görüldü (Şekil 2). İkinci vakada da yapılan KAG sonucu LAD ve Cx normal, RCA'nın sağ koroner sinüsten çıkmadığı,sol valsalvadan LMCA üstünden ters yönde çıktığı ve proximal %70, mid seviyede de subtotal darlık görüldü. Hastaya farklı kateterler denendikten sonra sol 4.5 judkins kateter kavsi biraz daha düzeltildikten sonra sol valsalvaya sırt kısmı yaslatılarak RCA ya entübe edilip PCI yapıldı (Şekil 3).



Şekil 1.



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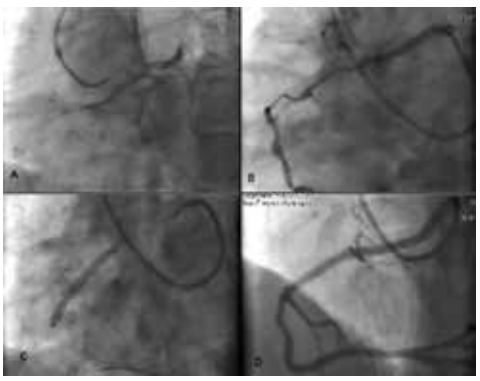

Şekil 3.

OPS4-33

LMCA, LAD and CX dissection during coronary intervention in a young patient with SLE: Due to prematüre atherosclerosis or vasculitis?

Sevinç Aktürk, Hüseyin Göksülük, Nazlı Turan, İrem Müge Akbulut, Demet Menekşe Gerede, Çetin Erol

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SLE is a chronic inflammatory disorder and mortality occur usually cardiovascular system involvement. Well-recognized cardiac manifestations of SLE include pericarditis, myocarditis, endocarditis, coronary vasculitis and rarely prematüre atherosclerosis.

Case Report: We report a 31year-old female patient who presented to the emergency department with a progresive shortness of breath during a week on a background of 20-year SLE history. Also she had neurological manifestations of SLE. She had been treated since twenty years with corticosteroids and immunosuppresants. She had no other major risk factors for coronary artery disease. Physical examination was normal. Electrocardiyogram showed ST depression and T wave inversion on anterior leads. The cardiac biomarkers were elevated. (troponin=6.87 ng/ml) Echocardiography revealed inferoposterolateral hypokinesia. With diagnosis of non-ST segment elevation myocardial infarction, dual antiplatelet agents and low molecular weight heparin were initiated. Coronary angioplasty performed through right femoral access, showed multiple lesions in left anterior descending artery extending from D2 to the apex (%70-70 consecutive lesions). There was a hazy, %30-40 lesion in circumflex artery and OM1 ostium was totally occluded (Figure 1). Following balloon angioplasty a 2.25x18 mm sized drug eluting stent was implanted to the ostium of OM1. Following stent implantation a coronary dissection extending from LMCA to Cx and LAD occured (Figure 2). After angioplasty, hypotension and cardiac arrest occured. VF detected while CPR and defibrillation was performed and the rhythm returned back to normal. Two bare metal stents were implanted extending from LMCA to LAD and Cx by using culotte tecnique (Figure 3). After angioplasty, the patient was followed in coronary care unit. A trans-thoracic echocardiography was performed and revealed anterior, posterior, inferior and lateral wall motion abnormalities and a left ventricular ejection fraction (EF) of 40%. The patient consulted to the rheumatology and immunsupresant agent was stoped and 100 mg iv prednol was added to treatment. After one week it was reduced to the 60 mg a day. We started cellcept 500 mg twice a day and titrated up to 1000 mg twice a day. Prior to discharge, the echocardiography was repeated and showed a left ventricular EF of 60%. The patient was discharged with the optimal medical treatment.

Discussion: The pathophysiology of coronary involvement in SLE remains unclear. active coronary vasculitis or prematüre atherosclerosis may be the cause In our patient the principle cause is thougt to be active coronary vasculitis due to the clinic improvement after corticosteroid and immunsupresant agent. This case can be useful with similar cases, because it shows us how to treat the patient and how to deal with the possible complications of active coronary vasculitis.

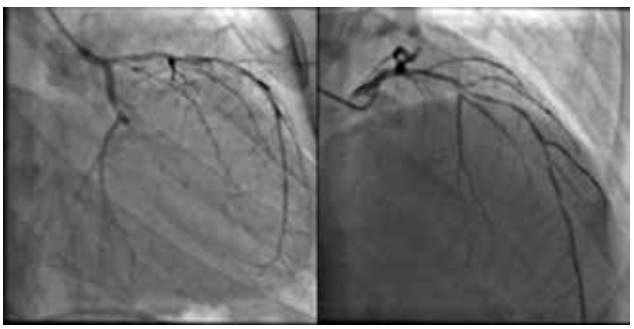

Figure 1. Diffuse coronary lesions due to SLE. OM1 ostium totaly occluded.

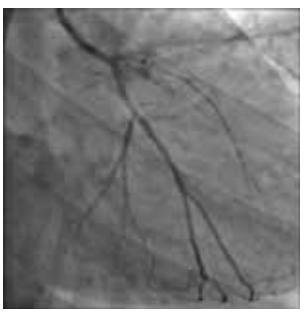

Figure 2. A coronary dissection extending from LMCA to Cx and LAD.

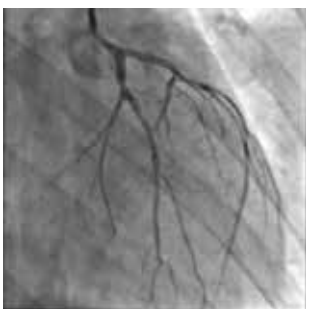

Figure 3. After dissectiontwo bare metal stents were implanted extending from LMCA to LAD and Cx by using culotte tecnique.

OPS4-34

Coronary artery aneurysm after stent implantation

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Department of Cardiology, Trakya University Faculty of Medicine, Edirne

We present coronary aneurysm formation in a patient with recently implanted bare metal stent.

Case Report: A 52 year old female patient with known history of hypertension, and ASA allergy was admitted to our clinic with anterior myocardial infarction and totally occluded Left anterior

descending artery was treated with bare metal stent. After 2 months the patient had undergone dual isotope myocardial perfusion scanning for the evaluation of viability in the chronic total RCA territory. However, MPS revealed %20 anterolateral ischemia and control coronary angiography was planned. Control coronary angiography revealed coronary aneurysm formation in the proximal LAD stent site (Figure 1a, b). We planned IVUS imaging and graft stent implantation to fully cover the aneurysm, and the patient was treated without a complication with graft stent (Figure 2a, b). Coronary artery aneurysms after coronary intervention are rare, with a reported incidence of 0.3% to 6.0%, and most "aneurysms" are in fact pseudoaneurysms rather than true aneurysms. Residual dissection and deep arterial wall injury (rupture or resection of the vessel media) caused by oversized balloons or stents, high-pressure balloon inflations, atherectomy, and laser angioplasty have all been associated with coronary artery aneurysms after coronary intervention. Drug-eluting stents (DES), which locally elute antiproliferative drugs, can dramatically inhibit neointimal growth, thereby suppressing restenosis, but at the same time both DES and BMS potentially causing coronary aneurysms due to other mechanisms, such as delayed re-endothelialization, inflammatory changes of the medial wall, using glococorticoids and colcium and hypersensitivity reactions. In our patient it is considered allergic ethiopathogenesis (patient has ASA allergy).

Conclusion: Type I aneurysm is a type of aneurysm that demonstrates rapid early growth with pseudoaneurysm formation detected within 4 week. This type is typically complicated by clinical pericarditis. The second kind of aneurysm described in the literatüre is that with a "subacute to chronic" presentation (type II) and is typically detected incidentally during angiography for recurrent symptoms or as part of protocol mandated follow-up (usually detected _6 months after the procedure)These aneurysms appear to have the most varied clinical presentations; some patients are asymptomatic, but some have complaints of angına. It seems more likely in this scenario that a chronic arterial response to a metal stent, polymer, and/or drug, may be the basis for aneurysm formation in this subtype. The final reported subtype in the published literature is mycotic or infectious in etiology (type III), Large mycotic aneurysms infected with Staphylococcus aureus after DES or BMS implantation have been reported. In patients undergoing coronary stent implantation in case of recurrent symptoms should be kept in mind the possibility of aneurysm.

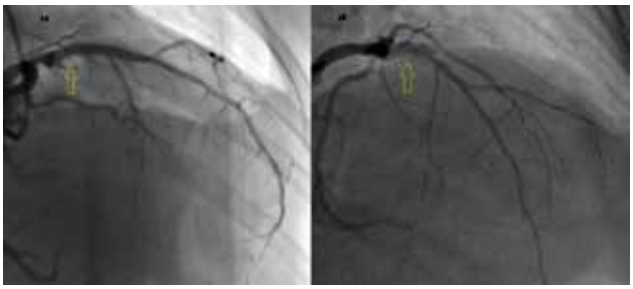

Figure 1. (A) Stent implantation to totaly LAD. **(B)** Coronary aneurism.

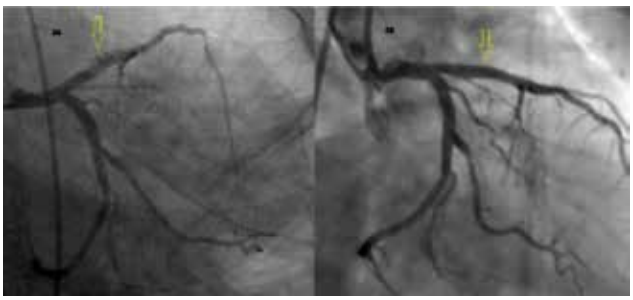

Figure 2. (A, B) Coronary aneurism and graft stent implantation.

OPS4-35

Stenting for giant aneurysm and stenosis of left anterior descending artery in a patient presenting with acute myocardial infarction

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A 77-year old female was brought to the emergency department at our institution because of out-of-hospital cardiac arrest and he had undergone cardiopulmonary resuscitation before the ambulance arrived. On admission, she was intubated and not hemodynamically stable (blood pressure: 80/60 mmHg, heart rate: 145/min). Her twelve-lead electrocardiogram (ECG) showed sinus tachycardia, right bundle branch block and ST segment elevation in leads V1-6 (Figure 1). Left ventricular dysfunction (ejection fraction, 40%), moderate mitral regurgitation and anterior and apical wall hypokinesis were detected on echocardiography. The patient received aspirin, clopidogrel, unfractioned heparin and was referred to the catheterization lab for primary percutaneous coronary intervention (PCI). The angiography showed giant aneurysm involving the mid portion and the diagonal branch of the left anterior descending artery (LAD), significant stenosis of LAD, proximal and distal to the aneurysm, chronic total occlusion of mid left circumflex artery (Cx) and also significant stenosis of nondominant proximal and mid right coronary artery (RCA) (Video 1, 2, 3). The surgical risk of emergency coronary artery bypass surgery (CABG) was determined as high by the heart team. Therefore we decided to perform percutaneous intervention to the LAD. A standard floppy guidewire was used to cross the lesions both proximal and distal to the aneurysm. These lesions were pre-dilated with 1.25x10 mm (Saphire II PTCA baloon, China), 2.0x12 mm (Lepu Medical, Beijing) and 2.5x13 mm (Mozec, noncompliant baloon) NC baloon (Video 4, 5), followed by stenting with 2.25x24 mm (Promus elementTM, Boston Scientific) for distal lesion and 2.75x16 mm (Promus elementTM, Boston Scientific) for proximal lesion (Video 6, 7). After that, post-dilation was performed to the overlapping area (Video 8). Final angiography showed thrombolysis in myocardial infarction (TIMI) III flow in the LAD and in its diagonal branches (Video 9). We could not use graft coated stent because aneurysm involved diagonal branch of the LAD. Blood pressure increased to 120/75 mmHg after the procedure and patient's hemodynamic status was improved. She was transferred to the intensive care unit under the supervision of anesthesiologists.

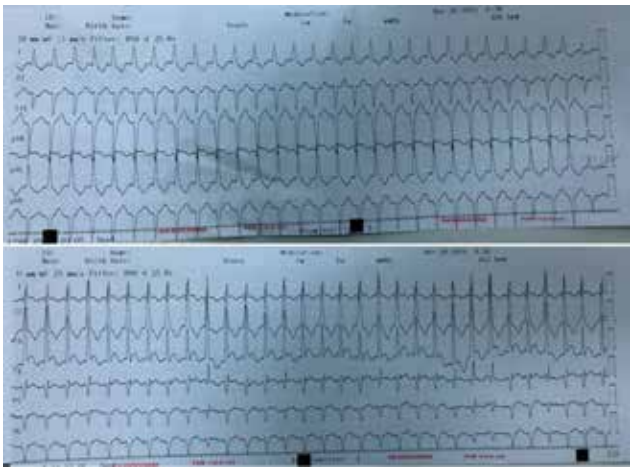

Figure 1.

OPS4-36

Lengthy single piece thrombus aspirated from right coronary artery in inferior ST elevation myocardial infarction

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A 47-year-old female was admitted to emergency department with acute onset of epigastric pain. Electrocardiographic recordings demonstrated changes consistent with an acute inferior wall ST segment elevation myocardial infarction (STEMI) (Figure 1). Patient was administered 300 mg of aspirin and 180 mg loading dose of ticagrelor p.o. and transferred to the catheterization laboratory instantly. Coronary angiography revealed total occlusion of the proximal right coronary artery (RCA) with excess thrombus burden (Figure 2). The RCA was engaged with a 6 French Judkins right coronary catheter and a 0.014 inch floppy coronary guidewire (Choice, Boston Scientific, MA, USA) was used to cross the lesion. After administration of intracoronary tirofiban, we performed ad hoc thrombectomy by using a thrombus aspiration catheter (Export AP Aspiration Catheter, Medtronic Vascular, MN, USA). At first attempt, suction has stopped despite aggressive negative pressure. Aspiration catheter was withdrawn from the guiding catheter to check the system. A lengthy intracoronary thrombus was aspirated as one piece (Figure 3). Subsequent coronary injection revealed proximal critical stenosis of RCA with TIMI III flow. No thrombus was seen in the distal RCA (Figure 4). A 2.75x24 mm drug-eluting stent (Biomatrix, Biosensors Inc., Singapore) has been implemented at the lesion site (Figure 5). Due to conflicting results from randomized clinical trials and registry databases for thrombus aspiration as adjunctive therapy to primary percutaneous coronary intervention, guidelines advice routine thrombus aspiration with class IIA recommendation. Without thrombus aspiration, we believe a satisfactory outcome would not have been reached with intracoronary glycoprotein IIb/IIIa injection, balloon angioplasty, and stent deployment in our case. A partially organized very long thrombus would have precluded an acceptable result.

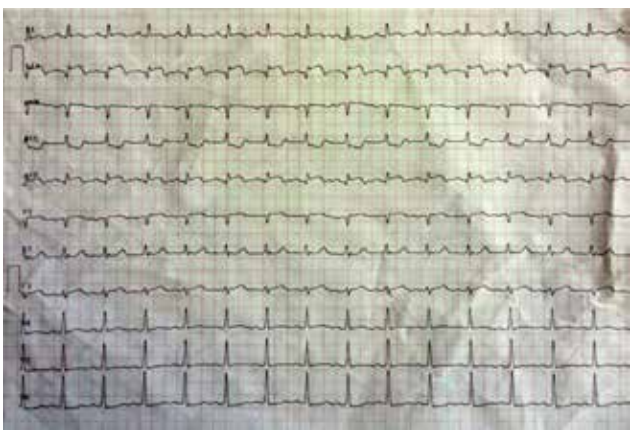

Figure 1. The ECG shows acute inferior STEMI.

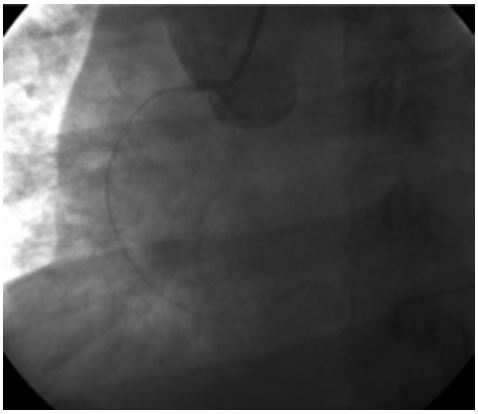

Figure 2. The image shows totally occluded proximal RCA artery with thrombi.

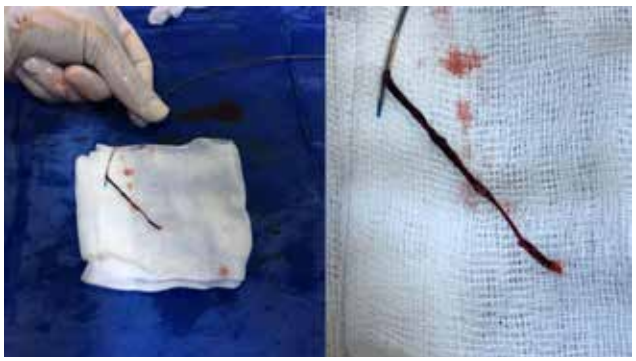

Figure 3. The lengthy single piece thrombi at the tip of thrombus catheter.

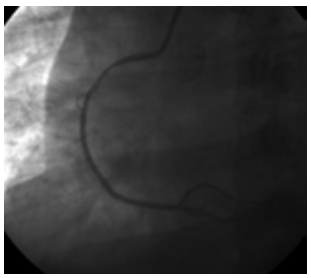

Figure 4. Control angiography showed critical stenosis at proximal RCA with TIMI 3 flow.

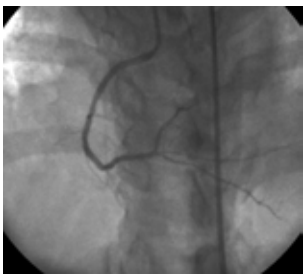

Figure 5. Final image of the procedure.

OPS4-37

Myocardial bridge mimics Wellen's syndrome

Mehmet Fatih Yılmaz, Emrah Acar, Süleyman Çağan Efe, İsmail Balaban, Alev Kılıçgedik, Muhittin Demirel, Elnur Alizade, İbrahim Akın İzgi

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39 year old woman admitted for angına pectoris. On admittion her ECG had

negative T wave in a Wellens pattern in precordial derivation between V1-6 (Figure 1). There were no cardiac risk factors in the background of the patient.The patient had never smoked and had no history of hypertension, hypercholesterolemia, or diabetes.Patient's laboratory results showed that high-sensitive troponin, creatin kinase (CK), CK-MB, hemogram,serum electrolytes and d-dimer were at normal levels. Serebrovascular findings were normal.The blood pressure was 120/75. In the echocardiography, left ventricular systolic function was normal, there were no sign of left ventricular hypertrophy and wall motion abnormalities.Upon patient's continuing chest pain in spite of medical treatment, coronary angiogram was scheduled. In the patients coronary angiography; right coronary artery (RCA) and circumflex artery had no stenosis. LAD had consecutive muscular bridges on proximal and distal segments (black arrows) (Figure 2a, b). There were significant in the entire systolic phase and in 1/3 of the diastolic phase (Video). Our heart team decided that the surgery was the appropriate treatment modality for the patient because of multiple bridging regions. Although the negative T wave is around 1-3% in the young population, it is an ECG sign that is being encountered in a lot of pathological diseases. The most common diseases are acute coronary syndromes, atherosclerotic coronery artery diseases,cerebrovascular accident,apical hypertrophic cardiomyopathy, hypertensive crisis, arrythmogenic right ventricular cardiomyopathy, pulmonary embolism and electrolyte imbalances. We wanted to share that in these events concerning young patients, T negative waves in precordial derivations should be kept in mind with the coronary multiple muscular bridges.

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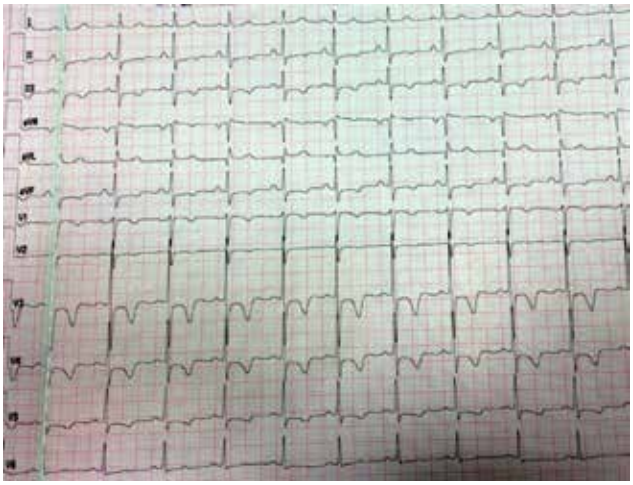

Figure 1.

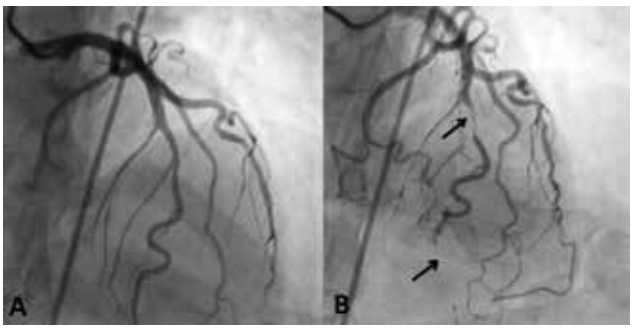

Figure 2.

OPS4-38

Cannabis kullanım sonrası gelişen ana koroner arter diseksiyon ve trombozu

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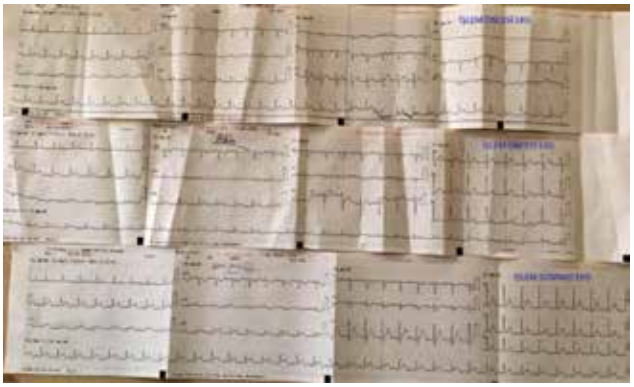
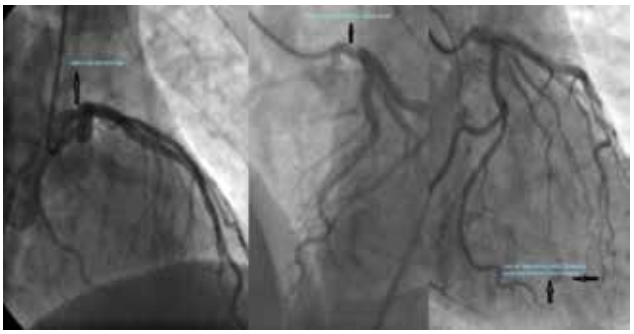
Giriş: Cannabis (esrar), öforik ve bağımlılık etkisi nedeniyle madde bağımlısı kişiler tarafından sık kullanılan, kardiyovasküler yan etkileri iyi bilinen uyuşturucu ajanlardan biridir. Biz bu yazımızda cannabis kullanım sonrasında ana koroner arter diseksiyon ve trombozu gelişerek inferior STEMİ ile prezente olan erkek hasta olgusunu bildirdirmeyi amaçladık.

Olgu Sunumu: Elli iki yaşında erkek hasta, diabetes mellitus, 35 paket/yıl sigara ve 10 yıldır günde 3-5 kez olup 1 yıldır olmayan cannabis kullanım hikayesi mevcut. Gece cannabis kullanım sonrasında baskı tarzında göğüs ağrısı olması üzerine acil servisimize başvuran hastanın çekilen elektrokardiyografide inferior STEMİ bulguları olan izlendi (Şekil 1). Fizik muayenesi normal olan hastanın tansiyon arteri 132/89 mmHg, kalp tepe atımı 92/dk ve düzenli izlendi. Killip-1 klinik bulguları olan hastaya işlem öncesi asetilsalisilik asit(ASA) 300 mg 1x1 verildi. Koroner anjiografi (KAG) onamı sonrası katater laboratuarına alınan hastanın sol ana koroner arter (LMCA) mid bölgesinde diseke plak ve üzerinde hareketli trombüs imajı izlendi. Circumflex-obtus marjinal-2 (CX-0M2) distali ve sol ön inen arter(LAD) distalinde akım izlenmedi. Trombüs embolisi sonucunda oluştuğu düşünüldü (Şekil 2). Sağ koroner arter (RCA)'de lezyon izlenmedi. Hasta tekrar sorgulandığında cannabis kullanımı olduğu öğrenildi. Hastaya intrakoroner 60 U/kg dozundan unfraksiyone heparin(UFH) ve 1.5 mg trofiban uygulandı. Hastaya koroner yoğun bakım ünitesinde UFH ve trofiban infüzyonu, ASA 100 mg 1x1, tikagrelor 90 mg 2x1, atorvastatin 80 mg 1x1, metoprolol 50 mg 1x1 tedavisi uygulandı. Trofiban infüzyonu 24 saat sonunda kesildi. Hastanın yapılan transtorasik ekokardiyografisinde sol ventrikül haraket kusuru izlenmedi. Ejeksiyon fraksiyonu %65 olup kapak, asendan aort veya perikardiyal patoloji izlenmedi. Takiplerinde unstabil kardiyak durumu olmayan ve anjinası tekrarlamayan hastanın ST elevasyonları geriledi. Hasta kardiyoloji konseyinde tartışıldı. Hastaya 5. gününde kontrol KAG yapıldı. LMCA lezyonunun geriliediği, LAD ve CX-OM2 distalinde normal akım paterninin olduğu izlendi (Şekil 3). Şikayeti olmayan hasta dual antiplatelet, b-bloker, statin ve oral nitrat tedavisiyle şifa ile taburcu edildi.

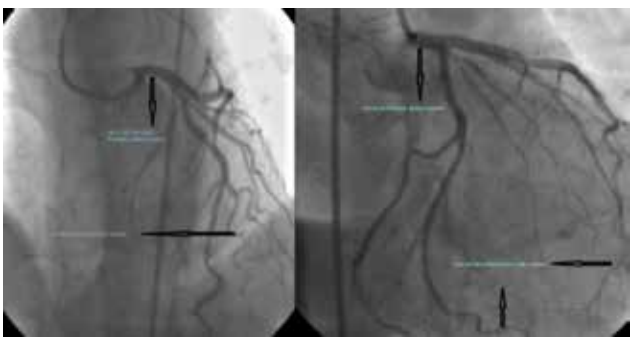
Sonuç: Cannabis kullanımı nöropsikiyatrik bozukluklar haricinde birçok sağlık problemine neden olmaktadır. Kardiyovasküler yan etkileri açısından literatür tarandığında hastaların sıklıkla akut koroner sendrom (AKS) kliniği ile prezente oldukları izlenmektedir. AKS kliniğine ise sıklıkla koroner vazospazm ve/veya diseksiyon lezyonu neden olmaktadır. Bunun yanında alt ekstremite arteriopatisi, kalp yetmezliği, malign aritmi ve kardiyovasküler nedenli ölümlere neden olabilmektedir. Bizim olgumuzda AKS kliniği ve koroner diseksiyon lezyonu olması literatür bulguları ile benzer olmakla birlikte diseksiyon lezyonunun LMCA bölgesinde olması ve trombüs embolisi sonrası inferior STEMİ kliniği gelişmesi açısından literatürdeki ilk olgu özelliğini taşımaktadır.



Şekil 1. Hastanın koroner anjiografi öncesi ve sonrası EKG görüntüleri.



Şekil 2. Hastanın ilk koroner anjiografi görüntüleri.



Şekil 3. Hastanın kontrol koroner anjiografi görüntüleri.

OPS4-39

Hastane dışında ani kardiyak ölüm sonrası sağ kalan bir hastada koroner arter fistülünün coil embolizasyon yöntemi ile kapatılması

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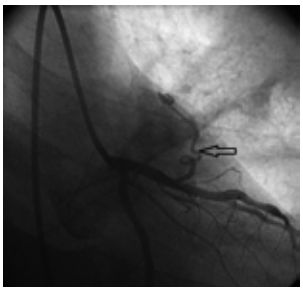
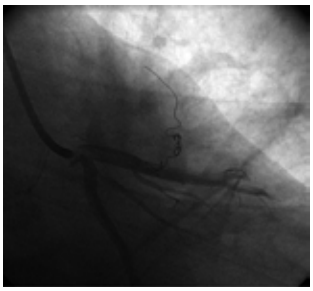
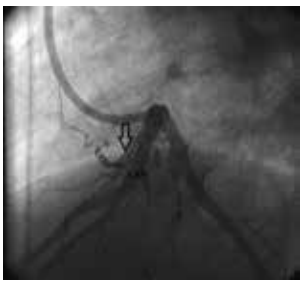
Bu olgu sunumunda sol ön inen arterden köken alıp pulmoner artere açılan fistülün başarılı bir şekilde koil embolizasyon yöntemi ile kapatılmasını ve işlem sırasında karşılaşılan komplikasyonun nasıl giderildiğini sunduk. Hastane dışında ani kardiyak arrest gelişip başarılı resüsitasyon sonrası sağ kalan 35 yaşında erkek hasta kliniğimize sevk edildi. Hastanın hastaneye gelişinde hemodinamik bulguları, biyokimyasal değerleri ve istirahat EKG kaydı normal idi. Transtorasik ekokardiyografik incelemede sağ atriyum ve sağ ventrikül hafif derecede geniş, sistolik pulmoner arter basıncı 35 mmHg idi. Diğer ekokardiyografi bulguları normal idi. Kardiyak arrest etyolojisini açıklamak için yapılan koroner anjiyografide koroner arterlerin normal olduğu, ancak sol ön inen arterden kaynaklanıp pulmoner artere açılan koroner fistül tespit edildi (Şekil 1). Fistülün hemodinamik önemini belirlemek için Qp/Qs oranı hesaplandı, 1.6 idi. Yapılan elektrofizyolojik çalışmada ventriküler taşikardi tetiklenmedi. Hastada ani kardiyak arrest geliştiği için ve hesaplanan şant oranı yüksek bulunduğu için fistülün kapatılmasına karar verildi. Sol ana koroner artere 7F EBU 3.5 kılavuz kateter ile yerleşildi. 0.014 inç hidrofilik kılavuz tel ile fistüle girildi. Kılavuz tel üzerinden mikrokateter ile fistüle ulaşıldı. Fistül içine 3 mm x 4 cm ve 4 mm x 6 cm ebatlarında embolizasyon koilleri (Barricade Coil System, Blockade Medical, Irvine, CA, USA) bırakıldı. İşlem sonrası yapılan kontrol görüntülemede fistülün başarılı bir şekilde koil ile kapatıldığı, fistül içindeki opak geçişinin tama yakın azaldığı görüldü. Ancak koillerden birisinin helikal sarmalı fistül içerisinde olmasına rağmen koilin proksimal ucunun sol ön inen artere taştığı fark edildi (Şekil 2). Koilin proksimal ucunu sol ön inen arterden fistül içerisine göndermek için 0.014 inç hidrofilik kılavuz tel fistül içine tekrar yönlendirildi. Sol ön inen arter proksimalinde 2.0x20 mm perkütan koroner anjiyoplasti balonu 4 atmosfer basınçta şişirildi. Balon şiş iken kateteri hafifçe distale doğru iterek koilin sol inen artere taşan kısmının tamamen fistül içerisine girmesi sağlandı. İşlem sonrası son anjiyografik görüntülemede fistülün başarılı bir şekilde koil ile kapatıldığı ve koillerin tamamen fistül içinde olduğu görüldü (Şekil 3). Hasta işlemden bir gün sonra taburcu edildi ve 3 aydır semptomsuz olarak takip edilmektedir. Girişimsel kardiyolojideki gelişmeler ile son yıllarda koroner arter fistüllerinin koil embolizasyon yöntemi ile kapatılması yaygınlaşmaktadır. Bu yöntem cerrahi fistül ligasyonuna göre daha az girişimsel gibi gözükmektedir. Ancak perkütan koil embolizasyonu sırasında koilin yerinden hareket etmesi, distal embolizasyon, koilin koroner artere taşması ve pulmoner emboli gibi ciddi komplikasyonlar gelişebilir. Bu yüzden koil embolizasyonu yapacak girişimsel kardiyologların bu komplikasyonlardan haberdar olması ve bu komplikasyonların nasıl giderileceği konusunda deneyimli olması gerekir.



Şekil 1. Sağ kaudal açıdan sol ön inen arterden köken alan fistülün anjiyografik görüntüsü.



Şekil 2. Koil embolizasyonu sonrası koillerden birisinin proksimal kısmının sol ön artere taştığı görülmekte.



Şekil 3. Sol ön inen arterin proksimalinde koroner anjioplasti balonu şişirilip koilin proksimal ucu tamamen fistül içine yönlendirildikten sonraki sol kraniyal açıdan görünüm. Koilin proksimal ucu sol ön inen artere taşmıyor. LAD: Sol ön inen arter.

OPS4-40

Right coronary artery arising as a terminal branch of the left circumflex artery, leading to myocardial ischemia

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Case Report: A 36-year-old man was admitted to our cardiology outpatient clinic with complaints of exertional dyspnea and angina (NYHA class II). A treadmill stress testing was performed to the patient and ECG showed a substantial ST-segment depression in the inferolateral leads. These changes were interpreted as ischemia. Coronary angiography was scheduled for the patient. Coronary angiograms with radial approach revealed that right coronary artery (RCA) was not in a normal position, but that it was originating from the distal left circumflex artery (LCx) as a terminal branch, coursing to the right atrioventricular groove (Figure 1). As further investigation, coronary computed tomography angiography (CCTA) was scheduled for the patient. CCTA demonstrated a single coronary artery from the left sinus of Valsalva. An anomalous RCA was originating from the distal LCx, as a terminal branch, coursing to right, into the right atrioventricular groove (Figure 2). The coronary arteries were free of atherosclerotic disease.

Conclusion: We demonstrated a very rare type of single coronary artery anomaly form in which, RCA was not in a normal position, but that it was originating from the distal LCx as a terminal branch. In this case; exertional dyspnea may be partly secondary to the myocardial ischemia. As a possible mechanism; anomalous RCA may increase the load on LCx. Therefore, especially during prolonged physical activity, LCx could not satisfy the increased demand of blood flow for the myocardium, that can result a steal phenomenon. Treadmill stress test and CCTA are useful, non-invasive and widespread methods which can provide an accurate diagnosis and a complete anatomic and functional assessment for ischemia causing coronary anomalies.

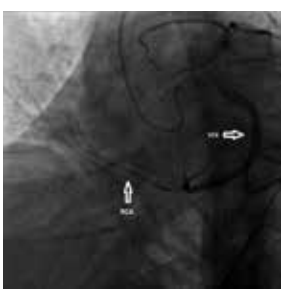

Figure 1. Coronary angiography showing single coronary artery.

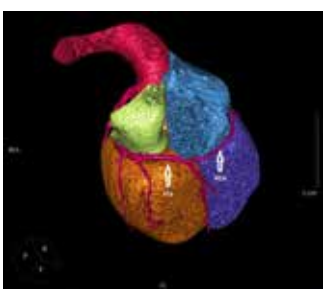

Figure 2. CT angiography showing single coronary artery.

OPS4-41

Ciddi radial arter spasmında yeni sheath çıkarma tekniği: Pasif traksiyon

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Altmış dokuz yaşında kadın hasta, göğüs ağrısı ve nefes darlığı şikayeti ile acil servise başvurdu. Özgeçmişinde hipertansiyon ve tip 2 diabet tanıları olan hastanın fizik muayenesinde her iki akciğerde yaygın ral mevcuttu. Kan basıncı (KB) 190/120 mmHg ve nabız 130 atım/dk saptanan hasta hipertansif akciğer ödemi olarak değerlendirildi. Elektrokardiyografi sinüs ritmi D1 ve aVL, V1-6 T negatifliği D3 ve aVF QS saptandı. Troponin (0.63-3.05 ng/ml) değerlerinde akut koroner sendrom ile uyumlu artış saptanması üzerine hasta koroner yoğun bakım ünitesine yatırıldı. Transtorasik ekokardiyografi'de EF= %35, anterior, anteroseptal ve apikal duvarlarda hipokinezi, hafif mitral yetersizliği, sağ atrium etrafında minimal perikardiyal efüzyon saptandı. Medikal tedavisi sonrası hemodinamisi düzelen hastaya yatışının 3. gününde sağ kol radial arterden 5F sheath (Primelife 5F, 7 cm) ile diagnostik koroner anjiografi uygulandı. Radial arter sheath yerleştirilmesi sonrası 200 mcg nitrogliserin ve 2500 IU unfraksiyone heparin uygulandı. Koroner anjiografi'de RCA non kritik darlıklar, CX orta segmente %80 ve LAD D1 bifurkasyonunda %95 darlık saptandı. Koroner girişim için hastanın 5F radiyal Sheath'i 6F radiyal Sheath (Terumo 6F 7 cm) ile değiştirilmesi planlandı, ancak 6F radial Sheath'in değişim teli (AZR 0.035-260 cm J tip PTFE coated guidewire) üzerinden değiştirilememesi nedeniyle, 6F Femoral sheath (Input 6F, 11cm) ile değiştirildi. Aynı seansta ek 6000 U unfraksiyone heparin uygulandı ve LAD'deki darlığa 2.0x15 mm balon predilatasyonu sonrası 2.75x18 mm ilaç kaplı (Endeavor) stent implante edildi. Tam açıklık sağlandı. İşlem sonrası radial sheath çıkarılmak istendiğinde sheath geri gelmedi. Antekubital fossa üzerindeki cilt derisinde sheath'in çekilmesi esnasında gerilme gözlendi ve hasta aynı zamanda ani kısa süreli şiddetli ağrı tarifledi. Sheath yıkanıp içerisinden nitrogliserin ve kalsiyum kanal blokeri verilmek istendi fakat sheath'ten kan geri gelmediği için uygulanamadı. Hastanın venöz damar yolundan IV nitrogliserin infüzyonu (KB 150/90 mmHg) başlandı. Katater laboratuvarında yapılan radial arter renkli Doppler incelemesinde damarın kontrakte olduğu ve sheath geri çekilmek istendiğinde radial arterde gerilme olduğu gözlendi. 15 dakika beklenmesine rağmen radial sheath geri alınamadı. Radial arter renkli Doppler tetkiki altında sheath'e pasif traksiyon (Sheath'in sabit güç uygulanarak geri çekilmesi) uygulandı. Yaklaşık 3 dakika sonra sheath'in yaklaşık 2-3 mm geri geldiği gözlendi. Pasif traksiyona devam edildiğinde toplam 10 dakika sonra sheath tamamen geri alınabildi. İkinci gün yapılan radial arter renkli Doppler incelemesinde hastanın radial akımının normal olduğu ve lümen içi patoloji olmadığı saptandı.

OPS5-01

ST elevasyonlu miyokard enfarktüsünün trombüs aspirasyonuyla tedavisi

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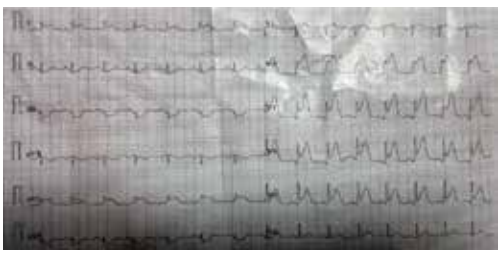
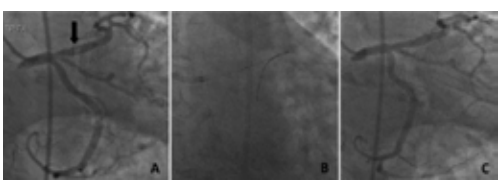
Akut koroner sendromun patofizyolojisi yaygın olarak plak rüptürü sonrası oluşan trombüs tıkacı ile karakterizedir. ST elevasyonlu MI koroner arterlerin tam tıkanması sonucu meydana gelir. Tedavinin temeli, oluşan trombüs tıkacının ortadan kaldırılmasıdır. Perkütan koroner girişim veya fibrinolitik tedavi ile tedavi edilebilir. ST elevasyonu ile başvuran vakamızda, stent kullanılmadan, trombüs aspirasyonu ile tama yakın rekanalizasyon sağlanmıştır. Altmış iki yaşında, sigara içicisi erkek hasta, sıkıştırıcı vasıfta olan göğüs ağrısının birinci saatinde acil serviste değerlendirildi. Başvuru EKG'si anterior miyokard enfarktüsü ile uyumlu idi. Hasta koroner anjiyografi laboratuvarına alındı. Göğüs ağrısı hafiflemeye başlamıştı. Yapılan koroner anjiyografide, LAD proksimalde trombüs izlendi. Trombektomi kateteri içerisinden 20 mg tPA yavaş infüzyonla intrakoroner uygulandı. Ardından, mükerrer manuel trombüs aspirasyonu uygulandı. İntrakoroner 8 mg tirofiban yapıldı ve idameye geçildi. TIMI-3 akım sağlandı. İşlem sonrası trombüs izlenmedi. Kontrol EKG'de ST elevasyonunun kaybolduğu izlendi. Yoğun bakım takiplerinde problem izlenmedi. Medikal tedavisi düzenlenen hasta taburcu edildi. Kiloya göre enoksaparin tedavisi 8 güne tamamlandı. Son yapılan klinik çalışmalarda rutin manuel trombüs aspirasyonunun kullanımı önerilmemektedir. Ancak trombüs yükünün fazla olduğu hastalarda kullanılabileceği belirtilmiştir. Biz de vakamızda intrakoroner litik tedavi sonrası trombüs aspirasyonu yaparak tama yakın açıklık sağladık.



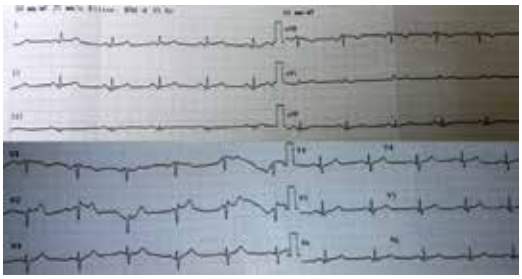
Şekil 1. Hastanın başvuru sırasında ki EKG'sinde D1-aVL ve V1-6 da ST elevasyonu ve inferiyor derivasyonlarda resiprok ST çökmeleri izlenmektedir.



Şekil 2. Koroner anjiyografi görüntüleri gösterilmektedir. **(A)** LAD proksimalindeki trombüs ok işareti ile gösterilmektedir. **(B)** Trombüs aspirasyonu gösterilmektedir. **(C)** İşlem sonrası LAD proksimalindeki trombüsün ortadan kalktığı izlenmektedir.

Girişimsel kardiyoloji / Koroner

Şekil 3. Koroner anjiyografi sırasında yapılan trombüs aspirasyon materyali gösterilmektedir.



Şekil 4. Koroner anjiyografi sonrası hastanın EKG'sinde ST rezolüsyonu izlenmektedir.

OPS5-02

Acute catastrophic intracoronary thrombus formation due to nonionic contrast media: Iopamidol

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A 78-year-old female patient with chest pain was referred to our clinic for coronary angiography. A 6F femoral sheath was inserted. However, 6F JL4 catheter wasn't easily advanced to the aortic root due to serious tortuosity of right external iliac artery. Moreover, the origin of left main coronary artery was more posterior than usual. So the catheterization of left main was difficult and 8 or 9 puff of iopamidol was given to find the origin and catheterize left main. When the first projection was recorded, thrombi were seen in proximal LAD, in first diagonal branch and in the proximal circumflex artery which weren't limiting the coronary flows (Figure 1). RCA couldn't be catheterized due to poor manuplation of JR4 and a new sheath cannulation was decided via left femoral artery. The patient experienced nausea and vomitting during left femoral artery cannulation. The blood pressure of the patient was 75/52 mmHg and the rhythm was sinus with 108 bpm. ECG was checked and 2 mm ST segment depression was detected in leads DII and DIII on monitor. IV bolus of 500 ml saline was given and left femoral artery cannulation was completed immediately. The patient described an increase of her chest pain and 3 mm ST segment elevation in leads DII and DIII was seen on the monitor. LM coronary artery was catheterized rapidly via left femoral sheath and a catastrophic picture was recorded (Figure 2). Cx was totally thrombotic and no coronary flow was detected. Predominantly LAD but also left main were thrombotic too, nevertheless, there was TIMI III flow in the LAD and the left main. UFH of 5000 unit (100U/ kg) was injected IV and cardiovascular surgery consultation was asked. After a short discussion with surgeons, percutaneous mechanical aspiration and medical treatment were agreed on. A bolus dose of tirofiban was injected slowly into left main and the maintenance dose was given IV for 72 hours. Beside these medical treatments, a 0.014 inch floopy guide-wire was advanced through Cx and thrombus aspiration catheter was advanced over the guide-wire. Three times of aspiration were performed but Cx coronary flow couldn't be achieved. Then thrombolytic therapy was decided as a second treatment option owing to failure of thrombus aspiration. Fifteen mg of tPA was administrated into left main and 37.5 mg in 30 minutes IV and 25 mg in one hour. UFH of 1000 U/hr was also given IV as a maintenance dose for 48 hours. Aspirin 300 mg and clopidogrel 75 mg were also given daily. After 4 days hospitalization, a control CAG was performed with ionic contrast media, ioxithalamate. Normal coronary arteries was shown with no thrombotic residue (Figure 3). The patient was discharged with aspirin 100 mg daily.

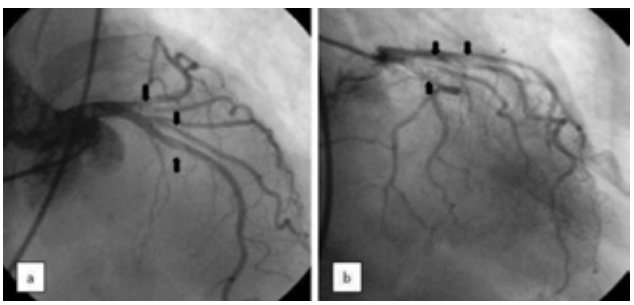

Figure 1. Left coronary angiogram **(A)** Right anterior oblique cranial view, **(B)** Right anterior oblique caudal view (thrombi are depicted by black arrows).

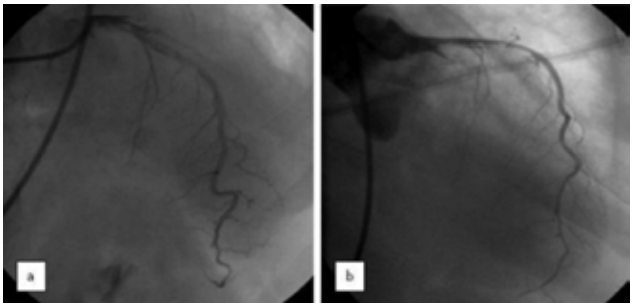

Figure 2. Left coronary angiogram **(A)** Right anterior oblique craniel view, **(B)** Right anterior oblique caudal view (total occlusion of circumflex with thrombus and TIMI III flow of LAD with thrombus load).

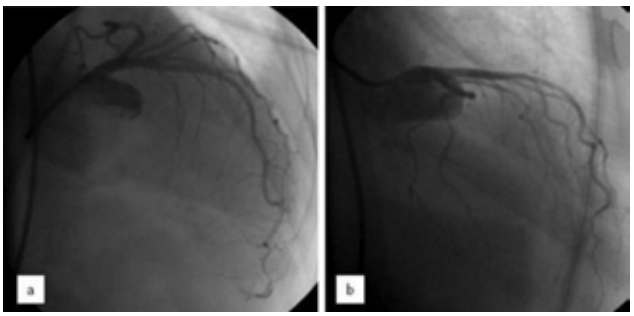

Figure 3. Left coronary angiogram **(A)** Right anterior oblique craniel view, **(B)** Right anterior oblique caudal view (all thrombus load resolved after medical theraphy and no thrombus residue).

OPS5-03

Percutaneous closure of left anterior descending coronary artery – pulmonary artery fistula using coils

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Coronary artery fistulas are anomalous connections between one or more coronary arteries and a cardiac chamber or a large vessel. They are usually congenital origin and most of patients are asymptomatic. 63-year-old male patient was admitted to our outpatient clinic with the complaints of chest pain. On physical examination, blood pressure was 130/80 mmHg, heart rate was 84/min. Cardiac auscultation revealed apical 2/6 systolic murmurs. Transthoracic echocardiography showed normal wall motion and mild mitral regurgitation. Exercise electrocardiography was positive. Coronary angiography revealed the left anterior descending coronary artery to pulmonary artery fistula (Figure 1a, video). Coronary artery fistulas can result in myocardial ischemia, heart failure, and bacterial endocarditis. The cause of myocardial ischemia is the coronary steal phenomenon. Symptomatic coronary artery fistulae should be treated with percutaneous intervention or surgical correction. Depending on the morphology of the fistulas, the most suitable occluder device should be selected including coils, vascular plugs or a patent ductus arteriosus device or muscular ventricular septal defect device. We planned to perform percutaneous coil embolization of the fistula because of myocardial ischemia. The three coil were placed in the body of the fistula (Figure 1b, Video). The final coronary angiography did not show any residual flow and procedure was terminated without complication. Percutaneous intervention can cause serious complications such as coil migration and distal embolization. Therefore, these patients should be closely monitored especially in patients with a large fistula and a high-flow shunt. We presented herein a case of successfully performed percutaneous coil embolization of a coronary artery fistula using the three coils.

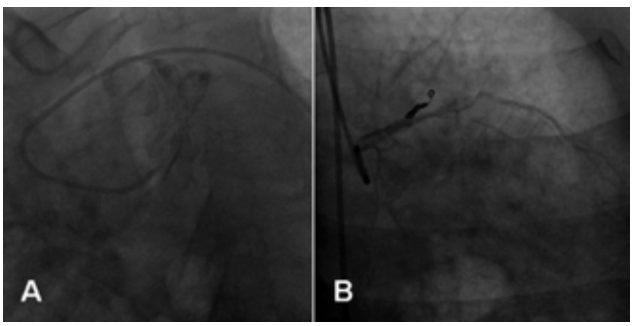

Figure 1. The left anterior descending coronary artery to pulmonary artery fistula **(A)**. The three coil were placed in the body of the fistula **(B)**.

Girişimsel kardiyoloji / Koroner

OPS5-04

Koroner iskemi oluşturan koroner arteriyel fistül

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KTO Karatay Üniversitesi Medicana Tıp Fakültesi Kardiyoloji Bölümü, Konya

Altmış bir yaşında erkek hasta eforla göğüste ağrı şikayeti ile değerlendirildi. Hastanın bilinen hipertansiyon öyküsü mevcut aile öyküsü yok. Hastanın ekg sinde sinüs ritmi tespit edildi. Hastanın ekokardiyografisinde ek patoloji tespit edilmedi. Hastaya dobutamin stres ekokardiyografi yapılması planlandı. Hastanın yapılan dobutamin stres ekosunda düşük doz ve pik dozda inferior, inferior septum ve lateral duvarda iskemi tespit edildi. Hastaya koroner anjiyografi yapıldı. Hastanın koroner anjiyografisinde LAD diagonal 1 ve RCA nın pulmoner artere fistülize olduğu tespit edildi. Fiztülize olan koroner arterlerin beslediği yerler ile dobutamin stres ekonun iskemi gösterdiği yerler benzer yerler olması iskeminin sebebinin fiztülizasyona bağlı koroner çalmaya bağlı olduğu düşünüldü. Dobutamin stres ekonun iskemi tespitindeki hassasiyeti önem arz etmektedir. Dobutamin stres ekoda fiztülizasyon olan bölgede iskemi tespit edilmesi koroner çalmanın bu hastada ne kadar etkili olduğunu göstermiştir. Hastada iskemi oluşturması sebebiyle koroner fistülizasyonun kapatılması planlanmıştır.

OPS5-05

Successful treatment of a rare congenital coronary anomaly with percutaneous technique in a patient with non ST elevation MI

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We present a 55 year-old female patient who applied to our clinic with the complaint of angina (CCS 3). Her exercise capacity was New York Heart Association (NYHA) Class 3. She had the history of coronary artery disease,hypertension, hyperlipidemia,and a 15 pack-year history of smoking. On physical examination, bilateral respiratory rales, 2+ bilateral pitting pretibial edema and irregular pulse were detected. On electrocardiographic examination, minimal ST depression on anterior chest derivations was detected. Moderate depression of left ventricle systolic function and dilatation of the left ventricle were also showed on transthoracic echocardiography. Coronary angiography was performed. Left coronary artery was not visualized at the left coronary sinus. During the visualization of the right coronary artery, it was seen that both right coronary artery and left main coronary artery were originated from the right coronary sinus and from a single ostium. This was confirmed by angiography (Figure 1). Afterwards, we implanted drug eluting stent fort the lesion on proximal portion of the left anterior descending artery. The result was excellent. Later we performed multi-slice computer tomography (MSCT) which showed that both left and right coronary artery originated from the right coronary sinus and from a single ostium without compression by the great arteries (Figure 2). In conclusion we showed that it is possible to treat a congenital coronary anomaly like ours successfully and we confirmed the success of the percutaneous intervention with MSCT angiography.

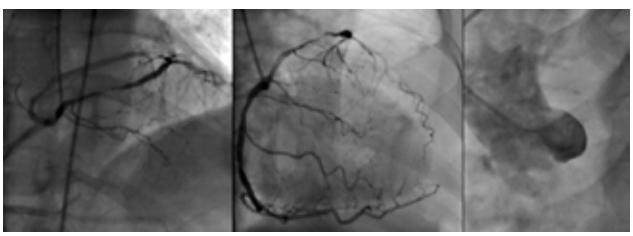

Figure 1. The appearance of the origination of left coronary artery from the right coronary sınus in coronary angiography.

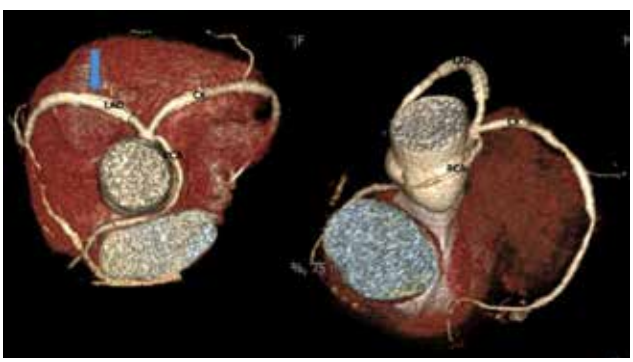

Figure 2. Multislice computed coronary tomographic angiography demonstrated that both left and right coronary arteries originated from the same right coronary sınus. Abbreviations: LMCA: Left main coronary artery, LAD: Left anterior descending artery,RCA: Right coronary artery, LV: Left ventricle, LA: Left atrium, Ao: aorta.

OPS5-06

Primary successful management with conventional methods of developing coronary perforation during coronary intervention

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Aim: We present 72-old-women who had coronary perforation during primary percutaneous coronary procedures developed treated with successful conventional methods

Methods: A 72-year-old woman patient with known history of hypertansion and coronary bypass graft operation was admitted to the emergency departmant with chest pain and vomiting. Blood pressure and heart rate were 80/55mmHg and 98 bpm respectively. In the emergecy department ECG showed ST segment elevation leads D2, D3, aVF.

Results: An immediate coronary angiography was performed with the diagnosis of inferior miocardial infarction. Unknown history of graft the patient 's totally occluded native RCA and RCA saphenous graft was observed aortography totally occluded.RCA saphenous graft lesions in patients who can not pass had decided attempts native RCA. The lesion was passed with extrastiff wire and ballooned. After them drug eluting stent was implanted. When angıography performed it was observed pericardial effusion in the area that kept opaque (Ellis type 2) (Figure 1a, b). Because we have no graft stent percutaneous coronary balloon advanced and inflated in 3 minutes. Distal RCA was total occluded. During follow-up there was no leakage from RCA. After 24 hours control angiography, TIMI 3 flow is observed and distal RCA was haisy. Drug eluting stent implanted to haisy area (Figure 2a, b). In follow-up hemodynamically stable and no increase in effusion patients were discharged by healing.

Conclusion: The therapeutic alternatives for coronary artery perforation include prolonged inflation with either the angioplasty balloon or a perfusion catheter as soon as the perforation is recognized to prevent further blood extravasation. Perfusion balloons having a perfusion lumen communicating with the blood vessel lumen will occlude the hole, while attempting to seal the defect, permit distal vessel perfusion and reduce the ischemia during the prolonged inflation. Stent grafts for emergency implantation in case of coronary artery perforation must be an obligatory inventory of catheterization laboratories. The coated stent is a safe and effective alternative that can be used for sealing a major coronary artery perforation and obviating cardiac surgery. The utilization of synthetic graft stent is less invasive, faster, and more effective when compared to surgical interventions and is generally considered to be the gold standard in the management of coronary artery perforation. Other alternatives include emergency bypass surgery for drainage and artery repair. As in case, to be thrombosed of proximal perforation in the absence of surgical facilities and the graft stent in coronary perforation is a method that can be used.

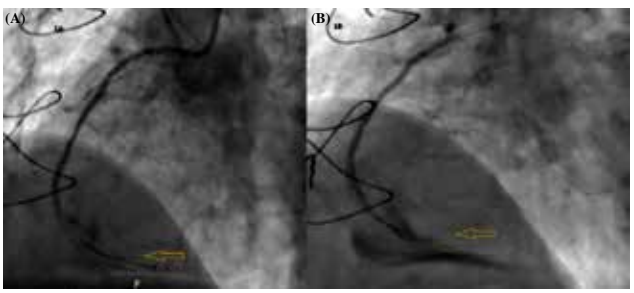

Figure 1. (A, B) Ellis type 2 perforation.

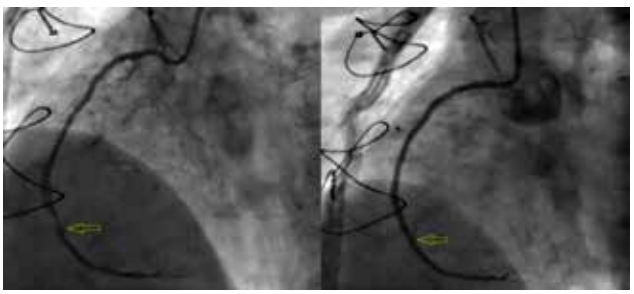

Figure 2. (A) Haisy image of perforation. **(B)** After stent implantation to coronary perforation.

OPS5-07

Skipped right coronary artery osteal lesion resulted in atrioventricular block and cardiopulmonary arrest

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Coronary osteal lesions necessitate careful eyes to be detected by invasive cardiologists. Skipping coronary osteal stenosis may result in broad range of consequences, from acute coronary syndromes to cardiopulmonary arrest. Here we present 52 year old female patient who was admitted to our emergency department with acute inferior myocardial infarction. Patient was rapidly taken to the catheter laboratory for intervention. Distal right coronary artery subtotal lesion was detected therefore percutaneous coronary interventions was administered to the culprit lesion (Figure 1). The last view of the right coronary artery revealed no more other lesions. Patient was transferred to the coronary care unit for follow-up. During the follow up period patient defined newly onset chest pain subsequent to atrioventricular block. Second coronary intervention started under cardiopulmonary resuscitation. Transient pacemaker was placed to the right ventricle from femoral vein access. The responsible lesion was thought to be stent thrombosis whereas right coronary arterial osteal lesion was detected. Right coronary artery osteal stenosis was opened successfully by stent implantation (Figure 2). After an hour patient was in no need to transient pacemaker. She was successfully discharged on her 5th day under medical treatment. Our case gets attention to the osteal lesions which can be skipped in acute myocardial infarction due to extensive spasm secondary to thrombogenic environment.

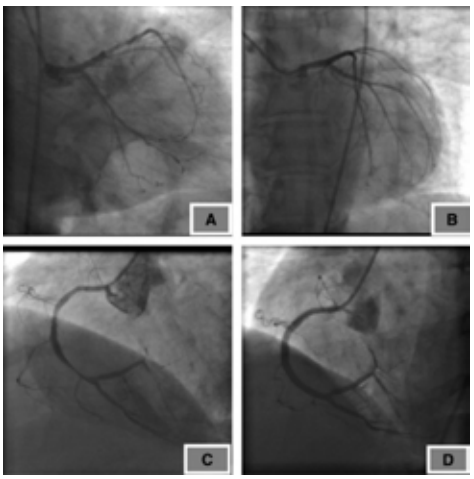

Figure 1. (A, B) Left anterior descending and left circumflex artery without stenosis. **(C)** distal right coronary arterial lesion was shown **(D)** last view after percutaneous coronary intervention to the right coronary artery.

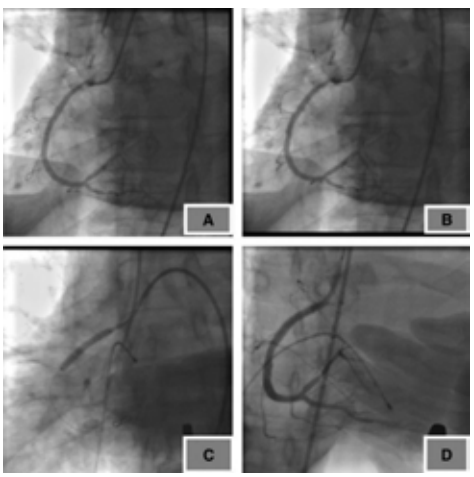

Figure 2. (A, B) Showing right coronary artery osteal lesion. **(C)** During stent implantation to the coronary osteal stenosis. **(D)** Showing last view after percutaneous coronary intervention.

OPS5-08

Dynamic left ventricular outflow tract obstruction after aortic valve replacement

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68 year-old female patient underwent aortic valve replacement with a 21 mm St. Jude Medical (SJM) mechanical valve because of severe aortic stenosis. Pre-operative echocardiographic assessment showed calcific and thickened aortic leaflets and there was at the left ventricular outflow tract (LVOT) peak gradient of 5 mm Hg (Figure 1a) and a transvalvular peak and mean gradient of 96 and 63 mmHg was measured by continuous doppler (CW) (Figure 1b). After aortic valve replacement (AVR), the patient transferred to intensive care unit and on following period, the patient was developed hypotensive and her blood pressure was 70/50 mm hg and pulse rate was 130 bpm. Dopamine infusion added her medical treatment and the patient remained severe hypotensive after dopamine treatment. Transthoracic echocardiog-raphy was performed to explain severe hypotensive in despite of inotropic agents. The bedside echocardiography showed an abnormal turbulent flow velocity at the LVOT and CW doppler of the LVOT showed a late peaking gradient (Dagger shaped pattern)

Kardiyak görüntüleme / Ekokardiyografi

(Figure 2, Supp. video 1). Peak and mean gradients across the prosthetic aortic valve were 40 and 21 mmHg (Figure 3a) and CW doppler study of LVOT showed a peak gradient of 69 mmHg with the Valsalva maneuver (Figure 3b). Dynamic LVOT obstruction was suspected and then the patient was started on beta-blocker treatment and given intravenous 0.9% saline fluid replacement. After treatment, the patient`s condition improved and her blood pressure rose to 120/72 mmHg. Echocardiographic evaluation performed after these treatment, it showed disappearance of the preexisting dynamic LVOT obstruction (Supp. Video 2).

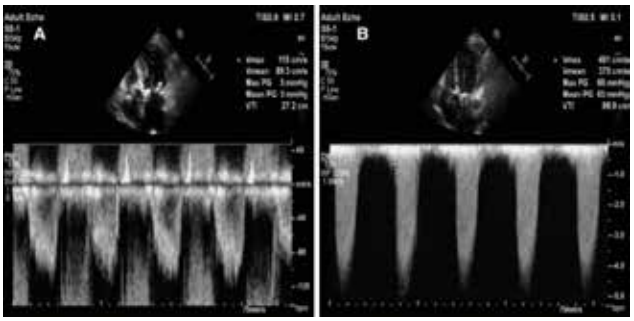

Figure 1. (A, B) Pre-operative transthoracic echocardiography shows LVOT and transvalvular aortic gradients.

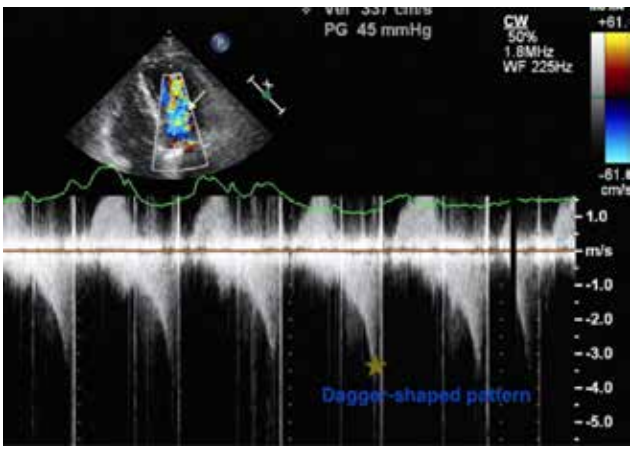

Figure 2. CW doppler study of LVOT shows a late peaking gradients (Dagger shaped pattern).

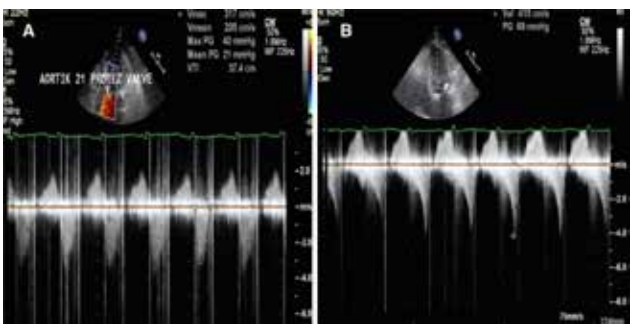

Figure 3. (A) CW doppler study shows peak and mean gradients across the prosthetic aortic valve. **(B)** CW doppler study of LVOT shows a peak gradient of 69 mm Hg with the Valsalva maneuver.

OPS5-09

The incidental diagnosis of a biventricular apical non-compaction case

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[2]*Department of Radiology, İstanbul University İstanbul Faculty of Medicine, İstanbul*

38 year old male patient was admitted to our outpatient department because of atypical chest pain. His medical history was unremarkable and physical examination was completely normal. ECG showed non specific ST-T changes. The results of routine biochemical tests were within normal limits. Exercise stress test result was negative in terms of ischemic heart disease. Transthoracic echocardiography was performed and apical trabeculation of left and right ventricule were detected (Figure 1). The blood flow into the intertrabecular recesses could be visualized by color Doppler (Figure 2). The ratio of non-compacted myocardium to compact myocardium at the end of systole was > 2:1. Left ventricular ejection fraction was in normal range as 50% and tricuspid annular plane systolic excursion was 2.1 cm. Morphologically, the heart valves were normal, and no coexisting congenital anomaly was found. These findings consisted with isolated biventricular apical non compaction. We performed cardiac magnetic resonance imaging which also confirmed our echocardiographic diagnosis. The patient had no significant arrhythmias during his 24-h Holter monitoring. There was no clinical or radiological embolic events for our patient. We decided to follow up our patient at regular intervals closely and to perform a echocardiographic screening programme for his first degree relatives. Myocardial non-compaction is a relatively rare congenital disorder characterized by prominent trabeculations and intertrabecular recesses that communicate with the ventricular cavity. According to echocardiographic studies, reported prevalence is between 0.014 and 1.3% in the general population. It is thought to be caused by intrauterine arrest of compaction of the myocardial fibres. The clinical presentations are ranging from asymptomatic patients to patients who develop arrhythmias, thromboembolism, heart failure, and sudden cardiac death. Differential diagnosis with thrombi, false tendons, apical hypertrophic cardiomyopathy, fibroma, obliterative processes, intramyocardial hematoma, cardiac metastases and intramyocardial abscesses must be considered In patients with myocardial non-compaction. Treatment options differs on an individual basis, ranging from regular follow up, medical management in mild cases to heart transplantation in patients with refractory symptoms. In this case report we aimed to suggest the diagnosis of suspected myocardial noncompaction should be carefully evaluated by imaging methods to avoid inappropriate and exaggerated diagnoses.

OPS5-10

Tamponadı taklit eden mediastinal kitle

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[2]*S.B. Ümraniye Eğitim ve Araştırma Hastanesi, Radyoloji Kliniği, İstanbul*
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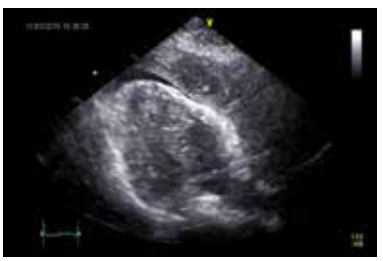
Mediastinal kitlelerin %50'si asemptomatik olup, sıklıkla nefes darlığı, öksürük, yutma güçlüğü, ses kısıklığı, hemoptizi gibi semptomlarla gelebilirler. Bu yazımızda, tamponad kliniği ile gelen fakat mediastinal kitle saptadığımız olguyu sunmak istedik.

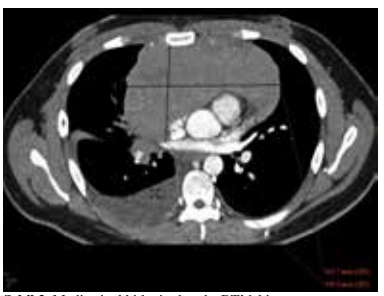
Olgu Sunumu: On dokuz yaşında erkek hasta, iki gündür olan nefes darlığı, ateş yüksekliği, öksürük ve çarpıntı şikayeti olması üzerine başvurdu. Fizik muayenede, her iki juguler venöz dolgunluk belirgin, S1, S2 ritmik, taşikardik, kardiyak sesler derinden geliyordu. Solunum sesleri sağ alt bazalde azalmış, batın rahat, pretibial ödem yoktu, periferik nabızlar açık idi. Laboratuar bulguları normaldi. Elektrokardiyografi: Normal sinüs ritmi, hız 116 dk/atımdı.Transtorasik ekokardiyografi:Kalp boyutları ve kapak yapıları normal, kalp etrafında kalbi çepecevre saran, en geniş yerinde 1 cm' i geçmeyen ve kollapsa neden olmayan perikardiyal mai, perikard dışında, doku dansitesinde en geniş yerinde yaklaşık 10 cm ölçülen kitle imajı veren lezyon izlendi. Bu kitlenin tamponadmış gibi sağ kalp boşluklarına bası bulgusu yaptığı izlendi (Şekil 1). Thoraks BT'de, sağda en geniş yerinde 41 mm olan plevral effüzyon, anterior mediastende vena cava superioru belirgin daraltan (120x170 mm) boyutlu, koronal planda kraniokaudal uzunluğu 173 mm ölçülen, yumuşak doku dansitesinde kitle lezyonu ve kitlenin izlenebildiği kadarıyla sağ atriyuma basısı izlendi (Şekil 2). Mediastendeki kitleden torakoskopi yardımıyla biyopsi alındı, biyopsi sonucunda hastaya akut lenfoblastik lösemi teşhisi konuldu. Kemotreapi ve radyoterapi tedavisi başlandı. Takiplerinde tedavi sonrası kitlede küçülme ve semptomlarda gerileme görüldü. Hastamız halen hematoloji tarafından takip edilmektedir.

Tartışma: Mediasten kitlelerinin büyük çoğunluğunu (%80) ikincil kitleler oluştururken, birincil kitleler (%20) daha az orandadır. Yaş gruplarına bakıldığında birincil mediastinal kitleler en sık 20-30 yaş aralığında ve erkek cinsiyette daha sıktır. İnsidans 4-9/10000 olarak raporlanmıştır. Mediastinal kitleler en sık anteriyor (%54-57) yerleşimlidirler. Anteriyor mediasten kitleleri genellikle timoma, lenfoma,teratom ve germ hücreli tümörlerdir. Hastamızın lezyonu anteriyor mediastinal bölgedeydi ve sağ atriyuma bası yapmaktaydı. Mediastinal lezyonlar sıklıkla komşu kompartmanlara doğru büyürler ve çevre dokuların etkisine bağlı olarak şekil alırlar. Orta mediastendeki tümörler de tamponada neden olabilirler. Hastamızda da kitle epikardiyuma bası yaparak, tamponadı andıran klinik bulgulara ve Vena Cava Süperior Sendromu gelişmesine neden olmuştu. Mediasten kitlelerinde tanı genellikle başka bir nedenden dolayı yapılan radyolojik incelemeler sonucu konulmaktadır. Olgumuzda perikardial tamponad ön tanısıyla transtorasik ekokardiyografi uygulanarak kitle saptanmıştı.

Sonuç: Kardiyak tamponad kliniği bulunan, ekokardiyografide perikardialsıvı miktarı az olan hastalarda perikard etrafındaki oluşumlar dikkatli değerlendirilmelidir.



Şekil 1. Ekokardiyografide kitlenin görünümü.



Şekil 2. Mediastinal kitlenin thoraks BT'deki görünümü.

OPS5-11

Infective endocarditis involving aortic, mitral and tricuspid valves in a young patient

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Department of Cardiology, Gülhane Military Medicine Academy, Ankara

Infective endocarditis (IE) has a distinct nature that it remains a diagnostic challenge. Infective endocarditis should be suspected in a variety of very different clinical situations. It may present as an acute, rapidly progressive infection, but also as a subacute or chronic disease. 34-year-old male patient was admitted to our coronary care unite with the cardiogenic shock. His medical history did not show any clinical feature. On physical examination, blood pressure was 60/30 mmHg, heart rate was 134/min. Cardiac auscultation revealed apical 3/6 systolic and aortic 2/4 diastolic murmurs. Transthoracic echocardiography showed destruction of aortic valve, severe aortic regurgitation, moderate mitral regurgitation. Transesophageal echocardiography short axis, three chamber long axis and four chamber long axis view revealed aortic non-coronary cuspis destruction (asterisk, 1b), severe aortic regurgitation (1D) and vegetations (arrows, 1a, b, c) (Video 1, 2 and 3). He died within two hours of admission despite all intervention. Echocardiography, either transthoracic or transesophageal echocardiography, plays a crucial role in the diagnosis, management and monitoring of IE patients. Echocardiography must be done as soon as IE is suspected. The in-hospital mortality rate of patients with IE varies from 15% to 30%. Patients with heart failure, periannular complications and/or S. aureus infection are at highest risk of death. When three of these factors are present, the risk reaches 79%. In our case, he had these three risk factors. We presented herein a case of infective endocarditis involving aortic, mitral and tricuspid valves in a young patient.

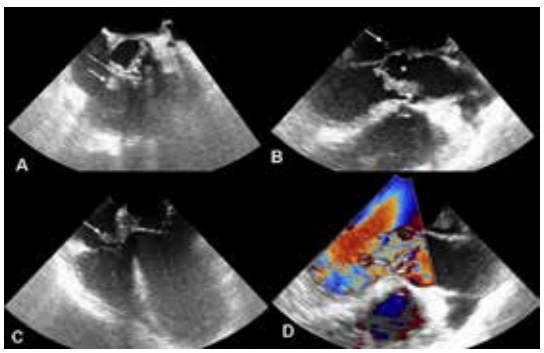

Figure 1. Transesophageal echocardiography short axis, three chamber long axis and four chamber long axis view revealed aortic non-coronary cuspis destruction **(asterisk, 1B)**, severe aortic regurgitation **(1D)** and vegetations **(arrows, 1 A, B, C).**

OPS5-12

A left sinus of valsalva aneurysm: A rare cause of dyspnea

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Sinus of Valsalva aneurysms (SVAs) are very rare cardiac malformations with left SVA being the rarest of all SVAs. Small and unruptured left SVAs are usually clinically silent and often discovered incidentally on imaging studies while they may be associated with aortic regurgitation (AR), ventricular septal defects, and infective endocarditis. We report a case of an unruptured left SVA causing exertional dyspnea. A 70-year-old hypertensive female patient was admitted to cardiology outpatient unit for preoperative evaluation of noncardiac surgery. She had exertional dyspnea for one year. Cardiac examination revealed normal S1, soft S2, and grade II/VI early diastolic murmur in the left parasternal area. Laboratory studies were within normal range. The resting electrocardiogram showed normal sinus rhythm. Transthoracic echocardiogram revealed normal left ventricular size and function with an ejection fraction of 60%, moderate aortic root dilatation at the level of valsalva, and aneurysmal dilatation of the left sinus valsalva (Figure 1). There was moderate AR and mitral regurgitation. Computed tomography angiography (CTA) demonstrated saccular dilatation at the level of the left sinus of valsalva, measuring 23x34 mm while left main coronary artery originated superiorly from the anterior wall of the aneurysm (Figure 2). Since the patient had dyspnea which was thought to be associated with left SVA and concomitant moderate aortic regurgitation, surgery was indicated. However, the patient refused surgery or any percutaneous approach. She was given antihypertensive treatment and was followed with regular echocardiography controls which showed no signs of further increase in size of the aneurysm or rupture. Unruptured SVAs are usually asymptomatic for decades and discovered incidentally on echocardiography. They are usually treated with surgery especially if they are large and associated with complications or symptoms. Follow-up of our case might give us idea for prognosis of SVAs which are left untreated.

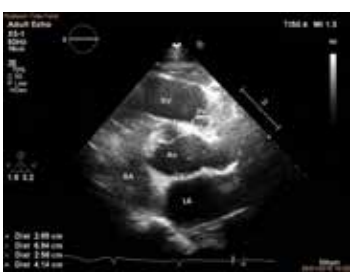

Figure 1. Parasternal short axis view showing moderate aortic root dilatation at the level of valsalva, and aneurysmal dilatation of the left sinus of valsalva (Ao: ascending aorta, LA: left atrium, PV: pulmonary valve, RA: right atrium, LA: left atrium).

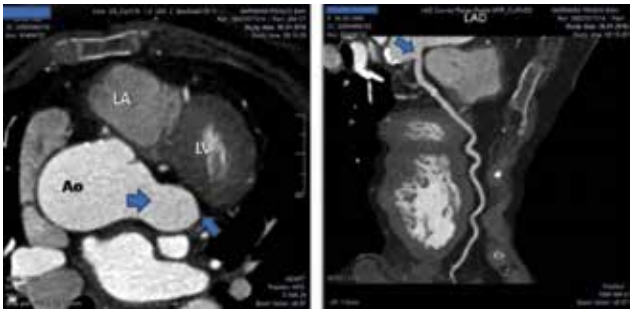

Figure 2. Computed tomography angiography. Arrows in the left demonstrating saccular dilatation at the level of the left sinus of valsalva while the arrow in the right showing left main coronary artery originating from the anterior wall of the aneurysm (Ao, ascending aorta, LA: left atrium, LV: left ventricle).

OPS5-13

A giant thrombus resembles myxoma

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A -50-years-old female presenting with cerebrovascular events and acute anteroseptal heart attack admitted to the emergency department. The electrocardiogram showed normal sinus rhytm and st elevation in V2-V3 lead. The computed tomography (CT) scan of the patient revealed acut focal ischemia at the right frontal lobe. The transthoracic echocardiography (TTE) showed a large mobile left atrial mass of size 4.2x1.5 cm. The mass was freely mobile in different planes and was free floating. It was prolapsing in to the mitral valve till the tips of the mitral valve leaflets from the left atrium but was not prolapsing across the mitral valve. In the foreground, TTE findings were consistent with like an atrial myxoma. For further evalatıon a tranesophageal echocardiography (TEE) was done. The TEE showed the presence of 4.2x1.5 cm measurıng nonhomogeneous mass filling in the left atrial appendix and the presence of a 4x2 cm measuring partially free floating in the left atrium but attached to the main mass by a thin bridge. In addition TEE demonstrated moderately dilated LA and the mitral valve displayed moderate regurgitation (functional). Further investigation, the TEE showed segmental motion abnormality in apex. The coroner anjiography showed the distal left anterıor descending artery (LAD) was occluded by a thromboembolism. The patient shortly underwent surgical removal of the mass. The prımary operatıve findings was a spherical purple gelatinosus fragmented mass. Excision of the mass includıng the shaft was performed, followed by suture ligation of the LAA. Histopathologıcal exemination confirmed a layered, partly necrotic mostly organized thrombus. Further investigation, genetic thrombosis panel was negative. The probable predisposing factors for thrombosus formation are mitral valve disease, non-valvular atrial fibrillation, thrombogenic gene mutations, severe left ventricular dysfuntion and other causes atrial contractile failure. The case presented here in is interestıng because left atrial giant trombüs is extremely rare in the absence of trombogenic risk factors.

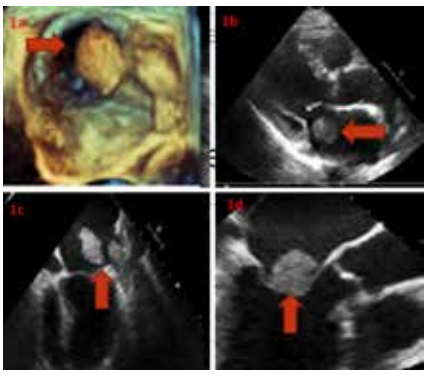

Figure 1. (A) Three dimensional TEE showing huge thrombosis filling in the left atrial appendix and highly mobile part attached to the main mass by a thin bridge. **(B)** Echocardiogram showing a non-homogenous spherical mass in the left atrium,simulating myxoma. **(C)** TEE showing a round mass in the left atrium and a big mass filling tle left atrial appendix. **(D)** TEE view (120 degree) showing a non-homogenous spherical mass prolapsing in to the mitral valve till the tips of the mitral valve leaflets from the left atrium.

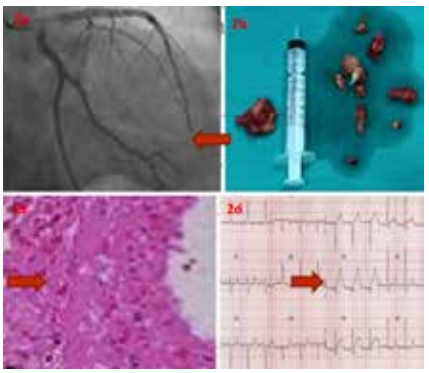

Figure 2. (A) Coronary angiography showing distal occlusion of left anterıor descendıng artery (LAD) by a thromboembolism. **(B)** Macroscopic image of the left atrial thrombus. **(C)** Histopathological image of the left atrial thrombus. **(D)** Electrocardiography showing acute st elevation in v2-v3 lead.

OPS5-14

Huge pericardial cyst impairing right ventricular filling

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A 42 year-old man admitted to our out-patient clinic with the complaint of mild exertional dyspnea. He was a heavy smoker and had a previous history of coronary artery disease. In his physical examination heart sounds were almost inaudible and respiratory sounds were markedly reduced with evident rhonci in all lung fields. In chest x-ray, aside from pulmonary parenchymal abnormal findings, a mass adjacent to right ventricule was noted (Figure 1a). In echocardiographic examination, a large pericardial cyst -impairing the relaxation of not only right atrium but also right ventricular base- was observed in transapical and subcostal views (Video 1, Video 2 and Figure 1b). A significant respiratory variation was detected in tricuspid in-flow Doppler recordings. Mitral in-flow was also effected mainly due to constriction of right ventricular filling by the cyst, thus an undulation in preload and a slight ventricular interdependence was established (Figure1c). Left ventricular volumes and functions were preserved. Inferior vena cava collapse during inspiration was in normal limits. Multislice CT scan confirmed the diagnosis. A contrast-free pericardial cyst (127x68x100 mm) was located at the right cardiophrenic angle (Figure 1d). Diffuse paraseptal and centrilobular emphysematous regions, pleuroparenchymal bands and nodular lesions were also detected in pulmonary parenchymal sections. The patient was discussed by local heart team and due to huge size of the cyst and reduced filling properties, a consensus on the necessity of surgical or percutaneous intervention was achieved. Unfortunately, he refused these treatment options. Pericardial cysts are rare abnormalities of mediastinum. Patients are mostly asymptomatic but excessive size, suspicion about malignant disease and local complications may raise the need of intervention.

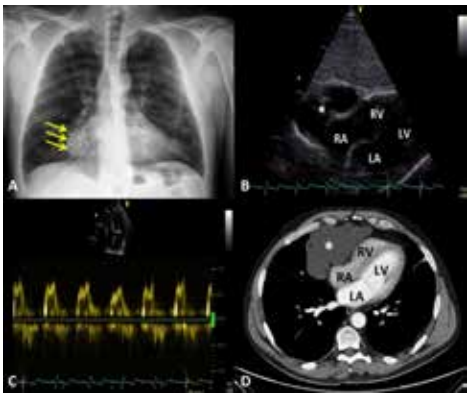

Figure 1. An elliptical mass adjacent to right ventricle **(arrows, A)**. Pericardial cyst demonstrated with transthoracic echocardiography from subcostal view **(star, B)**. Significant respiratory variation in mitral in-flow Doppler recordings **(C)**. A contrast-free pericardial cyst located at the right cardiophrenic angle **(D)**.

OPS5-15

Treatment of mitral bioprosthetic valve with unfractionel heparine

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A 75 year old woman admitted to our hospital with a complaint of progressively worsening shortness of breath for 2 weeks. She had bioprosthetic mitral valve replacement five years ago. On physical examination arterial blood pressure was 130/90 mmHg. Her heart rate was 120 bpm. An ECG showed atrial fibrillation. Routine blood investigations were normal and international normalized ratio was 1.6. Transthoracic echocardiography (TTE) revealed normal ejection fraction maksimum and mean gradient 10/5 mmhg across the mitral bioprosthetic valve. Transesosephageal echocardiography (TEE) revealed 17 mm hypermobile and 5-6 mm immobile thrombus on the bioprosthetic mitral valve (Panel A, B). The patient underwent to treatment with UFH for 17 days. Target of activated parsiyel platelet time was 50-70 second.Follow-up TTE and 2D-3D TEE was caried out. 2D and 3D TEE showed well functioning mitral valve prosthesis and remnant 3 mm mobil trombus on mitral bioprosthetic valve (panel C, D).The patient was discharged with warfarine to target INR 2.0-3.0. Her outcome there was no evidence of embolic complications or bleeding.

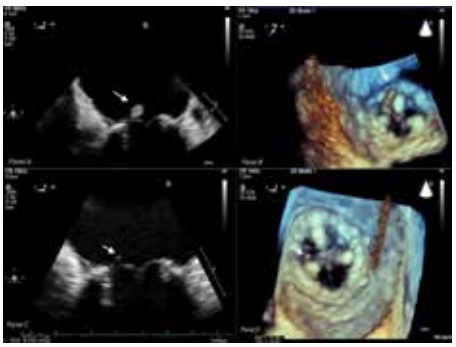

Figure 1.

OPS5-16

Nadir bir olgu: Sol atriyal miksoma sanılan, metastatik malign mezenkimal tümör

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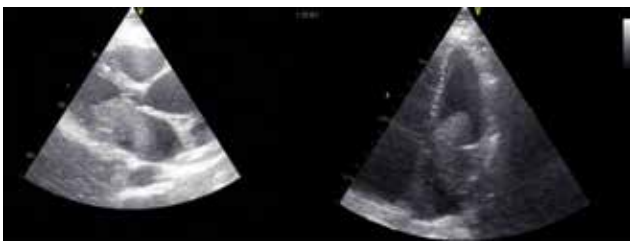
Primer kardiak tümörler nadir olup 10000 hastada 1-3 sıklık bildirilmiştir. Kalbin metastatik tümörleri ise sıklıkla akciğer, meme, malign melanom, lösemi ve lenfomadan kaynaklanmaktadır. Genellikle metastatik tümörler perikarda metastaz yapmaktadır. Klinik olarak perikardit, perikardiyal effüzyon veya perikard tamponatı şeklinde görülürler. Nadiren testis ve renal hücreli kanserler vena kava inferiyor, sağ atriyum ve sağ ventrikül metastazı oluştururken, bronkojenik karsinomlarda nadiren sol atriyuma metastaz yapabilirler.

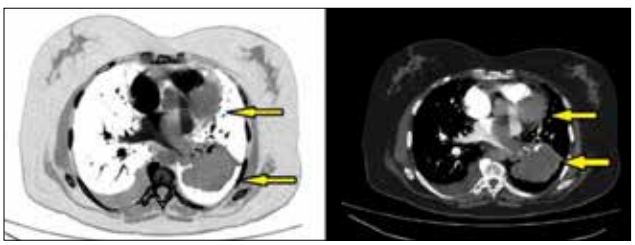
Olgu Sunumu: Altmış bir yaşında bayan hasta, nefes darlığı, halsizlik, çarpıntı ve ortopne şikayeti ile acil servise başvurdu. Başvuru esnasında hastanın elektrokardiyografisi normal sinüs taşikardisi idi. Hastanın özgeçmişinde sadece hipertansiyon vardı. Hastanın fizik muayenesinde S1 sertdi ve mitral dinleme alanında 2-3/6 derece diyastolik üfürüm mevcutdu. Transtorasik ekokardiyografisi yapılan hastanın sol atriyumda 3x5x3 cm mobil ve solid kitle tespit edildi (Şekil 1). Miksoma düşünülen hastanın kalp ve damar cerrahisi konsültasyonu sonucunda tümörün rezeksiyonuna karar verildi. Hastanın takiplerinde hemoptizisi olması üzerine Göğüs Hastalıkları konsültasyonu istendi. Konsültasyon sonucunda, toraks tomografisinde sol akciğerde kitle ve bu kitlenin sol atriyuma metastazı tespit edildi (Şekil 2). Bronkojenik kanser düşünülen hastaya endobronşial biyopsi yapıldı. Patolojide malign mezenkimal tümör olarak sınıflandırıldı. Kalp ve damar cerrahisi ile yapılan rekonsültasyonunda yüksek mortalite nedeniyle operasyondan vazgeçildi. Takiplerinin 10. gününde hasta exitus oldu.

Tartışma: Kalp diğer organlara göre daha az sıklıkla metastaza maruz kalmaktadır. Kalbin metastatik tümörleri genellikle perikarda sınırlıdır. Nadir vakalarda tümör intrakardiyak olarak metastaz yapar. Özellikle melanomlar %50 oranında kalbe metastaz yaparlar. İntrakardiyak metastazlarda en sık sağ atriyum ve sağ ventriküle olur. Bizim olgumuz da intrakardiyak ve sol atriyuma metastaz yapan mezenkimal tümör idi. İlk planda miksoma düşünülmüş ve operasyon planlanmıştı. Ancak hastanın tomografisinde metastazik bir tümör olduğu anlaşıldı.

Sonuç: Özellikle intrakardiyak tümör varlığında metastatik tümörler de akılda tutulmalı ve operasyon öncesinde metastatik tümör olabileceği düşünülerek, tümörün orjini aranamalıdır.



Şekil 1. Sol atriyal tümör ekokardiyografisi.



Şekil 2. Sol atriyal tümör tomografisi.

OPS5-18

Primary mitral valve tumor associated with ST elevation myocardial infarction and cerebral infarction

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Primer cardiac tumors originating from valve and annulus are extremely uncommon. Although myxoma is the most common heart tumor in general, papiller fibroelastoma represents the most common primary tumor of the heart valves. They may cause severe complications including stroke and myocardial infarction. We herein presented a patient diagnosed with ST elevation myocardial infarction and major stroke who refused the surgery for the mitral valve tumor located on anterior mitral leaflet.

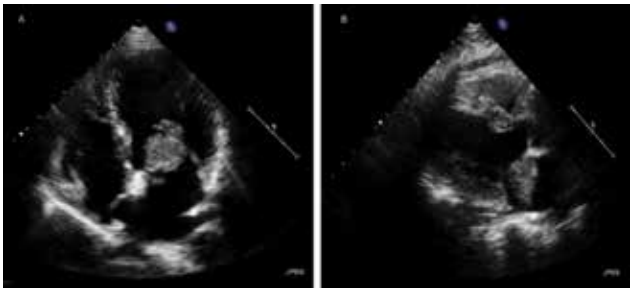

Figure 1. Transthoracic long-axis and four chamber two-dimensional echocardiographic images revealing a pedunculated tumor on the atrial side of the anterior mitral leaflet, prolapsing into the left ventricle during diastole **(A)** and into the left atrium during systole **(B)**.

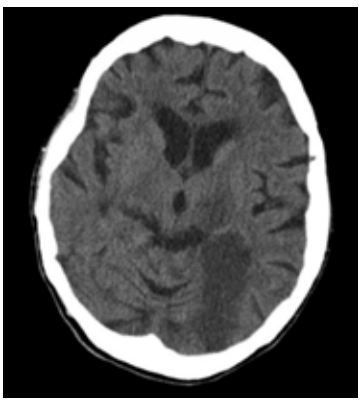

Figure 2. Computed tomography scan of the brain revealed a large left-sided hypo-dense.

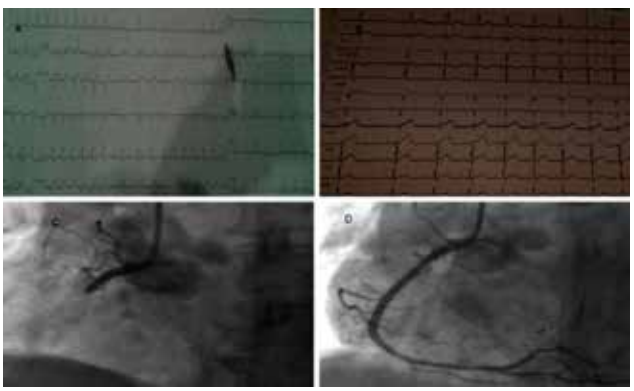

Figure 3. Electrocardiogram **(A)** at the time of admission demonstrates marked ST-segment elevation in leads II, III, and aVF with reciprocal changes in leads I, aVL. After revascularization, electrocardiography shows that ST segment returned to the isoelectric line **(B)**. Coronary angiographic images, the thrombotic occlusion of the mid right coronary artery **(C)** and the right coronary artery restored after primary angioplasty **(D)**.

OPS5-19

Papillary fibroelastoma of the mitral valve: An unusual cause of mitral valve obstruction

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A 50-year-old woman was admitted to our outpatient clinic with the complaint of dyspnea on exertion. Cardiac examination revealed a diastolic murmur best heard at apex, but otherwise unremarkable. Transthoracic echocardiography (TTE) demonstrated a mobile, spherical, hyperechoic, homogeneous mass attached to posterior mitral leaflet causing significant mitral stenosis. 2D and 3D transesophageal echocardiography (TEE) confirmed that the mass located at the posterior mitral leaflet was 1,1×1 cm in size and mitral annular calcification with moderate mitral stenosis (mitral valve area, 1.36 cm^2 and transmitral gradient: maximum 11 mmHg; mean 5 mmHg) (Fig. 1). Ultimately, the patient was referred to cardiac surgery. Surgical excision of the mass was successfully performed. The mass was diagnosed as a papillary fibroelastoma based on the histopathologic examination (Fig. 2). The postoperative course was uneventful. Most common primary cardiac tumors are myxoma and lipoma. Papillary fibroelastomas are relatively rare when compared with others. Although papillary fibroelastomas are usually asymptomatic and detected incidentally on the valves, they can present with life-threatening embolic complications such as myocardial infarction, stroke, pulmonary embolism. Valve obstruction caused by fibroelastomas is an unusual complication. To the best of our knowledge, this is the second case report showing a papillary fibroelastoma on mitral valve causing significant mitral valve obstruction.

Kardiyak görüntüleme / Ekokardiyografi

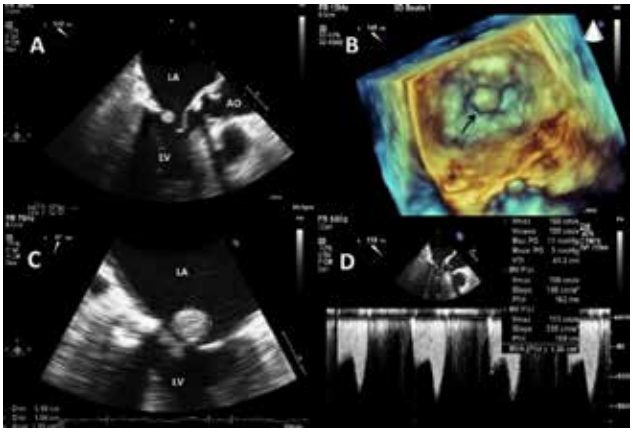

Figure 1. TEE showing a spherical, hyperechoic, homogeneous mass attached to posterior mitral leaflet: 2D echo view **(A)**, 3D echo view (black arrow) **(B)**. TEE demostrating the mass of 1,1x1 cm in size **(C)** causing moderate mitral stenosis with a mean transmitral gradient of 5 mm Hg and mitral valve area of 1,36 cm^2 **(D)**.

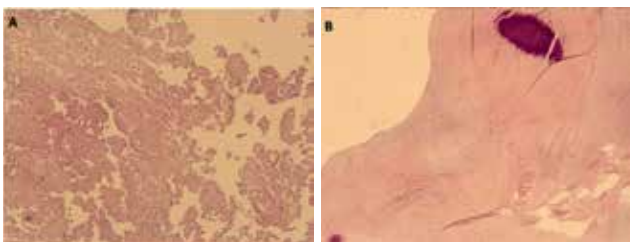

Figure 2. (A, B) Histopathologic examination: The microscopic appearance is characteristic, with branching papillary fronds with avascular collagen and elastic cores.

OPS5-20

Asymtomatic subacute type a aortic dissection and surgical treatment in a patient with previous aortic replacement for ascending aortic aneurysm

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Giant ascending aortic aneurysm is a rare condition which requires emergent surgery due to high risk of mortality. We hereby present an asymptomatic male patient with prosthetic aortic valve and Stanford Type A Aortic Dissection with a huge aneurysm. A 54-year old male patient admitted to our outpatient clinic for routine control. He had a history of aortic valvular replacement and aortic reduction aortoplasty operation due to bicuspid aortic valve and severe aortic regurgitation associated with type A aortic dissection on the 57 mm aneurysm 5 years ago. His medication included beta blocker and warfarin with optimal titration. The vital signs and biochemical parameters were all normal. Apical heart beat deviated to leftward and mechanical valve was auscultated on physical examination. ECG also showed no any abnormal finding. Transthoracic echocardiography (TTE) revealed normal LV systolic functions (LVEF 57%) and mild mitral and tricuspid regurgitation in addition to aortic valvular prosthesis with normal function. Ascending aorta was dilated in all visible segments and larger than 100 mm suspecting aortic dissection (Fig. 1a, b). Computed Tomography (CT) confirmed the prosthetic aortic valve and huge ascending aortic aneurysm with subacute type A dissection associated with aneurysm beginning 3 cm upon the valve. Although the ascending aorta was in 57 mm diameter before the previous operation, the sizes were reached to 55x60 mm in sinus Valsalva level, 74 mm in sinutubular region and total of 110 mm in the dissected ascending tubular region (Fig 1c-e). Descending aorta was in normal sizes (29 mm). Coronary artery anatomy was in normal condition. After the Bentall operation (Fig 1f) for the dissected aortic aneurysm, a complete AV block developed and a permanent pacemaker was implanted. The patient discharged in a good condition following surgery. Dissected aortic aneurysm may reach to very huge sizes without the rupture of the adventitia. Standard Bentall operation may be performed safely; but a control in periodic manner has vital role to survive patient.

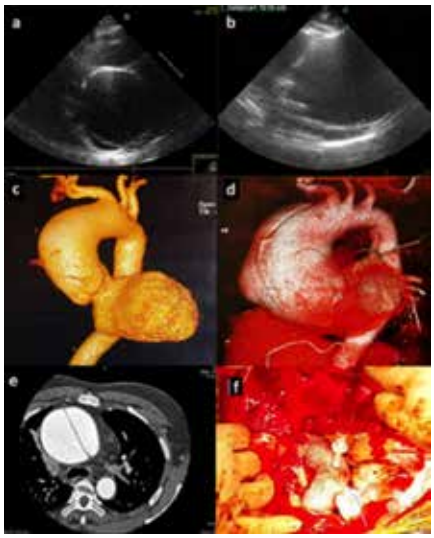

Figure 1.

OPS5-21

Pulmoner venookluziv hastalığı olan olguda başarılı bilateral akciğer nakli öncesi ve sonrası sağ ventrikül fonksiyonlarının ekokardiyografi ile değerlendirilmesi

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Yetmiş sekiz yaşında erkek hasta NYHA class III dispne ve hemoptizi şikayeti ile başvurdu. Özgeçmişinde pulmoner venookluziv hastalığa sekonder pulmoner hipertansiyon tanısı vardı. Bu sebeple hasta akciğer nakil listesine alındı ve takip edildi. Ek kronik hastalığı yoktu. Soygeçmişinde iki kardeşinin tanısını bilmediği akciğer hastalığına sekonder ölüm öyküsü var. Hastanın fizik muayenesinde her iki akciğer bazalinde ralleri ve pretibial ödemi vardı. Dinlemekle triküspit odakta holosistolik üfürüm duyuldu, kalp tepe atım sola kaymış idi. Ekg normal sinüs ritminde idi ve sağ dal bloğu vardı. Akciğer grafisinde her iki akciğer bazalinde plevral efüzyon ve kardiyotorasik indeks kalp lehine artmış izlendi. Ekokardiyografide sol ventrikül sistolik fonksiyonları korunmuş, sağ ventrikül dilate, D-shape septum izlendi (Video 1, 2). Orta derece triküspit yetersizliği ve triküspit kapak yetersizliği jetinden hesaplanan pulmoner arter sistolik basıncı yaklaşık 110 mmHg görüldü. TAPSE 1 cm, sağ ventrikül sistolik motion 10 cm/sn ve FAC %19 olarak değerlendirildi (Şekil 1). Uygun dönorün bulunması üzerine hastaya başarılı bilateral akciğer nakli yapıldı. Nakil sonrası yapılan ekoda sol ventrikül sistolik fonksiyonları normal, sağ kalp boşlukları normal boyutlarda, hafif triküspit yetersizliği, pulmoner arter sistolik basıncı yaklaşık 25 mmHg olarak görüldü (Video 3, 4). Hasta nakil sonrası şikayetlerinin de gerilemesi ile taburcu edildi. Pulmoner venookluziv hastalık venöz trombüslerin damar endotelinde yaptığı yaygın damar endotel hasarı ile oluşmaktadır. Genetik faktörler, otoimmün hastalıklar, toksinler ve bazı ilaçlara maruziyet bu duruma sebep olabilir. Pulmoner venookluziv hastalığın kanıtlanmış akciğer nakli dışında tedavisi bulunmamaktadır.

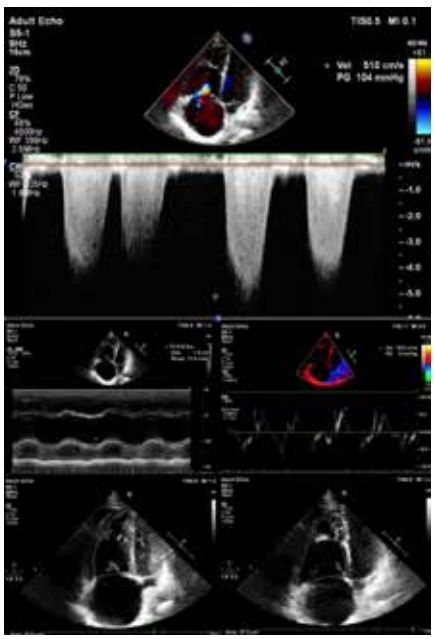

Figure 1.

Kardiyak görüntüleme / Ekokardiyografi

OPS5-22

Successfully thrombolytic treatment of a giant right atrial thrombus

Tolga Onuk, Göksel Çinier, Ahmet Öz, Koray Demir

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45 Years old male patient presented to the hospital with acute dyspnea duration of 3 hours. He complained of swelling and pain on his left leg. His medical history revealed diabetes mellitus and arterial hypertension. His oxygen saturation was 83% in room air, arterial blood pressure was 86/46 mm/Hg, body temperature was 36.7 C. On physical examination, he was dyspneic and tachypneic. Minimal crackles were present at the base of left lung on pulmonary auscultation. 12-lead electrocardiography revealed sinus tachycardia with heart rate of 113 beats per minute (bpm) and prominent R waves on chest leads V1 and V2. Afterwards transthoracic echocardiography (TTE) was performed which demonstrated ejection fraction (EF) 63% (calculated by modified Simpson's method), apparently enlarged right ventricle (RV) (4.8 cm at the base and 3.9 cm at the mid-level on apical four chamber view), moderate tricuspid regurgitation, systolic pulmonary artery pressure (SPAP) of 78 mm/Hg tricuspid annular plane systolic excursion was 0.9 cm and inferior vena cava diameter of 2.5 cm with less than 50% collapse after taking a sniff. During apical four chamber view, a large thrombus (3.4x4.2 cm) was observed which was floating in right atrium and protruding into the right ventricle through tricuspid valve. Subsequently, the patient underwent contrasted computed tomography (CT) of thorax with the initial diagnosis of massive pulmonary embolism. CT demonstrated bilateral multiple thromboemboli in segmentary branches of pulmonary arteries. The patient admitted to the coronary intensive care unit. Recombinant tissue plasminogen activator (t-PA) alteplase (100 mg alteplase in two hours) and subcutaneous enoxaparin were started. 3 hours after the treatment, tachycardia was resolved to 86, arterial blood pressure was 112/75 mm/Hg bpm and oxygen saturation was 97% in room air. Control TTE revealed minimally enlarged RV ventricle (4.4 cm at the base and 3.6 cm at the mid-level on apical four chamber view), mild tricuspid regurgitation and SPAP 25 mm/Hg. Interestingly, large thrombus in the right atrium was dissolved. There were no sign of bleeding event. Doppler imaging revealed deep venous thrombosis in left leg. After 4 days on warfarin treatment, the international normalized ratio (INR) value was effective (between 2-3) and the patient was discharged.

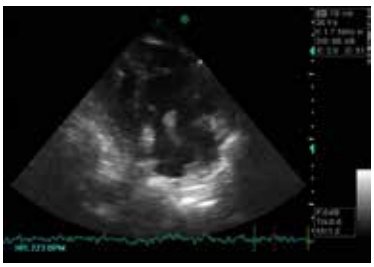

Figure 1. Right atrial thrombus.

OPS5-23

Pericardial effusion secondary to a rare causing:cervical metastatic cancer

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64 years old woman presented with dyspnea. She was diagnosed with cervical carcinoma back in one year and was initially treated with total abdominal hysterectomy and bilateral salpingooopherectomy and was followed without recurrence. There was low QRS voltage in the limb leads, echocardiography revealed normal left ventricle function and large pericardial effusion with the finding of cardiac tamponade and a mass which is 29x33 mm in diameter at right ventricle wall (Figure 1). The mass extending into the right ventricle tract outflow has heterojen appearance with focal hypodense areas (Figure 2). The mass was highly suspicious for a cardiac metastasis. Pericardiocentesis was performed and 500 mL of bloody fluid was drained. A pigtail catheter was then placed for a day. The patient remained stable following the pericardiocentesis. At her follow-up visit, repeat echocardiogram showed minimal pericardial effusion with no evidence of cardiac tamponade. We performed PET-CT to rule out possible relapse of cervical carcinoma and detected multiple metastasis in body including myocardium, pericardium (Figure 3), vagina cuff, rectum wall and sacroiliac joint.The fluid showed no growth of both bacterial and fungal cultures and no acid fast bacilli but showed evidence of malignancy. The pathologic examination showed squamous cell islands in pericardial fluid (Figure 4).

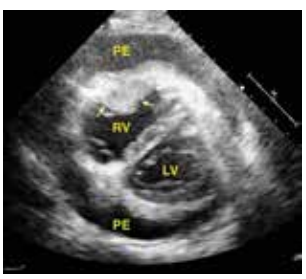

Figure 1. Transthoracic echocardiography shows massive pericardial effusion and the mass (asterisk) in the right ventricle.

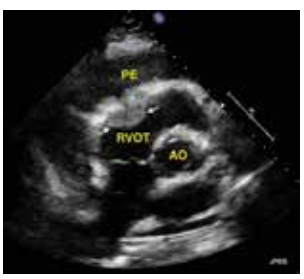

Figure 2. Transthoracic echocardiograhy on short axis aortic valve level shows the mass in the right ventricular outflow and massive pericardial effusion.

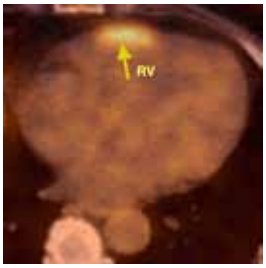

Figure 3. The PET-CT imaging shows the malign mass in the right ventricle-pericardium.

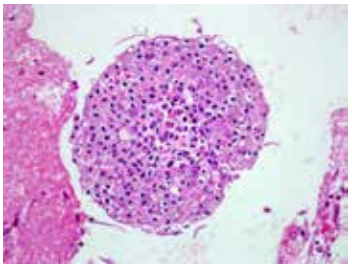

Figure 4. The pathologic examination showed squamous cell islands in pericardial fluid.

OPS5-24

Bileaflet tricuspit valve with severe aort valve stenosis

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The patient was 56 year old man presenting to our outpatient clinic with palpitation, progressive dyspnea. He had no other spesific past history. Sistolic murmur was noted in right sternal border auscultation. ECG was normal sinus rhythm. Chest X-ray was normal. Routine blood work yielded normal results. On transthoracic echocardiography (TTE), mild mitral and tricuspid regurtation mild-modarete aortic regurtation and severe aortic valve stenosis with 88/56 mmHg gradient was revealed. On two-dimensional transesophageal echocardiograhy (2D TEE) was revealed severe aortic valve stenosis with 80/45 mmHg gradient, mild mitral and tricuspid regurtation, mild-modarete aortic valve regurtation as in the TTE (Figure 1). On real time three-dimensional echocardiography (RT3DE) was revealed bileaflet tricuspid valve. Posterior leaflet was rudimenter. (Figure 2). Tricuspid valve (TV) is a complex multicomponent structure. Classic anatomic studies postulated that it consists of three leaflets; anterior,septal and posterior. It is not possible to obtain cross sectional view of the three TV leaflets TV leaflets by the standart TTE views. With RT3DE, atrioventricular valves could be visualized from the ventricular or atrial aspects; detailed TV features and function were described.

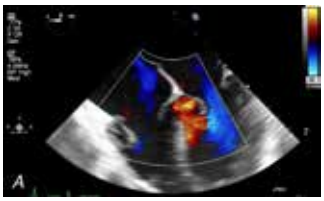

Figure 1.

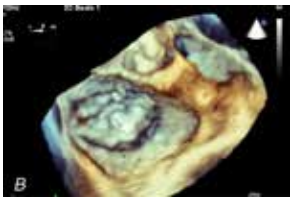

Figure 2.

OPS5-25

Hızlı ve öfkeli

Elif İlkay Yüce[1], Onur Akhan[1], Hamida Mukhtarzade[1], Hakan Gökalp Uzun[1], Ganbar Mammadov[1], Meral Kayıkçıoğlu[1], Emrah Oğuz[2]

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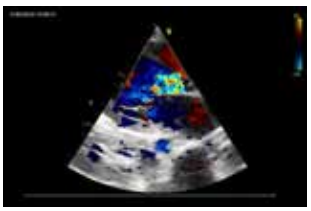
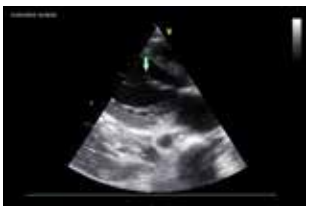
Giriş: İnfektif endokardit kalbin endokardiyal yüzeyinin enfeksiyonu olup ölümcül seyreden bir hastalıktır. S.aureus gelişmiş ülkelerde en sık etkendir. Klinik seyri fülminan olup metastatik infeksiyon sıktır ve hastaların %40'ından fazlası kaybedilir. Bu olgumuzda da stafilokok kaynaklı ve fulminan seyir gösteren enfektif endokarditli bir hastadan bahsedilecektir.

Olgu Sunumu: On dokuz yaşında kronik hastalık öyküsü olmayan hastanın yaklaşık 6 saat süren geçici görme kaybı, baş dönmesi ve eklemlerinde şiddetli ağrı yakınmaları ile başvurması sonrası yapılan kranial MR'ında akut-subakut infarkt lehine bulgular izlenmesi üzerine nöroloji servisine yatırıldı.Ekstremitelerinde veziküler tip döküntüleri ve ateş yakınmaları olması üzerine tarafımıza danışılan hastanın yapılan ekosunda kapak patolojisi ve vejetasyon lehine bulgu saptanmadı. Kan kültürlerinde s.aureus üremesi saptanması ve kardiyak bakısında yeni üfürüm gelişmesi üzerine yapılan kontrol TTE tetkikinde EF'ler normal, MY 3, AY 4, TY 1, SPAP 24 olup aort kapakta 0.8x0.4 cm, mitral kapakta 0.7x0.4 cm boyutlarında vejetasyon lehine görünüm ayrıca mitral kapakta parsiyel korda rüptürüne ait olabilecek görünüm izlenmesi üzerine enfektif endokardit ön tanısı ile kliniğimize transfer edildi. Akciğer ve batın muayenesi olağan olup kardiyak bakısında tüm odaklarda 3/6 sistolik üfürüm saptandı. Yatış ekg'si sinüs taşikardisi uyumlu; kan basıncı,böbrek ve karaciğer fonksiyon testleri olağandı. Nöroloji kliniğinde başlanan gentamisin-daptomisin tedavisine rifampisin eklendi. Hastanın batın usg'sinde kolelithiasis ve abse uyumlu görünüm saptanması üzerine istenen tüm vücut BT'sinde dalakta 6x5 cm boyutlarında abse ve renal septik emboli uyumlu bulgular saptandı. Genel cerrahi ve girişimsel radyolojiye danışıldı. Girişimsel radyoloji yüksek risk nedeniyle girişim planlamadı. İzlemde genel cerrahi tarafından laparoskopik yöntemle abse drenajı yapıldı. Abse kültürlerinde de s.aureus üremesi gözlendi. Solunum sıkıntısı ve satürasyon düşüklüğü nedenli yapılan toraks BT'sinde pnömoni ve pnömotoraks uyumlu bulgular saptanması üzerine göğüs hastalıkları tarafından tedavisi düzenlendi, göğüs cerrahisi tarafından acil cerrahi girişim düşünülmedi. Kontrollerde pnömotoraksta regresyon izlendi. TEE tetkikini tolere edemeyen hastanın kalp damar cerrahisine danışılması sonrası AVR+MVR planlandı. Kontrol kranial BT'de odaklarda progresyon izlenmedi. Enfeksiyon hastalıkları tarafından son düzenlenen tedavisi gentamisin, daptomisin, rifampisin, meropenem, moksifloksasilin şeklindeydi. Kalp damar cerrahisine transfer edilen hasta AVR+MVR operasyonu yapılması sonrası warfarin tedavisi ile taburcu edildi.

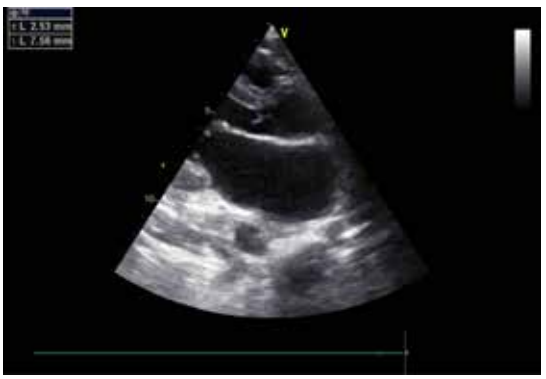
Tartışma: İnfektif endokarditli hastaların tanı ve tedavisi için multidisipliner yaklaşım, her hastaya standard ve ivedi bir tedavi uygulanmasını sağlayabilir. Hastalarda metastatik odakların atlanmaması ve metastatik odak varsa acil cerrahiye verilmesi hayati öneme sahiptir. Olgumuzdaki gibi fulminan seyreden komplike vakalar bu durumun önemini vurgulamaktadır.



Şekil 1. TTE PSUA - Renkli Dopler Görüntüsünde aort yetmezlik jetinin LVOT'u doldurduğu görülmektedir.



Şekil 2. TTE PSUA Görüntüsünde mitral kapakta vejetasyon ve parsiyel korda rüptürüne ait olabilecek görünüm izlenmiştir.



Şekil 3. TTE PSUA kesitinde aort kapakta 0.75x0.25 cm boyutlarında vejetasyon lehine görünüm izlenmektedir.

OPS5-26

Ultra-slow thrombolytic treatment for thombosis of bioprosthetic mitral valve

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[2]*Çorum State Hospital, Çorum*
[3]*Department of Cardiology, Kars State Hospital, Kars*

A 23 year old female was referred with progressive dsypnea and vertigo. Her mitral valve had been replaced bacause of Barlow mitral valve disease by a bioprosthesis in plan a pregnancy. On physical examination blood pressure was 110/70 mmHg and heart rate was 85 bpm. An ECG showed atrial fibrilation. Transthoracic echocardiography revealed a mean diastolic pressure gradient of 10 mmHg across mitral valve. 2D and 3D transesophageal echocardiography revealed multipl motional obstructive thrombus on bioprosthetic mitral valve (Panel A and B, supplementary video 1). Ultra-slow t-PA treatment (1 mg/1 hour) was started for 60 hours. After treatment she showed an improvment of symptoms. Repeat two and three dimensional transesophageal echocardiography showed a mean diastolic pressure gradient of 8 mm Hg, well functioning bioprosthetic valve and remnant 8 mm mobile thrombus (Panel C and D, supplementary video 2). She was discharged on warfarin sodium within INR target of 2 to 2.5.

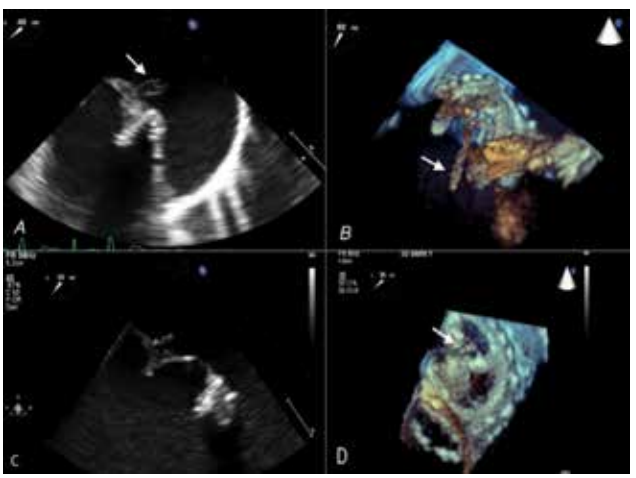

Figure 1.

OPS5-27

A rare cause of cardiac arrest in young adults; subaortic membrane

Mustafa Demir, Cengiz Öztürk, Turgay Çelik, Ali Osman Yıldırım, Sıddık Erdoğan, Murat Ünlü, Zekeriya Arslan, Atila İyisoy

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Obstruction of left ventricular outflow tract (LVOT) accounts 3-10 percent of patients with congenital heart diseases. Discrete subaortic membrane responsible for obstruction of LVOT in 8-10% of these patients. Discrete subaortic membrane reflects a membranous or fibro muscular ring below the aortic valve. It may be isolate or be associated another congenital heart diseases. The etiology of subaortic stenosis (SAS) is not fully understood. It is rarely diagnosed in babyhood and usually manifested in childhood as a LVOT obstruction. We report a rare cause of cardiac arrest diagnosed as subaortic membrane in a young man. A 18 years old male was present with emergency room with cardiac arrest after heavy exercise. The patient is rescued after 30 minutes of cardiopulmonary resuscitation. The patient was unconscious for two days. His family said that he did not have any complaints and comorbid disorders before events. On physical examination, the arterial blood pressure was 100/60 mmHg with a regular pulse, respiratory auscultation findings were unremarkable. However, there was a 3/6 systolic murmur on the right sternal border radiating to the right side of the neck. ECG showed T wave inversion in leads V4-V6 and sinus rhythm (Fig. 1). The bedside transthoracic echocardiography (TTE) showed left ventricular concentric hypertrophy, normal ejection fraction. First of all, we thought that the preliminary diagnosis was hypertrophic cardiomyopathy (HCM) as known common cause of sudden cardiac arrest in young adults. But, we noticed a discrete subaortic membrane causing severe obstruction in the LVOT with detailed TTE examination (Fig. 2). Doppler echocardiography revelaed a maximum 60 mmHg and mean 40 mmHg pressure gradients in the LVOT (Fig. 3). We decided to perform TEE procedure to reveal the exact anatomy before surgery. TEE showed us clearly the discrete subaortic membrane structure and relationship between aortic valve (Fig. 4). The discrete membrane length was measured 6.5 mm and caused mild to moderate aortic regurgitation (Fig 5, 6). Coronary angiogram showed normal coronary artery anatomy. After two days, the patient consciousness slowly opened. 10 days later the patients discharged and referred cardiovascular surgery in order to make subaortic surgery. Patients with SAS can show a range from asymptomatic to varying degrees of symptoms including dyspnea at rest or with exertion, palpitations, chest pain or syncope. The combination of imaging modalities is needed to distinguish subaortic stenosis from HCM with obstruction. It is critical to make the appropriate diagnosis for the treatment options and is important to make screening test for diagnosing SAS as a cause of sudden death in young patients.

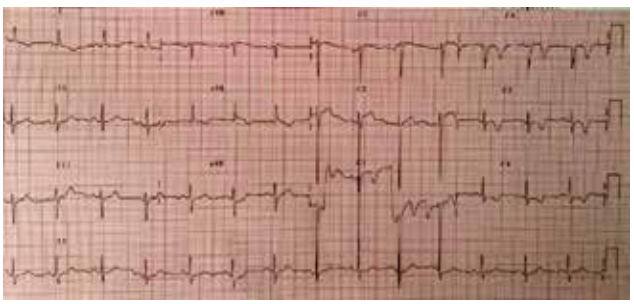

Figure 1.

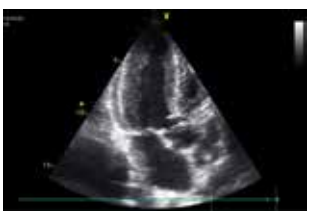

Figure 2.

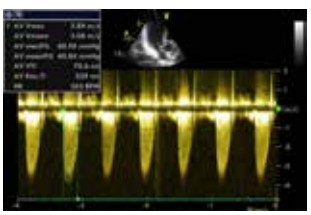

Figure 3.

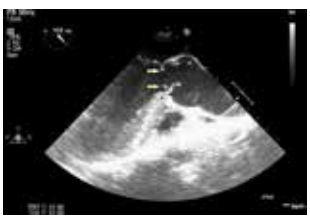

Figure 4.

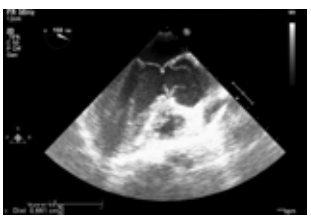

Figure 5.

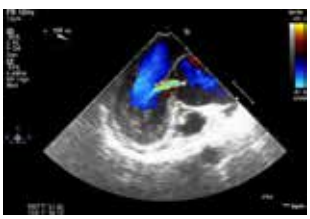

Figure 6.

OPS5-28

An unusual complication of cardiac myxoma

Abdullah Nabi Aslan[1], Zeynep Şeyma Turinay[2], Hakan Süygün[2], Telat Keleş[2], Tahir Durmaz[2], Engin Bozkurt[2]

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[2]*Department of Cardiology, Yıldırım Beyazıt University Faculty of Medicine, Ankara*

A 55-year-old woman presented to our polyclinic complaining of left arm pain of 2 days in duration. Physical examination and electrocardiogram were unremarkable. Transthoracic echocardiography revealed a large, highly mobile mass with irregular contours measuring 29x25 mm in the lower part of the interatrial septum (IAS) (Fig 1a, b, video). Left subclavian artery Doppler ultrasonography confirmed the thromboembolic complication of this lesion. Transesophageal echocardiography showed an irregular-edged mass with 1.52x3.75 mm in diameter attached to IAS without a pedincle (Fig 2, video). Cine cardiac magnetic resonance imaging (MRI) showed a 18x9 mm highly mobile mass with irregular borders at the inferolateral part of the IAS (Fig 3a and 3b). The absolute diagnosis of cardiac myxoma made based on the radiological findings. Then, the patient was referred to the cardiovascular surgery for cyst exicison. Surgical excision of the lesion was performed and the patient was discharged from the hospital with a scheduled follow-up.

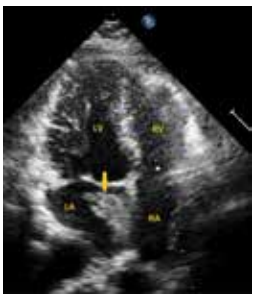

Figure 1. Two-dimensional transthoracic echocardiographic parasternal long axis view of myxoma (arrow).

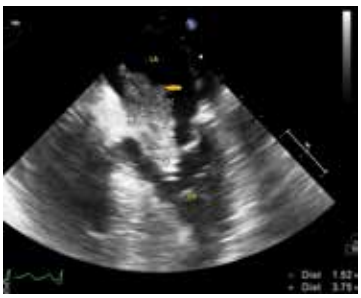

Figure 2. Transesophageal echocardiography image showing myxoma (arrow).

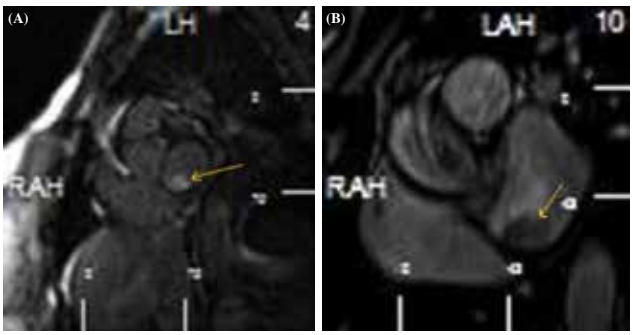

Figure 3. (A) Cardiac magnetic resonance imaging late godalinium enhancement short axis view showing myxoma. **(B)** Cardiac magnetic resonance imaging bright blood image short axis view showing myxoma

OPS5-29

Pericardial effusion mimicking aortic dissection

Emrah Acar, Muhittin Demirel, Mehmet Fatih Yılmaz, Ahmet Guler, Alev Kılıçgedik, Cevat Kırma

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A 70-old female admitted to our emergency department with progressive dispnea and chest pain. In medical history; she had undergone single vessel coronary artery by-pass greft operation one and a half month ago. On the physical examination heart rate was 135 bpm and rythm was regular, blood pressure was 89/61 mmHg, oxygen saturation was 90%-95%. Serial electrocardiographic records showed dynamic changes on lateral derivations. A dissection-like image on ascending aorta was seen on transthorasic echocardiographic parasternal long axis view (see figure, panel A). But because of obesity the patient echogenity was suboptimal, so an emergency transesophageal echocardiography (TEE) was performed for suspected ascending aortic dissection but there was no sign of aortic dissection (see figure, panel B). On TEE short-axis view, fluid accumulation was seen between aortic root and left atrium (see figure, panel C-D). Multislice chest computed tomographic findings were similar to TEE imaging. In addition the collected fluid pinched off the left main coronary artery (see figure, panel E). After surgical drainage of the fluid, symptoms were resolved and dynamic ECG changes on lateral derivations disappeared. Aortic dissection of type A is a rare, but potentially life-threatening disease. The prognosis is determined by an accurate and immediate diagnosis.Aortic dissection can masquerade as another disease and other diseases may look like aortic dissection as like pericardial effusion such as in our case.Pericardial effusion should be kept on mind for the differantial diagnosis of aortic dissection and coronary ischaemia.

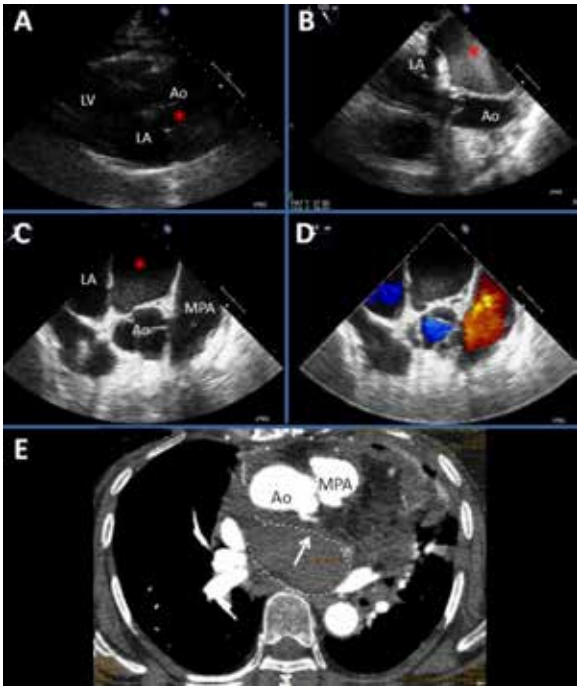

Figure 1. Transthoracic parasternal long axis view **(A)** showing dissection like image (asterisk) on ascending aorta. Aortic dilatation and dissection were not seen on transesofageal modified long axis view **(B)**. On transesophageal short axis **(C)** and coloured **(D)** views, localisated fluid accumulation between aorta, left atrium and main pulmonery artery. Multislice computed tomography image **(E)** demonstrated the collected fluid pinched off the left main coronary artery.

OPS5-30

Sağ atriyuma invazyon gösteren hepatoselüler karsinom

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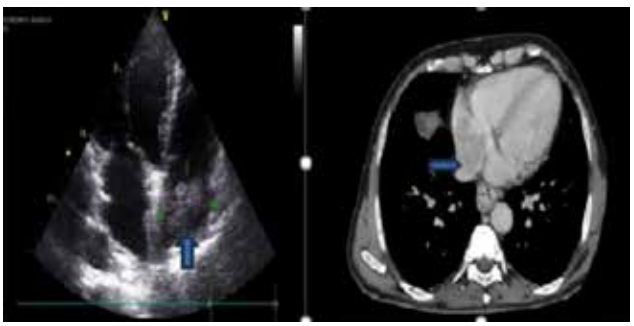
Giriş: Hepatosellüler karsinom genelde kronik karaciğer hastalığı zemininde gelişen, karaciğerin primer malign tümörüdür. Akciğer, beyin, kemik ve surrenal bezlere metastaz sık rastlanan metastaz yeri olmasına rağmen kardiyak metastaz nadir gözlenmektedir. Burada Hepatit B virüsüne (HBV) bağlı Hepatosellüler Karsinom gelişen ve kardiak invazyon gözlenen olgu sunuldu.

Olgu Sunumu: Altı yıldır Hepatit B virüs enfeksiyonu nedeniyle karaciğer sirozu tedavisi gören, bir yıl önce de Hepatoselüler karsinom tanısıyla Transarteryel Kemoembolizasyon işlemi yapılan hasta artan nefes darlığı ve bacaklarda şişlik şikayeti ile acile başvurdu. Hastanın yapılan fizik muayenesinde; bilateral boyun venöz dolgunluğunda artış, hepatomegali, batında asit ve belirgin bilateral pretibial ödem mevcuttu. Hastaya yapılan Transtorasik Ekokardiyografide; ejeksiyon fraksiyonu %55, sol ventrikül çapları normal, sağ atriyumda inferior vena cavaya uzanım göseteren sınırları düzensiz yaklaşık 60x31 mm kitle imajı izlendi. Toraks bilgisayarlı tomografide; sağ atriyumda vena cava inferiorun açılma düzeyinde yaklaşık 53x36 mm sınırları belirsiz tromboze kitle imajı izlendi. Kalp takımı toplantısında sağ atriyumdaki kitle imajı için operasyon kararı verildi. Operasyonda vena cava inferiordan sağ atriyuma uzanan kitle ile hepatik ven lümenini tromboze kitle çıkarıldı. Operasyon başarı ile tamamlandı. Operasyon materyalinin patolojik incelemesi hepatosellüler karsinom ile uyumlu sonuçlandı. Hastanın onkoloji bölümünce takibi devam etmektedir.

Tartışma: Hepatosellüler karsinom karaciğerin en sık primer malign tümörüdür. Bilinen en sık neden Hepatit B virüs enfeksiyonlarıdır. Hepatosellüler karsinom vasküler yapıları invaze etmeye eğilimli olup, portal sistem, inferior vena kava ve atriuma invaze olabilmektedir. Bu olguda nefes darlığının nedeni araştırılırken hepatoselüler karsinomun kalp invazyonuna rastlanıldı. Başarılı cerrahi operasyon ile hasta tedavi edildi. Hepatosellüler karsinomu olan hastalar nefes darlığı ile başvurduğunda kalp ve damar invazyonu olabileceği akılda tutulmalı, transtorasik ekokardiyografi ile değerlendirmelidir.



Şekil 1. Sağ atriyum invazyon görüntüleri. Transtorasik ekokardiyografi ve bilgisayarlı tomografi.

Kardiyak görüntüleme / Ekokardiyografi

OPS5-31

Dirençli Crohn hastasında sitoferez sırasında gelişen sağ atrial trombus

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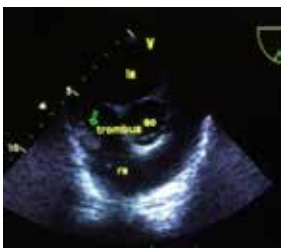
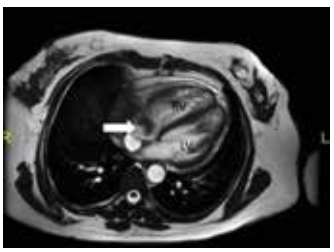
Crohn hastalığı artmış tromboemboli riskiyle birliktelik gösteren sistemik bir hastalıktır. Sistemik vasküler komplikasyonlar görülmesine rağmen kardiyak tutulum oldukça nadirdir. Crohn hastalığında dirençli vakalarda intravenöz kateter eşliğinde adzorbtif sitoferez tedavisi uygulanmaktadır. Bu vakada, dirençli Crohn tanısı olan genç kadın hastada kateter ilişkili sağ atrial trombus örneği sunacağız. Kırk beş yaşında, 2 yıldır tedaviye dirençli Crohn hastalığı ile takipli kadın hastada son 1 aydır sağ juguler kateter ile haftada iki kez adsorbtif sitoferez hikayesi mevcuttu. Sitoferez seansı esnasında genel durum bozukluğu, halsizlik ve nefes darlığı gelişmesi üzerine hastaya transtorasik ekokardiyografi yapıldı. Sağ atrium içinde 2.4x1.9 cm çaplarında yumuşak eko dansiteli, öncelikle trombus olduğu düşünülen ekojenik yapı izlenmesi üzerine hastaya transözefageal ekokardiyografi yapıldı. Sonucunda sağ atrium içerisinde atrial septum karşı duvarında 1,2 cm kalınlığında, yaklaşık 3 cm uzunluğunda, duvara yapışık ve inferior vena kavaya doğru uzanan, triküspit kapakla ilişkisiz, öncelikle trombus ile uyumlu olan yumuşak ekojeniteli görünüm izlendi (Şekil 1). Hastaya kitle ekartasyonu açısından yapılan kardiyak MRG'da sağ atriumda, inferior duvardan lümene uzanan yüzeyi düzensiz trombus ile uyumlu olabilecek yumuşak doku saptandı (Şekil 2). Ön planda kateter ilişkili trombus düşünülerek hastanın kateteri çekildi. Hastaya varfarin tedavisi başlandı. Takibinde sağ atriumdaki trombusa bağlı muhtemel pulmoner tromboemboli (PTE) açısından BT anjiografi çekildi ve ana dallarda PTE dışlandı. INR efektif düzeyde izlenen hastanın takiplerinde sağ atrial trombusta küçülme izlenmiş olup, hasta kardiyak açıdan asemptomatik olarak izlemine devam etmektedir. Behçet hastalığında ve SLE'da olduğu gibi Crohn hastalığı da arterial ve venöz tromboemboliye yatkınlık yaratmaktadır Sağ atriyal trombus uzamış santral venöz katater ve transvenöz pacing elektrotları ile sıklıkla görülmektedir. Literatürde inflamatuar barsak hastalığında tromboembolik komplikasyonlar %1.3 oranında saptanmış ve yüksek mortalite ile ilişkili bulunmuştur. Bu nedenle crohn hastalarında sitoferez veya başka nedenlerle yapılan kateterizasyonlarda son derece dikkatli olunmalı, venöz yatak travmatize edilmemeli, mümkün olan en kısa sürede katater çekilmedir. Bu tarz girişimli hastalarda, yeni gelişimli dispne öncelikle pulmoner emboli açısından ileri tetkik edilmeli ve kardiyak trombus açısından ekokardiyografi görülmelidir. Sunduğumuz vaka, Crohn hastalığı seyrinde dispne ile prezente olan ve kateterizasyona bağlı olduğu düşünülen bir intrakardiyak trombus vakasıdır. Görüntüleme bulguları ile de desteklenen ilginç ve öğretici bir vaka olduğunu düşünmekteyiz.



Şekil 1. Transözefageal ekokardiyografi ile saptanan sağ atrial trombus görüntüsü.



Şekil 2. Kardiyak MR görüntülemede sağ atrial trombus görüntüsü (okla gösterilmiştir).

OPS5-32

An interesting restrictive cardiomyopathy case which was previously misdiagnosed as atrial septal defect

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A 29 years old female was admitted to one private hospital inpatient clinic with complaints of dyspnea, fatigue, fever and persistent productive cough, in 16th week of pregnancy. The patient was put on antibiotherapy with clinical signs of crackles in middle zone of right lung. The patient was consultated with cardiology department whose CRP and sedimentation markers continued to rise although medical therapy. The echocardiography showed normal left ventricle systolic function, biatrial enlargement, pulmonary hypertension (PASP 57 mmHg), left and right ventricle size in normal range with suspicion of atrial septal defect (ASD) and pulmonary venous return anomaly. Afterwards, the patient was referred to our university hospital as a high risk pregnant due to severe pulmonary hypertension. Echocardiography revealed similar signs to echocardiography performed before, except suspicion of drop out from left atrium to right atrium. Besides, abdomen ultrasonography revealed dilation of intrahepatic veins may be consistent with cardiac failure. Monthly echocardiography was recommended for the patient whom transesophageal echocardiography (TEE) could not be made regarding the risk of pregnancy. In this period an another echocardiography was reported with suspicion of ASD and aortopulmonary window in a private hospital. The patient was admitted our cardiology inpatient clinic with worsening functional capacity (FC III NYHA) and cough especially in nights. On examination, blood pressure was 110/70 mmHg, pulse rate was 110 bpm and also crackles on the mid and basal right lung, hepatojugular reflux, hepatomegaly and S4 was detected. ECG revelaed sinus tachycardia with premature atrial complexes and Pro BNP was 2046 mg/dL. The new echocardiography signs which includes restrictive pattern of mitral flow velocities was founded consistent with restrictive cardiomyopathy. Even in childhood, functional capacity was not capable of I NYHA was learnt from further anamnesis. Furosemid and enoxaparine was started to given for heart failure and spontaneous echo contrast in the left chambers, respectively with proper antibiotherapy. After this treatment the patient recovered from symptoms such as dyspnea and cough and lung auscultation signs turned to normal. Afterwards, the patient was transferred to obstetrics department and gave a healthy birth by caesarean section with no complication. After delivery, in a stable clinical mood, hemodynamic measurements and TEE was performed revealing post capillary pulmonary hypertension and excluding ASD. These tests confirmed the diagnosis. Treatment was completed at cardiology clinic and the patient was discharged. Biatrial enlargement without any enlargement of ventricles was the keypoint for true

diagnosis for us. Given the benefit of this case is to present that echocardiography express more clinical value when considered with anamnesis, physical examination and may be able to prevent misdiagnosis in this way.

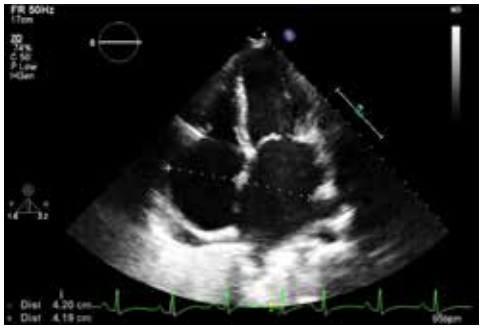


Figure 1. Echocardiography 4 chamber view.

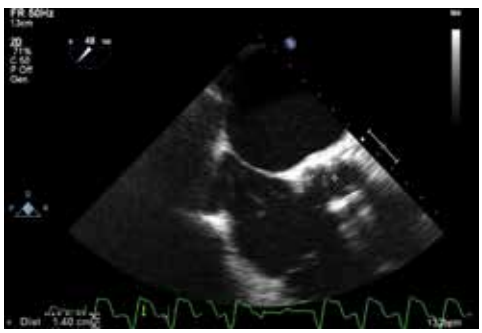
OPS5-33

Kronik atrial fibrilasyonlu bir hastada perkütan biyoprotez aort kapak replasmanı (TAVI) sonrası geç dönemde kapak trombüs olgusu

Ömer Şatıroğlu, Oğuzhan Bodur, Hüseyin Durak, Ramazan Gengörü, Hkan Duman, Zakir Karadağ, Murtaza Emre Durakoğlugil, Turan Erdoğan

Recep Tayyip Erdoğan Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Rize

Seksen altı yaşında erkek hasta, eforla nefes darlığı yakınması ile kliniğimize başvurdu. Özgeçmişinde, 2 yıl önce dış merkezde, ileri aort darlığı sebebiyle perkütan yolla, Edward saphien xt, biyoprotez aort kapak implantasyonu (TAVI) öyküsü mevcuttu. Ayrıca kronik atrial fibrilasyon (af), son dönem kronik böbrek yetersizliği (diyaliz tedavisi almakta), kronik obstruktif akciğer hastalığı, konjestif kalp yetersizliği (hafif sol ventrikül sistolik disfonksiyonu), gibi kronik hastalıkları vardı. Hasta sürekli oral antikuagulan tedavisi (varfarin) almaktayken, idrar yolunda durduralamayan kanama, varfarin overdozu ve aşırı Uluslararası Düzeltme Oranı (INR), yüksekliği sebebiyle, varfarin tedavisi üroloji hekimi tarafından kesilmişti. Hastada 10 gündür varfarin tedavisi kullanmama hikayesi mevcuttu. Hastanın fizik muayenesinde, kalp oskultasyonunda, aort odağında 3/6 sistolik üfürüm mevcuttu. Ekokardiyografik incelemede, ejeksiyon fraksiyonu %45, biyoprotez aort kapakda (Edward saphien xt, TAVI), gradient artışı ve şüpheli thrombus imajı görüldü. Tanıyı kesinleştirmek için transözefagiyal ekokardiyografi yapıldı, TAVI aort kapak düzeyinde, kapak hareketlerini kısıtlayan thrombus imajı ve sol atriyum, apendikste ise yoğun spontan eko kontras imajı izlendi (Şekil 1), (Video 1, 2, 3). Hastaya ilk 3 gün parenteral antikuagulan heparin ve varfarin tedavisi verildi. Yakın, INR, aktif parsiyel tromboplastin zamanı (aptt) takibine alındı. Sonrasında varfarin tedavisi ve kanama yönünden yakından takibe alındı. Klinik durumu stabil seyreden ve biyoprotez aort kapakta trombüsü kaybolan hasta, oral antikuagulan (varfarin) tedavisi altında, yakın INR takibi ile taburcu edildi.



Şekil 1.

OPS5-34

Büyük çapa sahip sağ koroner arterdeki darlığın brakiyal yoldan renal stent implantasyonu ile tedavi edilmesi

Ercan Karabey, Osman Beton

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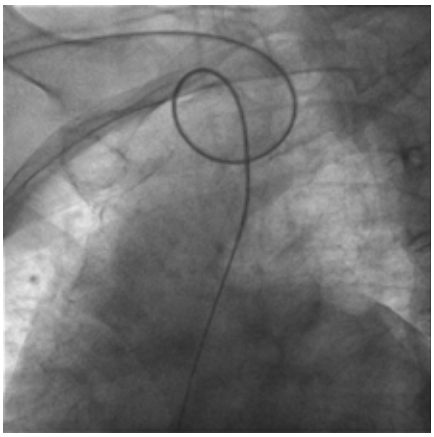
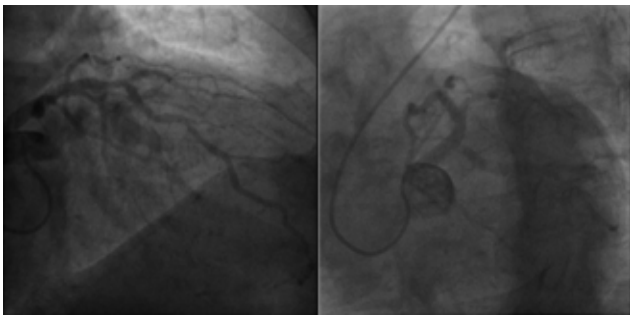
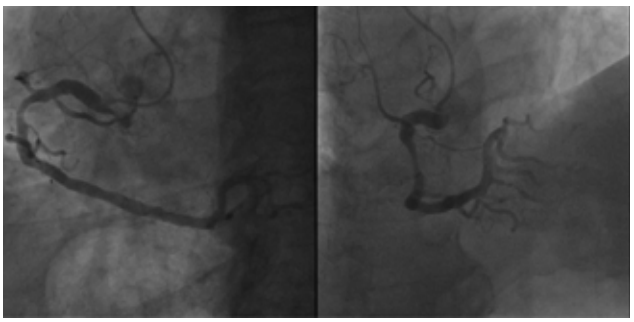
Elli dokuz yaşında erkek hasta, polikliniğe 1 aydır olan eforla gelen göğüs ağrısıyla başvurdu. Tipik angina pectoris tarifleyen hastanın ağrısı 10 dakika kadar sürüyor ve istirahatle geçiyormuş. Angina sol kola, çeneye, mide bölgesine yayılıyor ve beraberinde bulantı oluyormuş. Mesleği kasap olan ve ağır işler yapan hastanın yaşam ve iş kalitesi angina pektoris ile iyice düşmüş. 2011 yılında unstabile anjiina pectoris tanısı ile hastaya koroner anjiyografi (KAG) yapılmış, LAD ortada ardışık %30 darlıklar, CX ortada total tıkalı ve RCA proksimalde %40 darlık saptanmış. CX'deki %100 darlığa PTCA ve 3.5x18 mm stent uygulanmış. Özgeçmişinde 2 yıldır olan diabettes mellitus tip 2, 10 yıldır olan hipertansiyon, 8 yıldır olan hiperlipidemi ve 38 yıldır olan günde bir paket sigara içiciliği mevcut olan hastanın fizik muayenesinde, boy 198 cm ve ağırlık 140 kg (VKİ: 35.7 kg/m^2), kan basıncı 160/90 mmHg, kardiyovasküler sistemde S1, S2 ritmik, S4 (+), mitral odakta 1/6 sistolik üfürüm saptandı. Diğer sistem muayeneleri doğal bulundu. Laboratuvar testlerinde HbA1c % 7.6, CRP 10.0 mg/dl, LDL 170mg/dl, total kolestrol 280 mg/dl, HDL 35 mg/dl saptandı, diğer rutin testler normaldi. Ekokardiyografide sol ventrikül hipertrofik (IVS: 1.5 cm, Aduvd: 1.5 cm) ve EF %50 saptandı. Efor testinin pozitif saptanması üzerine hastaya KAG planlandı. KAG işlemi, önce sağ ve sol femoral arterden girişim yapılarak standart 100 cm ve 110 cm diagnostik kateter kullanılarak yapılmaya çalışıldı, fakat hastanın uzun boylu olması ve tortüyoz damar yapısı nedeniyle kateterler koroner sisteme oturmadı. Sağ brakiyalden girişim yapıldı, fakat subklavian tortüyozite nedeniyle, yine koronerler görüntülenemedi (Şekil 1). Son olarak, sol brakiyal arter yolundan sol koroner sisteme AL2 kateter ile ulaşıldı ve sol sistem görüntülendi. LAD ortada ardışık %30 darlıklar ve CX ortadaki stent açık saptandı (Şekil 2). Sağ koroner sistem, çıkış anomalisi nedeniyle AL2 kateter ile görüntülenebildi ve proksimalde %80 darlık olduğu saptandı (Şekil 3). Fakat RCA'nın referans çapının 6.0 mm olması (elde uygun stent olmadığından) ve işlemin uzun sürmesi nedeniyle ayrı seanta RCA'ya PTCA yapılmasına karar verildi. Ayrı seansta, RCA'daki darlığa, sol brakiyal arter yolu kullanılarak 7F AL2 guiding kateter ile derin oturum sağlandı. Şaftı kalın ve flexibilitesi çok düşük olan 6x18 mm boyutlarında renal Herculing stent (CoCr) direkt implante edildi ve tam açıklık sağlandı (Şekil 4). Takiplerinde hasta asemptomatik seyretti.



Şekil 1.



Şekil 2.



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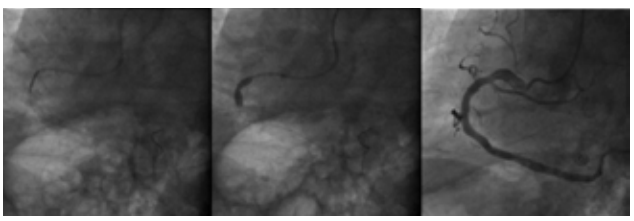

Şekil 4.

OPS5-35

Incidental echocardiographic finding in patients with worsening chronic obstructive pulmonary disease: A giant myxoma

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Primary tumors of the heart are extremely rare and usually benign, and have an autopsy frequency of 0.001–0.28%. Myxoma is the most common type of these tumors, comprising 75–80% of all primary tumors of the heart. Although they can originate from any chamber of the heart, they are more frequently located in the left atrium. A 73-year-old man was followed up for seven years with diagnosis of chronic obstructive pulmonary disease by general practitioner. He had worsening in symptoms of COPD for last six months then refferd to department of pulmonology at our hospital. On high resolution computed tomography (HRCT) examination, heart chambers enlargements were reported (any intracardiac mass was reported in HRCT examination) so patients consulted the cardiology department to exclude heart failure. Pateint had normal ejection fraction (according to modified Simpson method and Teicholz method) but transthoracic echocardiography displayed a large mobile mass (50x40 mm) attached to the interatrial septum with a pediculum in left atrium. The mass was protruding into the left ventricle in systole. The echocardiographic appearance of the mass resembled a myxoma and the patient was referred for open-heart surgery. Although myxomas are histologically benign but they can result in fatal complications due to embolization and intracardiac obstruction. Shortness of breath is a common cardiac symptom. The recommended treatment of myxoma is surgical resection to prevent complications.

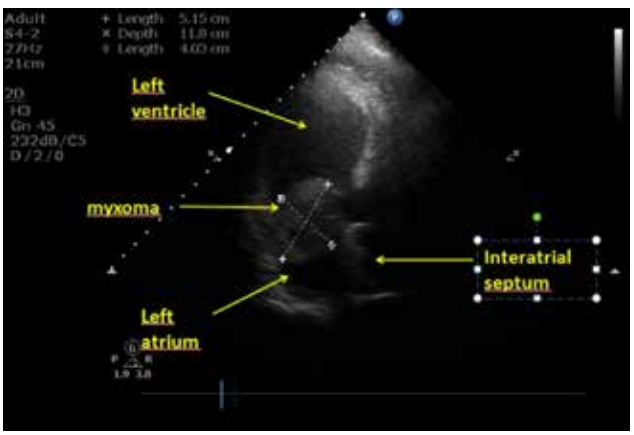

Figure 1. A giant myxoma in left atrium.

OPS5-36

A handicap in emergency departments: Acute myocardial infarction masquerading acute aortic dissection

Muhammed Keskin[1], Mert İlker Hayıroğlu[1], Tolga Onuk[1], Ümran Keskin[2], Mehmet Bozbay[1], Şahin Avşar[1], Ahmet Öz[1], Mehmet Eren[1]

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[2]*Department of Internal Medicine, S.B. Ümraniye Training and Research Hospital, İstanbul*

Acute myocardial infarction (AMI) related to acute aortic dissection (AD) is a rare complication. A 71-year-old man patient with no prior history of coronary artery disease was admitted with acute onset chest pain. The character of the pain was a retro-sternal squeezing sensation, radiating to jaw and left shoulder. The patient had a history of hypertension and type II diabetes mellitus. Physical examination revealed normal vital findings with bilateral blood pressure of 142/91 and 138/86 mmHg. Electrocardiogram showed ST-segment elevation in leads D2,3-aVF and resiprocal changes in lead D1 and aVL. The patient was diagnosed with AMI and allowed to catheterization laboratory subsequent to administration of aspirin 300 mg and ticagrelor 180 mg. During the transport, cardiac arrest with asystole was occurred. Coronary angiography (CAG) was performed while performing chest compression. CAG revealed patent coronary arteries without a total occlusion. Aortography via a pigtail catheter was performed to exclude any acute aortic pathology. Aortogram revealed an AD flap that started from sinotubular junction and progressed through to the arcus aorta. A fast transthoracic echocardiogram (TTE) was also performed under chest compression and showed a dilated aortic root (4.3 cm) with a dissection flap in the ascending aorta. No pericardial effusion was obtained. Despite the fact that heart surgeon team immediately consulted, the patient could not be resuscitated and cardiac resuscitation was terminated 90 minutes after asystole. The patient did not ever develop a spontaneous rhythm during the resuscitation. This case emphasises the importance of fast TTE in AMI to exclude the acute AD. The initial antiplatelet aggregation and anticoagulation may increase the risk of postoperative bleeding in these patients. Although a fast TTE may exclude the acute AD, it may increase door-to-balloon time that is vital in AMI.

Kardiyak görüntüleme / Ekokardiyografi

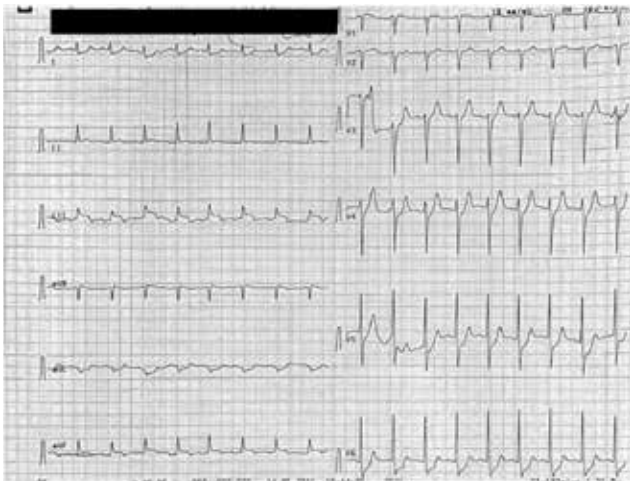

Figure 1. Electrocardiogram shows ST-segment elevation in leads D2,3 and AVF, and ST-segment depression in lead D1 and aVL.

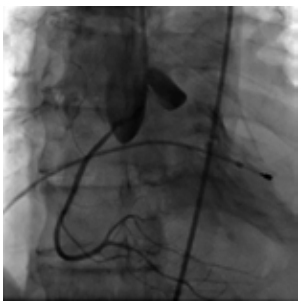

Figure 2. Coronary angiogram shows patent right coronary artery.

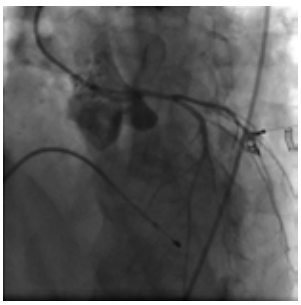

Figure 3. Coronary angiogram shows left anterior descending and left circumflex arteries.

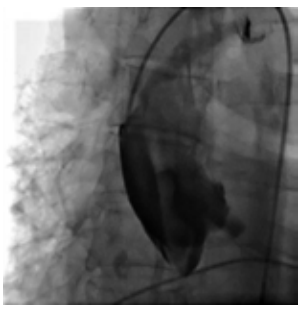

Figure 4. Aortogram shows dissection flap starting from sinotubular junction and progressed through arcus aorta.

OPS5-37

Anomalous origin of right coronary artery from left coronary sinus and interarterial course

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Anomalous origin of the right or left coronary artery from the sinus of Valsalva is a rare congenital abnormality, and most of patients are asymptomatic. However, if it is associated with an interarterial course, these patients may have symptoms and/or evidence of myocardial ischemia. 47-year-old male patient was admitted to our outpatient clinic with the complaints of chest discomfort. On physical examination, blood pressure was 120/70 mmHg, heart rate was 86/min. Cardiac auscultation revealed normal heart sounds, and no heart murmurs. Transthoracic echocardiography showed no wall motion abnormalities and morphological changes. Exercise electrocardiography was positive. Coronary Computed Tomography Angiography revealed anomalous origin of the right coronary artery from the left Valsalva sinus, with interarterial course between aorta and pulmonary trunk, without apparent narrowing of its proximal coronary ostium, slit-like coronary ostium and non-critical lesions (Figure 1). Cardiac catheterization demonstrated the same findings (Figure 2, Video). ECG gated Tc-99 m MIBI stress myocardial scintigraphy did not show ischemia. We decided to follow up him with conservative approach because of no evidence of ischemia and reluctance of the patient. Surgery should be applied in order to prevent sudden death with known symptoms of myocardial ischemia. Conservative approach can be used in the absence of symptoms or myocardial ischemia. Multiple surgical techniques have been utilized, including coronary bypass graft placement and reimplantation of the anomalous coronary to the appropriate sinus. We presented a case of anomalous origin of a coronary artery from the opposite sinus with an interarterial course without evidence of ischemia.

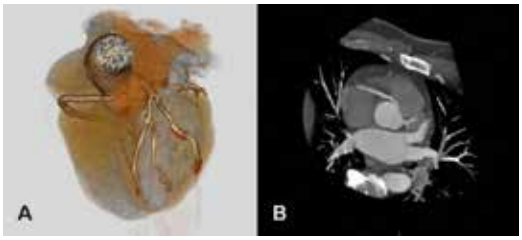

Figure 1. Anomalous origin of the right coronary artery from the left Valsalva sinus **(A)**, with interarterial course between aorta and pulmonary trunk **(B)**, without apparent narrowing of its proximal coronary ostium, slit-like coronary ostium.

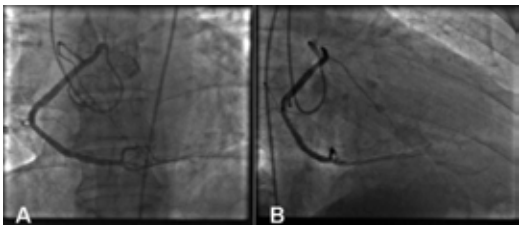

Figure 2. (A, B) Anomalous origin of the right coronary artery from the left valsalva sinus.

OPS5-38

Masif perikardiyal efüzyonlu hastada unutulmaması gereken ön tanı: Tüberküloz perikardit

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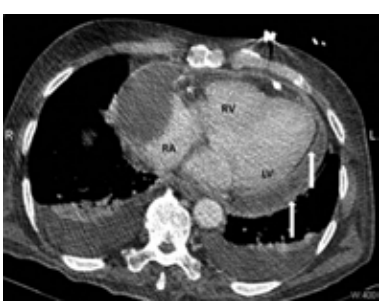
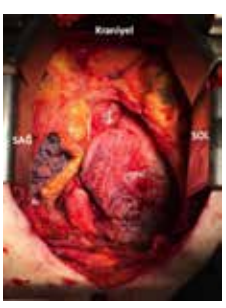
Tüberküloz (tbc), Afrika ve Asya kıtasında halen masif effüzyon ve konstriktif perikarditin en sık sebebi iken, gelişmiş ülkelerde oldukça arka sıralarda yer almaktadır. Perikardiyal tbc, ektrapulmoner tbc'nin morbidite ve mortalitesi yüksek ciddi bir komplikasyonudur. Kesin tanısı klinik şüphe üzerine ileri araştırma ile konulan tbc perikarditinde direkt tanı perikard sıvısı veya perikard biyopsisi ile olmaktadır. Perikardiyosenteze rağmen hızlı tamponad kliniği geliştiren ve sıvı incelemesinde basil saptanmamasına rağmen perikardektomi materyali incelemesi ile tbc perikarditi tanısı alan ileri yaş bir hastayı sunduk. Yetmiş yedi yaşında erkek hasta, son 2 aydır iştahsızlık, yorgunluk ve son 1 haftadır gelişen göğüs ağrısı ve nefes darlığı ile kliniğimize başvurdu. Laboratuar incelemesinde sedimentasyon: 85 mm/s, CRP: 158 mg/L olup diğer kan sayımı ve biyokimyasal değerleri normaldi. Çekilen ekokardiyografisinde kalp etrafında en geniş yerinde 2 cm ölçülen masif efüzyon, sağ atriumda kollaps ve solunumla mitral E dalgasında %25 azalma izlendi, vena kava inferior kollabe olmamaktaydı. Tamponad kliniğinde olan hastaya perikardiyosentez uygulanarak perikardiyal dren yerleştirildi. Perikardiyal mayi eksüda vasfında olup, sitolojisi belirgin lenfositoz göstermekteydi. Gönderilen tetkik sonuçlarında üreme olmayan ve basil saptanmayan hastanın sıvı ADA düzeyi 65 (N:<30 IU/L) geldi. İzlemde dreninden gelen olmamasına rağmen perikardiyal sıvı artışı devam eden hastanın ileri tetkik için yapılan toraks BT'sinde belirgin perikardiyal kalınlaşma ve özellikle sağ atriuma bası yapan sıvı koleksiyonu izlendi (Şekil 1). Tbc şüphesi olan hastaya perikard biyopsisi ile tanısını kesinleştirmek ve effüzyonun tekrar boşaltılması için kalp damar cerrahisi tarafından müdahale edildi, ancak yetersiz drenaj ve işlem sırasında gelişen tamponad kliniği nedeniyle hastaya torakotomi yapılarak perikardiyektomi işlemi yapıldı, hastada viseral perikardın kalın ve fibrinöz yapıda olduğu izlendi (Şekil 2). Perikard patolojisinde granulamatoz yapıda perikard ve asit fast basil (+) gelen hastada 4'lü anti-tbc tedavisi başlandı. İzlemde klinik durumu ilk haftadan sonra düzelmeye başlayan ve CRP yanıtı olan hastanın medikal tedavisi düzenlenerek ayaktan takip olmak üzere taburculuğu yapıldı. Tüberküloz perikarditi ülkemizde son yıllarda azalmakla beraber özellikle yaşlı ve immün sistemi deprese olan hastalarda araştırılması gereken bir klinik durumdur. Hastamızda ilk perikard sıvısından PCR çalışılması veya perikard biyopsisi alınması durumunda daha erken tanı koyma ve tedavi şansımız olacağını düşünmekteyiz. Perikardiyal sıvıda görülen lenfositoz ve ADA yüksekliği indirekt olarak Tbc'den şüphelendirmeli, direkt metodlarla da tanı desteklenmelidir.Tekrarlayan, hızlı gelişen efüzyonlarda veya şüpheli fibrinöz effüzyon görünümlerinde perikard biyopsisi veya sıvıdan gönderilecek PCR direkt tanıda yeterli olacaktır. Günümüzde antitbc tedavisi ile mortalite %80'lerden %25'lere inmiştir.



Şekil 1. Toraks BT'de kalınlaşmış ve kontrastlanma gösteren perikard yaprakları (oklar) ve özellikle sağ atriuma bası yapan perikardiyal efüzyon görülmektedir.



Şekil 2. Perikardiyektomi sonrası viseral perikardın kalın ve fibrinöz yapısı.

Kardiyak görüntüleme / Ekokardiyografi

OPS5-39

Kronik atrial fibrilasyonu olan bir hastada dev biatrial trombüs

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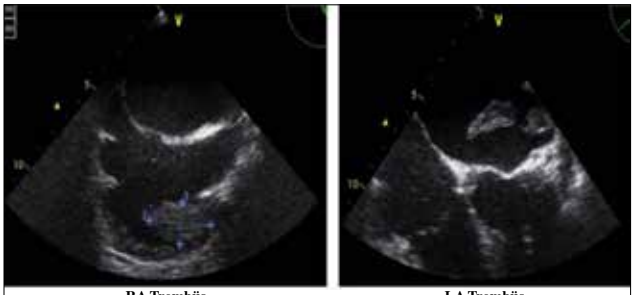
Giriş: Sol atriyal apendiksteki trombüslerin zemininde sıklıkla tedavi almayan ciddi mitral darlığı ve atriyal fibrilasyon vardır. Bunun nedeni bu patolojilere bağlı olarak sol atriyal apendiksin kasılma fonksiyonunun bozulması ve staza bağlı olarak kan akım hızının azalmasıyla gelişen spontan eko kontrasttır.Sağ atriyal trombüs, sol atriyal trombüse göre çok daha ender olarak görülmektedir. Ender görülmesine karşın, trombüs sağ atriyum duvarından bağımsız ve serbest durumda ise masif pulmoner emboli ya da paradoksal emboli gibi ölümcül komplikasyonlara yol açabilir.Her iki atriumda trombüs izlenmesi ise oldukça nadir bir durumdur.Serebrovasküler olay sonrası kliniğimize danışılan ve biatrail dev trombüs saptanan hastayı vaka sunumu olarak hazırladık.

Olgu Sunumu: Yetmiş yaşında uzun süredir atrial fibrilasyonu olan hasta iskemik kaynaklı serebrovasküler olay nedeniyle nöroloji kliniğine yatırıldı.Hastanın anamnezinde uzun süredir kullandığı warfarin tedavisini kendi isteği ile 2 ay önce bıraktığı öğrenildi.Yapılan transtorasik ekokardiyografide görüntü kalitesi çok kötü olan hastanın sol atriumda kitle imajı izlendi ve etyolojinin aydınlatılması amaçlı hastaya transösefagial ekokardiyografi (TEE) yapılmasına karar verildi. TEE de hastanın sol atrial appendiksini dolduran ve atrium içerisinde serbest hareket eden kitle ve sağ atriumda da büyük bir kitle izlendi (Şekil 1, 2, Video). Hastaya cerrahi tedavi ile kitlelerin eksizyonu yapıldı ve sonuç trombüs olarak yorumlandı.

Tartışma: Atrial kitleleri göstermede transözefajiyal ekokardiyografi transtorasik ekokardiyografiden çok daha etkindir.Bizim vakamız oldukça obez ve görüntü kalitesi kötü olan, bu nedenle sol atriumda şüpheli kitle imajı olarak rapor edilen bir hastaydı.Bununla beraber sağ atrium içerisinde herhangibir kitle izlenmemişti.Bu nedenle TEE yapıldı ve hastada her iki atriumda kitle izlendi. Atrial trombüsler hastanın ve trombüsün durumuna göre medikal ve cerrahi olarak tedavi edilebilir. Hastamız her iki atriumda büyük trombüslerin varlığı, sol atrium içerisindeki trombüsün hareketli yapısı ve yakın zamanda iskemik bir serebrovasküler olay geçirmesi nedeniyle cerrahi tedaviye yönlendirildi.Başarılı cerrahi girişim sonrası eksize edilen materyal trombüs olarak yorumlandı ve hasta şifa ile taburcu edildi.



Şekil 1.

OPS5-40

Three dimensional echocardiogrpahic view of a giant free-floating thrombus in right atrium

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Intracardiac space occupying lesions are always a source of excitement and a sort of alarm when seen during an echocardiographic examination. Most important questions to be answered about the mass at the report are probably the size, shape, structure, haemodynamic importance of the mass, besides its place and attachment site. It might not be possible to understand its turning movement around its own axis just by two dimensional echocardiography and therefore three dimensional echocardiography gives more valuable about intracardiac masses.In this paper we reported a case with giant free-floating trombus in right right atrium detected by using three dimensional transesophageal echocardiography. 72 year old women admitted to our emergeny department with a complaint of presyncope and nausea beginning 6 hour before. She had also dyspnea for about 15 days. In her history she had hypertension and hyperlipidemia. In hospital admission blood pressure was 90/60mmHg and pulse rate was rhythmic and 100 beats per minute. In her chest examination, respiratory rate was 18 per minute and pulmonary sounds were normal. Grade 1-2/6 systolic ejection murmur was heard at cardiac examination. Electrocardiogram revealed sinus rhythm with sinus tachycardia. Hemoglobine 11 gr/dl and other laboratory values were unremarkable. Two dimensional transthorasic echocardiogram revealed mobile thrombus in right atrium. However it could not be enough to understand its structure and to differentiate if it is free floating or pedunculated. For this reason three dimensional transesophageal echocardiography was performed. It was detected free floating actual tube like nature of the mass (Figure 1). The patient undergone cardiac surgery in order to excise mass (Figure 2). After cardiac surgery patient remained stabil and discgarged with warfarin. She is regularly checked in out patient clinic. A Free floating mass in the right atrium is a very rare type of intracardiac mass which can obstruct artioventricular orifice. This type of mass can cause emboli, cardiac collaps, shock and sudden death besides less noisy symptoms as dyspnea, presyncope or syncope. It is important to realize the nature of the mass especially if there is not enough clue about its origin. Two dimensonal transthoracic echocardiography is very useful to detect the mass however it might not be enough to understand its structure and to differentiate if it is free floating or pedunculated. Three dimensional transesophageal echocardiography may be more helpful for detectng more accurate assessment of the size, shape and composition of the mass.

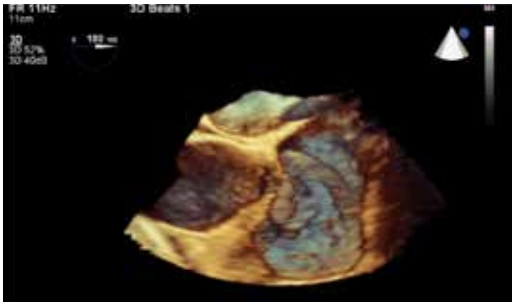

Figure 1. The three dimensional transesophageal echocardiography revealed the actual tube like nature of the mass.

Figure 2. The view of the thrombus after surgery.

OPS5-41

A good or bad luck? Sometimes low EF should be lifesaving

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Background: Acute aortic dissection was first described more than 200 years ago by Morgagni and since then has become the most common acute process related to aorta that requires surgery. The purpose of this paper is to report a chronic aortic dissection diagnosed and viewed with beta blockers because of the patient's refuse for surgery.

Case Report: A 65 year old man has admitted to our cardiology policlinic with worsening dyspnea. He was heavy smoker and his medical history included heart failure of about 8 years and hypertension. His physical examination was normal except bilateral basal rales and pretibial edeme in lower ekstremity. There was atrial fibrillation on electrocardiography and chest radiography showed widening of the mediastinum with fluid collection in the sinuses. These should be from the installing signs of failure we thought and we directed him to witdraw echocardiography. Transthoracic echocardiography showed aneurysmal dilatation, a dissection flap in the ascending aorta without pericardial effusion, global hypokinesia of left ventricle and severe aortic regurgitation (Video 1, 2). Computed tomographic angiography revealed an ascending aortic aneurysm and a type I DeBakey aortic dissection. The dissection involved the ascending aorta, aortic arch and descending aorta, terminating just below the renal arteries (Figure 1, 2). Urgent surgery was unavoidable but the patient was persistently refusing the operation. In this instance, surgical team quietly walked away and we were alone with the patient. A few hours later he was hospitalized unwillingly and beta blocker (100 mg metoprolol) was started. He was like a bomb, you could never know when would explode. During hospitalization we and especially he were lucky and he was discharged after 3 days with 200 mg metoprolol adding to his previous medical therapy. Despite all our prints he did not accept operation. Three years later he was alive, still painless dissected and not agreed for surgery. 'I got used to live with this' he was saying…

Discussion: Aortic dissection should be considered in the differencial diagnosis when catastrofic presentation, systemic hypertension and unexplained vascular origin findings and appropriate screening tests must be done urgently. The combination of transthoracic and transesophageal echocardiography can be used in the detection of intimal flap, dilated aortic root, trombus formation, expansion of the aortic wall, aortic regurgitation and pericardial effusion or tamponade. Despite the widespread availability of surgical and percutaneous treatment with the correct imaging modalities, in hospital mortality has still remained 30% and 58% in patients with type A dissection without surgical treatment. In this case, we reported a giant aortic aneurysm and chronic dissection of about 3 years without rupture or pericardial effusion or any neurological deficit believed to be dependend on low ventricular output and low systemic blood pressure.

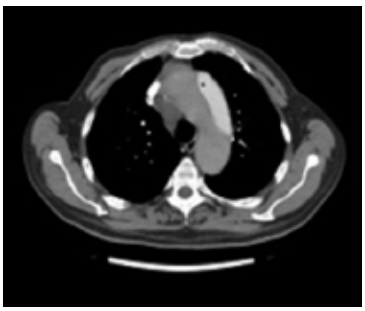

Figure 1. True and false lumen of dissection in arcus aorta at computed tomographic angiography.

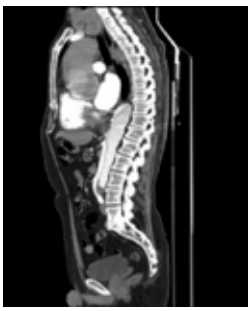

Figure 2. Aneurysmal dilatation of the ascending aorta and extending the dissection below the renal arteries.

32. ULUSLARARASI KATILIMLI TÜRK KARDİYOLOJİ KONGRESİ OLGU SUNUMLARI YAZAR DİZİNİ

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