TÜRK KARDİYOLOJİ DERNEĞİ
Kuruluş: 1963
Sağlıklı Kalplerle Geleceğe

31.
ULUSLARARASI KATILIMLI
TÜRK KARDİYOLOJİ KONGRESİ

22 - 25 EKİM 2015
MAXX ROYAL - ANTALYA

OLGU SUNUMLARI *CASE PRESENTATONS*

SÖZLÜ OLGU SUNUMLARI *ORAL CASE PRESENTATIONS*
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AVRUPA KARDİYOLOJİ AKREDİTASYON KURULU (EBAC)

31. Uluslararası Katılımlı Türk Kardiyoloji Kongresi Avrupa Kardiyoloji Akreditasyon Kurulu (EBAC) tarafından 18 saat uluslararası Dış Sürekli Tıp Eğitim kredi puanıyla kredilendirilmiştir. Her katılımcı sadece eğitim faaliyetiyle geçirilen saatlerin kredisini talep edebilir. EBAC, Avrupa Uzman Hekimler Birliği'nin (UEMS) bir kuruluşu olan Sürekli Tıp Eğitimi Avrupa Akreditasyon Konseyi (EACCME) ile işbirliği içinde faaliyet göstermektedir.

TTB STE KREDİSİ

Türk Kardiyoloji Yeterlilik Kurulu'nca 118 saatlik bilimsel etkinlik içerdiği saptanan 31. Uluslararası Katılımlı Türk Kardiyoloji Kongresi; Türk Tabipleri Birliği Sürekli Tıp Eğitimi Kredilendirme Kurulu tarafından 19 STE kredisiyle kredilendirilmiştir. Katılımcılar yalnızca barkod okuyucularla katılımları saptanan oturumların kredilerini -maksimum 19- kredi alabileceklerdir.

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Değerli Meslektaşımız,

Türk Kardiyoloji Derneği 52 yıl önce kurulmuş olup, 22-25 Ekim 2015 tarihleri arasında 31. Uluslararası Katılımlı Türk Kardiyoloji Kongresi'ni gerçekleştirmektedir.

Geçen yıl 3000'in üzerinde katılımın olduğu Kongremize yurt dışından, Avrupa Kardiyoloji Derneği üye ülkeleri, Komşu Ülkeler, Balkanlar ve Türk Cumhuriyetlerinden meslektaşlarımızın katılımı beklenmektedir.

Katılımcı sayısı ve yüksek kalitedeki bilimsel içeriği ile ulusal ve uluslararası düzeyde önde gelen bilimsel kongrelerden biri olan Türk Kardiyoloji Kongresinin zengin içeriğini tüm katılımcılarımıza doyurucu bir bilimsel program halinde sunmayı amaçlıyoruz. Uluslararası boyutu bu yıl daha da güçlenen Kongremiz yine hem TTB hem EBAC tarafından kredilendirilmiştir.

Bilimsel oturumlarda Türkiye'den ve Dünyadan, konularında büyük ağırlığı olan değerli konuşmacılar ve tartışmacılar yer alıyor. Uluslararası Dernekler ESC, ACC, EuroPCR, ICRR, EHRA ile ayrıca Kalp Damar Cerrahisi Derneği ve Sudi Arabistan Kalp Derneği ile ortak oturumlar programımızda yer almaktadır.

Kongre programımızda yer alan "Sempozyum", "Karşıt Görüş", "Nasıl Yapalım" oturumları ile kalp-damar hastalıklarıyla ilgili son bilgilerimizi güncelleyip tartışacağız. Geçen yıllardaki yoğun ilgi nedeniyle sayısını artırdığımız, özel sertifika verilen "Görüntülü İnteraktif Kurslar" ile bilgilerimizin yanı sıra becerilerimizi de geliştireceğiz.

Üç yıldır bize ev sahipliği yapan Maxx Royal Kongre Merkezi bu yıl Kongremizi dördüncü kez ağırlayacaktır.

31. Uluslararası Katılımlı Türk Kardiyoloji Kongresinde buluşmak dileği ve saygılarımızla.

Prof. Dr. Lale Tokgözoğlu
TKD Başkanı

Prof. Dr. Mahmut Şahin
TKD Gelecek Başkanı

TURKISH SOCIETY OF CARDIOLOGY
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To the future with healthy hearts

31ST TURKISH CARDIOLOGY CONGRESS
WITH INTERNATIONAL PARTICIPATION

Dear Colleagues,

We would like to welcome you to the 31st Turkish Cardiology Congress with International Participation which will take place between 22 and 25 October 2015 in Antalya.

Last year over 3000 participants participated in the Congress. This year we expect to receive a high level of international participation from member states to the European Society of Cardiology, from Neighbouring Countries, Balkan Countries and Turkic Republics.

The Turkish Society of Cardiology Congress with its high quality scientific content and large number of participants offers an up to date scientific program to its participants. The international dimension of the Congress is strengthened further this year; and both the Turkish Medical Association and EBAC has accredited the Congress.

Opinion leaders -both from Turkey and abroad- will participate in the scientific sessions. You will also have the chance to participate in joint sessions organized in collaboration with the international societies; such as ESC, ACC, EuroPCR, ICRR, and EHRA.

We will discuss and update our knowledge on cardiovascular diseases in different sessions in the format of "Symposium", "Debates" and "How to Sessions". We will also improve our skills through "Interactive Courses" with a certificate provided to course participants. We increased the number the courses thanks to the intensive demand experienced in the last years.

The congress will be held again in the Maxx Royal Congress Center, in the beautiful city of Antalya.

We hope to see you at 31st Turkish Cardiology Congress with International Participation.

Looking forward to a productive meeting...

Prof. Dr. Lale Tokgözoğlu
President

Prof. Dr. Mahmut Şahin
President Elect

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SO-01

Refrakter sık ventriküler taşikardi atakları olan bir ARVD hastasında ranolazin etkisi

İlknur Can[1], Azmi Eyiol[2], Venkat Tholanakanhalli[3]

[1]Necmettin Erbakan Üniversitesi Meram Tıp Fakültesi, Konya
[2]Beyhekim Devlet Hastanesi, Konya
[3]University of Minnesota, USA

Aritmojenik sağ ventrikül displazisi (ARVD) olan hastalarda ventriküler taşikardiler (VT) sıklıkla ortaya çıkmakta ve çok sayıda intrakardiyak defibrillatör (ICD) tedavisi ile sonlanmaktadır. Bu hastalarda en etkili olarak kullanılan amiodaron'un uzun dönemde mortal olabilen yan etkileri mevcuttur. Refrakter VT atakları olan hastalarda son yıllarda bir geç Na kanal blokeri olan ranolazin'in minimal yan etki ile oldukça etkili olduğu bildirilmektedir. Biz ARVD tanısı olan ve sık VT atakları olan hastamızda ranolazin tedavisi ekledikten sonra VT ataklarının tamamen kaybolduğunu gözlemledik. Vaka sunumu: ARVD tanısı olan 44 yaşında erkek hastaya dökümante VT atağı nedeni ile Kasım 2014 tarihinde ICD implante edilmiştir. Hastanın medikal tedavisine amiodaron eklendikten sonra, takip eden aylarda hastada defalarca ICD tedavisi (6 kez VF zonunda şok tedavisi) gerektiren hızlı VT atakları olması üzerine hastaya Şubat 2015 tarihinde endo- ve epikardiyal ventriküler taşikardi ablasyonu yapılmıştır. İşlemden sonra hastanın VT atakları devam etmiş, yalnız işlem sonrası VT hızı 220 civarından 170-194/dak ya düşmüştür. Hasta amiodaron tedavisine devam ettiği halde Mayıs 2015 yılında merkezimize tekrar başvurduğunda hastanın VT atakları için ICD'nin takılmasından itibaren 157 kez antitaşikardi pacing (ATP) tedavisi aldığı görülmüştür. Özellikle hasta ablasyon işlemi sonrasında ICD şok tedavisi almadığı halde sık VT atakları nedeni ile baş dönmesi şikayeti olmuştur. Hastanın tedavisine bu aşamadan sonra ranolazin 2x500 mg eklenmiştir. Hastanın 2 haftalık izleminde hiç VT atağı izlenmemiştir. Sonuç: ARVD hastalarında ranolazin'in VT atakları üzerine başarılı etkisi daha önce bildirilmemiştir. Literatürde ARVD tanısı olan bir hastada refrakter VT atakları üzerine etkisiz olduğu bildirilmiştir. Medikal tedavi altında olan ve ablasyon tedavisi işlemi de dahil seçenek kalmayan refrakter VT atakları olan hastamızın ranolazin tedavisi sonrasında 2 haftalık takibinde hiç VT atağı görülmemiştir.

SO-02

Sağ-sol koroner kuspid birleşim yerinden kaynaklanan ventriküler ekstrasistollerin başarılı ablasyonu

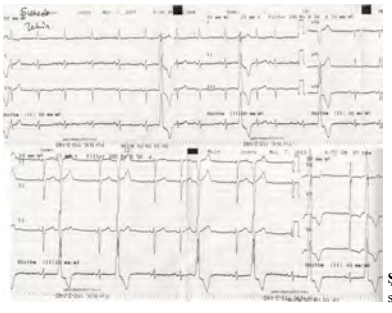
İlknur Can, Halil İbrahim Erdoğan, Alpay Arıbaş

Necmettin Erbakan Üniversitesi Meram Tıp Fakültesi, Konya

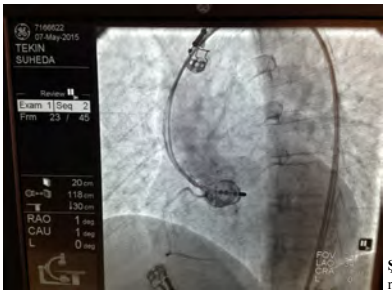
Giriş: Ventriküler miyokardiyal uzantıların ventrikülo-arteryel bileşkenin ötesine pulmoner arter ve aortaya uzantığı gösterilmiştir. Bu uzantılar koroner kuspidlerin bazaline ve hatta kuspidlerin birleşim yerlerine kadar uzanabilmektedir. Sol ventrikül çıkım yolu ekstasistoleri daha sıklıkla koroner kuspidlerden kaynaklanırken daha nadiren de kuspidlerin birleşim yerlerinden kaynaklanır.

Olgu: Çarpıntı şikayeti ile başvuran 44 yaşında kadın hasta yaklaşık 3 yıldır bu şikayeti için metoprolol 50 mg kullanmakta idi. EKG'sinde tespit edilen tipik sık ventriküler ekstasistolleri (VES) (Şekil 1) ve 24 saatlik Holter kaydında 24.000 adet VES olması üzerine ablasyon işlemi planlandı. Hastanın metoprolol tedavisi kesildikten sonra laboratuara alındı. Sağ femoral ven ve sağ femoral arterden giriş yapıldı. 4mm uçlu ablasyon kateteri ile öncelikle sağ ventrikül çıkış yolu tarandı. Erken nokta bulunamadığı için sol ventrikül çıkım yolu haritalaması için sağ femoral arterden retrograd aortik yöntemle sol ventrikül çıkım yoluna ulaşıldı. Detaylı haritalama sonucunda en erken noktanın sağ koroner kuspid ile sol koroner kuspidin birleşim yeri olduğu tespit edildi (Şekil 2). Bu noktada VES sırasında bipolar ablasyon kateteri yüzey EKG deki VES'in 34 ms önündeydi. Ablasyon öncesi aortografi ile koroner ostiumlardan güvenli mesafede olduğumuz tespit edildi. 30w, 50 derece ile radyofrekans ablasyon uygulandı. İlk 10 saniyede VES'lerin kaybolduğu görüldü. 1 saat bekleme süresi sonrasında ve taburculuğu takiben yapılan 3 günlük ritm Holter kayıtlarında hiç VES izlenmedi. Hastanın kullanmış olduğu metoprolol kesildi. Hasta takibe alındı.

Sonuç: Sol ventrikül çıkım yolu ekstrasistolleri sağ veya sol koroner kuspdlerden daha nadiren bu kuspidlerin birleşim noktasından kaynaklanabilir. Bu bölge için tipik EKG özellikleri olan V1 de QS, V2 nin inen kolunda çentik ve geçiş noktasının V3 olması özellikleri bizim hastamız da mevcut idi.



Şekil 1. EKG; ablasyon işlemi öncesinde sık VES.



Şekil 2. Aortagrafi; ablasyon kateteri koroner kuspid birleşiminde.

SO-03

A rare case of arrhythmogenic right ventricular cardiomyopathy co-existing with isolated left ventricular non-compaction

Burcu Akyol, Nizamettin Selçuk Yelgeç, Ahmet Taha Alper, Ahmet İlker Tekkeşin, Ceyhan Türkkan

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

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a clinical entity characterized by ventricular arrhythmias and a specific right ventricular pathology. On the other hand isolated left ventricular non-compaction is a sporadic or familial cardiomyopathy characterized by prominent trabeculae and deep intertrabecular recesses in left ventricle. Coexistence of these cardiomyopathies at one patient is extremely rare. A male patient 36 years of age presented for frequent ventricular tachycardias of RVOT origin. A cardiovascular MRI test revealed a non-compacted myocardial layer at apical anterior and apical lateral segments with an end diastolic non-compacted to compacted myocardial ratio of greater than 2.3. But right ventricular volumes and wall motion was normal. During ablation procedure we noticed two different left bundle VT morphologies which suggested the possibility of ARVC. This patient met one major and three minor criteria of current Task Force criteria of ARDC (T wave inversion in V1-6 derivations as major criteria, VT of RV outflow configuration and, more than 500 ventricular extra systoles per 24 hours by holter and ARVC confirmed by current task criteria in a second-degree relative as minor criteria), but the coexisting left ventricular non-compaction is a coincidental finding which has no active role in this patient's arrhythmias. All arrhythmias were originated from right ventricle. In early stages of ARVC, imaging findings might be negative. On the other hand non-compaction demonstrated by MRI might be silent. Again this case demonstrates the importance of diagnostic evaluation by various invasive and noninvasive techniques and detailed analysis of clinical history and presentation to establish the presence and type of heart disease and the origin and pathology causing lethal arrhythmias. Reliance to only imaging findings may result in misdiagnosis of the origin of arrhythmias.

Figure 1. Long axis MRI; long axis view of heart showing non-compaction.

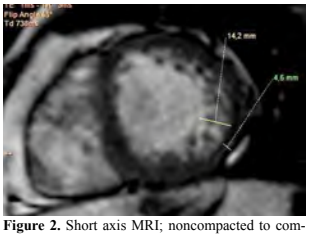


Figure 2. Short axis MRI; noncompacted to compacted ratio greater than 2,3.

SO-04

Brugada syndrome; cause of syncope during tens therapy

Abdulmelik Yıldız[1], Mustafa Budancamanak[1], Cennet Yıldız[2]

[1]Ekotom Medical Center, İstanbul
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Introduction: Brugada syndrome is an genetic disorder characterized by sudden cardiac death and associated with mutations of SCN5A gene, which encodes cardiac sodium channel. Fever can precipitate Brugada-like electrocardiographic changes. Syncope and sudden death usually ocur at rest or during periods of bradicardia. TENS is the application of low-level electrical current through special electrodes placed on the skin for analgesia.

Case Report: 38 year old male patient with preauricular abcess developed presyncope during high-grade fever and diagnosed as having Brugada syndrome with type 1 ECG pattern. His Electroencephalography was normal. Coronary angiography showed myocardial bridging of the left anteriyor descending coronary artery after second diagonal branch and no hemodynamically significant coronary artery disease. Technesium Myocardial Perfusion SPECT and Cardiac Magnetic Resonance imaging results were normal. Ajmaline test confirmed the diagnosis, and ventricular fibrillation induced during electrophysiological study. There was no family history of sudden death. The patient diagnosed with left shoulder adhesive capsulitis and TENS was applied for 20 min a constant frequency of 80 HZ for pain relief. During therapy the patient experienced syncope, treated in emergency department and returned to normal within a few minutes. The patient was monitored and ECG recordings were taken at 1, 5, 10 and 15 minutes during second session. ECG recordings showed bradycardia and dynamic ECG changes between type 1 and type 3 pattern. After ten minutes the patient developed presyncope. TENS treatment was stopped and subsequent sessions have been cancelled.

Discussion: Brugada syndrome, an entity described in 1992, is characterized by episodes of ventricular tacyarrhythmias or unexpected sudden cardiac death in patients with a structurally normal heart, and a characteristic ECG consisting of a right bundle-branch block pattern and ST segment elevation in the precordial V1 through V3 leads. Ventricular arrhythmias more often occur during rest or sleep when the vagal tone is predominant. Increased vagal tone mediated by acetylcholine decreases the calcium currents, which could lead to arrhythmogenesis via phase 2 reentry. During TENS low-level electrical currents delivered across the intact surface of the skin via electrodes. According to the " Gate Control Theory" of pain, TENS treatment is thought to activate the body's pain inhibitory system. It also stimulates the release of endogenous opioids. This low-level electrical currents affect sodium and potassium channels in conduction system and generation of action potential in muscles, nerves and heart. TENS treatment leads to an increase in parasympathetic activity, resulting in bradycardia. It is known that local increase in body temperature and release of endogenous endorphins during therapy may cause sleepiness. Increased vagal activity has been associated with the development of cardaic arrhythmias in Brugada syndrome. Fever-induced Brugada syndrome is a well-known entity. In the presented case, there was no conclusive evidence for VF as a potential cause of syncope. Dynamic ECG changes between type 1 and type 3 pattern with bradycardia during second session, a prolonged QTc interval without hypotension could however be interpreted as supporting evidence for VF.

SO-05

Do cardiologists need education of optimisation and follow-up of implantable cardiac devices?

Çağın Mustafa Üreyen, Şakir Arslan, Göksel Çağırcı, Cem Yunus Baş, İsa Öner Yüksel, Deniz Demirci, Duygu Ersan Demirci, Ahmet Serbülent Savcıoğlu

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A 37-year-old male patient with dilated cardiomyopathy came to our outpatient pacemaker clinic for routine follow-up. A CRT-D was implanted 6 months ago in an another hospital to diminish heart failure symptoms and number of hospitalizations. His EF was 27% and moderate mitral regurgitation and a dilated left ventricle were ascertained on echocardiography. He was taking 10 mg of ramipril, 100 mg of metoprolol succinate, 25 mg of spironolactone and 40 mg of furosemide once a day. On questioning, he denied any symptomatic recovery after CRT-D implantation. His physician told the patient that he may not benefit from CRT and up to 30% of CRT patients are non-responders. Interrogation of CRT-D demonstrated that pacing thresholds, sensed P/R wave amplitudes, lead empedances and battery voltage were all normal. However, percentage of biventricular pacing was 81% and it was a low level to benefit from CRT-D. The basic rationale of RV pacing is to pace minimum. On the other hand, the logic is completely opposite in patients with CRT. The higher the pacing ratio, the better the efficacy of CRT-D. Thus, accepting this patient as nonresponder was entirely an inconvenient consideration. AVI was 150 msec and it was set to 120 msec. Moreover, metoprolol succinate was recommended as 100 mg b.i.d. by virtue of the fact that mean heart rate was 83/min despite 100 mg of metoprolol. Hence, intrinsic AV conduction time was also prolonged and more optimal average heart rate was achieved. Recording of mean biventricular pacing ratio was initialized to see the effect of resetting and medications. After three months, the patient was again evaluated. Biventricular pacing ratio was 98% and the patient described a partial amelioration of his complaints. He described that he was still not able to do heavy duties, albeit he could now take his daughter to her school and take her to playground. This case emphasises the importance of follow-up and optimisation of implantable cardiac devices that should be executed by cardiologists who possess adequate knowledge and experience for better follow-up and prognosis. Adequate knowledge and experience necessitates an incorporated education of optimisation and follow-up of cardiac devices into education of cardiac device implantation. This incorporated education would solve the problem of unfavorable and inconvenient optimisation and follow-up of patients with implantable cardiac devices.

SO-06

Evaluation of Tp-e interval and Tp-e/QT ratio in patients with mitral annular calcification

Abdulmecit Afşin, Mahmut Yılmaz, Şeyda Değer, Hasan Pekdemir

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Objective: Mitral annular calcification (MAC) is a common clinical entity in the elderly population and it is regarded as a manifestation of cardiovascular disease. Recent studies have shown that prolongation of the interval between the peak and the end of T wave on electrocardiogram (Tp-e), which is accepted as an index of transmural dispersion of ventricular repolarization, and Tp-e/QT ratio are associated with ventricular arrhythmia. In the present study, we aimed to evaluate ventricular repolarisation by using Tp-e interval and Tp-e/QT ratio in patients with MAC.

Material and Method: Fifty patients with MAC (27 females and 23 males; mean age 71.6±8.0 years) and 50 patients without MAC (26 females and 24 males; mean age 69.3±6.2 years) were included in the study. All participants underwent detailed pyhsical and transthoracic examinations. Maximum and minumum QT and Tp-e intervals as well as corrected values according to heart rate were calculated from 12-derivations electrocardiography. QT dispersion and Tp-e / QT ratio were calculated. All parameters were compared between groups.

Results: Patients with MAC had significantly higher values of Tp-e interval (75,8±11,6 vs 62,1±8,7; p<0,001), cTp-e interval (84,9±14,3 vs 67,5±9,7; p<0,001), Tp-e/QT ratio (0,19±0,02 vs 0,15±0,02; p<0,001), cTp-e/QT ratio (0,19±0,03 vs 0,15±0,02; p<0,001), cQTmin (390,1±31,5 vs 373,8±26,1; p=0,006) when compared to control subjects. There were positive correlations between E/Em ratio and cTp-e interval (r=0,396; p=0.004), and between E/Em ratio and cTp-e/QT ratio (r=0,535; p<0,001) in the MAC group. Further, positive correlations were found between left atrium and cTp-e interval (r=0,50; p=0,001), left atrium and cTp-e/ QT ratio (r=0,30; p=0,028) in the MAC grup. QTmax and QTd was similar between groups.

Conclusion: According to our study findings, patients with MAC had elevated Tp-e interval, cTp-e interval, Tp-e/QT ratio and cTp-e/QT ratio in comparision to those without MAC.

SO-07

Giant coronary aneursym causing acute anterior myocardial infarction

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A 70-year old man with hypertension was admitted to our coronary ICU with acute anterior MI. Emergent primary PCI was planned and coronary angiography was performed. LAD artery was totally occluded in the proximal segment just after a huge 24x16 mm sized aneursym (Figure 1). Emergent CABG operation was performed in 75 minutes to this patient because of multivessel disease including the RCA and left circumflex artery. Aneurysm was ligated and coronary by-pass was performed using LIMA and saphenous grafts. The post-operative course of the patient was uneventful. He was discharged with medical therapy including ASA, clopidogrel and atorvastatin. He was asymptomatic at his polyclinic visit in the first month.

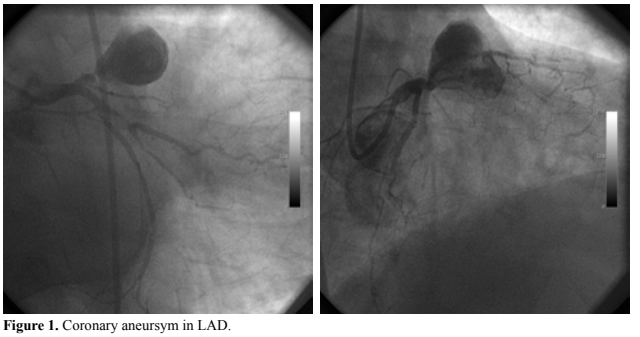

Figure 1. Coronary aneursym in LAD.

SO-08

Acute ruptured fibroatheromatous plaque in left main artery causing no ischaemia

Cengiz Öztürk, Turgay Çelik, Atila İyisoy, Şevket Balta, Mustafa Demir, Hamidullah Haqmal, Ahmet Öztürk

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A 81-year-old male patient with no prior medical history admitted to our hospital typical chest pain since 24 hours. He was diagnosed acute coronary syndrome without ST segment elevation. His ECG demonstrated minor T wave inversions in the precordial leads and his blood tests were normal except slightly elevated (serial) troponin I. Transthoracic echocardiogram was also normal and no regional wall motion abnormality was observed. In coronary angiography a dissected and ruptured atherosclerotic plaque formation in the left main coronary artery (LMCA) and severe stenosis in the first diagonal artery were appeared (Figure 1). To confirm the diagnosis, intravascular ultrasound (IVUS) examination was performed and a ruptured and empty plaque was observed with no thrombus (Figure 2). Because of the well preserved lumen area, intensive pharmacotherapy was decided to continue. The acute rupture of a plaque in the LMCA was not causing myocardial ischaemia/infarction in the anterior left ventricular wall. The culprit lesion was the first diagonal artery. Thereafter, a drug-eluting stent was implanted to the first diagonal artery and the patient was subsequently treated as NSTEMI (Figure 3). The article discusses different management strategies in patients with confirmed ruptured and empty plaque in LMCA.

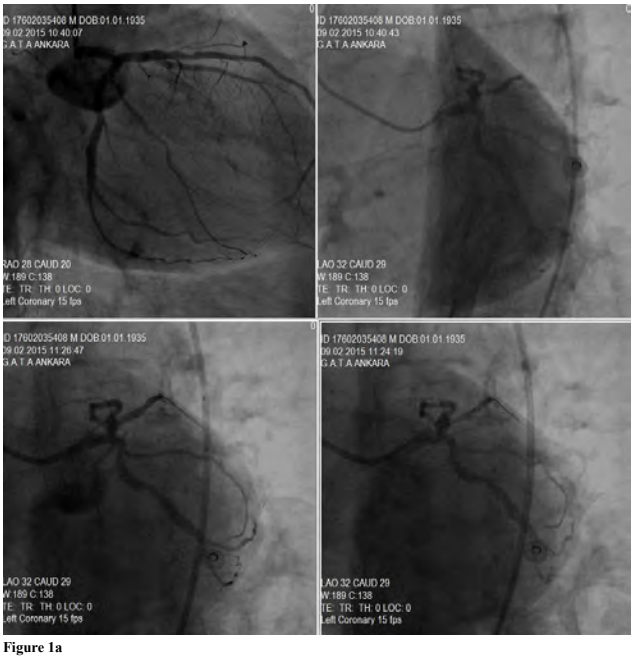

Figure 1a

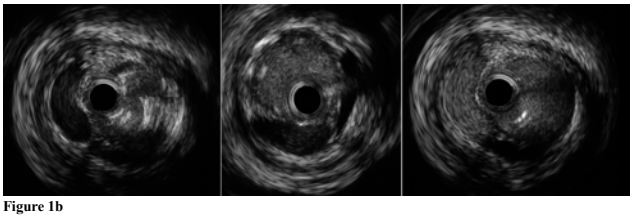

Figure 1b

SO-09

Recurrent spontaneous dissection in different coronary arteries

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[2]*Malatya State Hospital, Malatya*

Spontaneous coronary artery dissection (SCAD) is a rare but potentially lethal event commonly affecting young women. Accordingly, limited evidence is currently available regarding its pathophysiology, prevalence, associated conditions, diagnosis, management and prognosis. In here, we represent a young patient with recurrent SCAD in different coronary arteries. A 31-year-old female presented complaining of 2 hours of continuous burning chest pain, radiating into the left arm, associated with dyspnea. She had no prior medical problems. There was no history of any connective tissue disease and no significant family history of ischemic heart disease. An electrocardiogram revealed anterolateral myocardial infarction. Urgent coronary angiography showed a dissection of the proximal left anterior descending artery (Figure 1) treated successfully by multiple overlapping stents. After one week, she was admitted to coronary care unit with repeat chest pain and acute pulmonary edema. A repeat angiogram showed 60-70 % proximal stenosis of the LAD (pre-stent) and 80% proximal stenosis of the second diagonal branch. She underwent successful bypass surgery using a saphenous vein graft to the diagonal and LIMA to LAD. Following the uneventfull one year course, the patient presented again further episode of chest pain. An ECG revealed inferior ST segment elevation. Urgent coronary angiography demonstrated a long dissection of the RCA with total occlusion of the posterior descending artery (Figure 2, Video 1). All grafts were patent and the patient were treated by two overlapping stents resulted with restoration of Thrombolysis in Myocardial Infarction (TIMI) III flow (Video 2). The long-term outcome of patients who survived their SCAD event is generally good, however, they are at risk for recurrent dissection and major cardiovascular events like in our patient, and thus should be closely monitored by cardiovascular specialists.

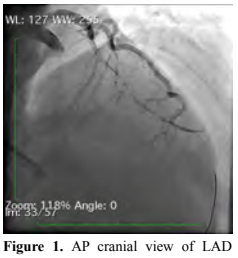

Figure 1. AP cranial view of LAD artery.

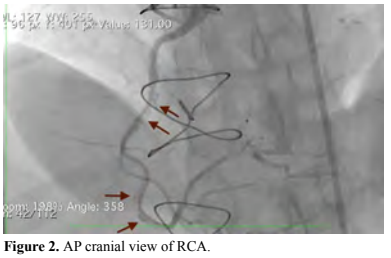

Figure 2. AP cranial view of RCA.

SO-10

Coronary artery dissection due to electrical shock resulted in miyocardial infarction

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Electric shock induced myocardial infarction is a rare condition. Electric shocks can lead to a wide vairety of clinical conditions from skin burns to fatal arrhythmic complications. Coronary angiogram of those patients are usually normal. Coronary thrombosis after electric shock has been accused of this consequence. The right coronary artery is the most affected artery. It's believed to be due to the location of the right coronary artery, which is in close proximity to the chest wall. This case is about a 49 years old female patient presented with myocardial infaction right after an electric shock. The coronary angiogram revealed a congenital double right coronary artery and coronary dissection. According to best of our knowledge, this is first case in the literature. The complete revascularization is achieved by implantation of a bare metal stent in the dissection site.

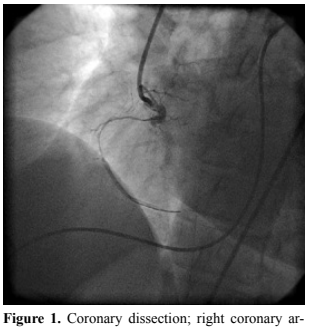

Figure 1. Coronary dissection; right coronary artery dissection.

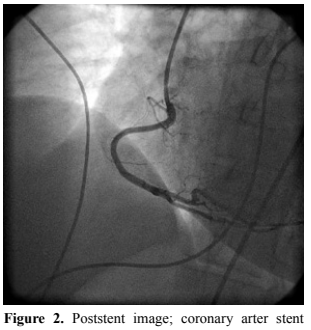

Figure 2. Poststent image; coronary arter stent implantation.

SO-11

Acute anterior myocardial infarction associated with clomiphene citrate in a young woman who want to become pregnant

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[2]*Ankara University Faculty of Medicine, İbni Sina Hospital, Ankara*

Background: Clomiphene citrate is a drug which is commonly used in female infertility. It works by stimulating ovulation in a women who does not ovulate or who ovulates irregularly. Generally clomiphene citrate is recognized as safe for ovulation but sometimes it may cause life-threatening side effects.

Case: A 36 year-old women who had been prescribed clomifene citrate for ovulation was admitted to hospital for chest pain lasting for two hours. She had never smoked, she did not have any cardiac risk factor for myocardial infarction and there was no family history for coronary artery disease. An electrocardiogram showed diffuse st elevation in leads v1-v4 with resiprocal changes in leads d2, d3 and avf. She was taken to catheter laboratory and coronary angiography revealed occlusion of the midportion of left anterior descending artery (LAD) with heavy thrombus burden. Circumflex and right coronary arteries were normal. After balloon dilatations, a 2.75x15 mm drug eluting stent was implanted in mid part of LAD. The patient had an uncomplicated recovery. Maximal values of troponin and CPK were 50 ng/ml and 347 U/ml respectively. An echocardiogram performed before hospital discharge revealed apical akinesis, anterior, lateral hypokinesis and an ejection fraction of 45% with mild mitral regurgitation.

Conclusion: Although clomiphene citrate is relatively safe drug for ovarian stimulation it may also be associated with serious side effects such as myocardial infarction. Physicians should be aware of the potential risk of clomiphene citrate, especially in patients with associated risk factors for coronary artery disease.

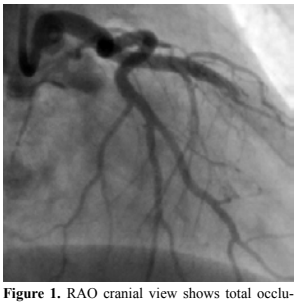

Figure 1. RAO cranial view shows total occlusion of LAD.

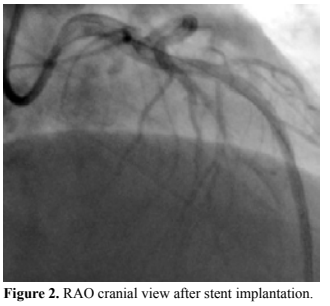

Figure 2. RAO cranial view after stent implantation.

SO-12

Acute myocardial infarction in young woman with a history of systemic lupus erythematosus and kidney transplantation

Sayyed Hamed Moghanchzadeh, Taner Ulus, Yüksel Çavusoğlu, Bülent Görenek, Necmi Ata

Eskişehir Osmangazi University Faculty of Medicine, Eskişehir

A 22- year- old female patient with a history of systemic lupus erythematosus (SLE) for 10 years and kidney transplantation for 5 years admitted to emergency department with non-spesific chest pain. Her medications included tacrolimus 1 mg, two time a day and methylprednisolone 6 mg one time a day. ECG on admission showed an elevation of the ST segment in anterior and inferior leads (Fig 1). High sensitive Troponin I levels were elevated to 1.98 ng/ml. In the first hour of the onset of the chest pain, primary percutaneous coronary intervention (PCI) revealed a total occlusion of the proximal left anterior descending (LAD) artery (Fig 2a). A 4.0x26 mm bare metal stent implanted in the site of occlusion and distal flow of the LAD was reestablished. (Fig 2b). After PCI the patient was treated with tirofiban and unfractionated heparin infusion. Physical examination revealed no spesific findings. Her chest and back pain was relied. Laboratory tests revealed a creatinin of 1.62 mg/dl, LDL 52 mg/dl. On the fifth day of addmision an echocardiogram revealed an akinesia in the apex and apicolateral region of the left ventricle and ejection fraction was 33%. TThe tacrolimus dose was continued, but the methylprednisolone dose was reduced to 4 mg once a day by a nephrolog. The renal ultrasonography and Doppler revealed no patological finding in the transplanted kidney. Patient was treated medically with aspirin 100 mg, clopidogrel 75mg and metoprolol 50mg once daily in addition to her current medications. Any ischemic recurrence was detected over the three-month follow-up. The incidence of MI in young SLE patients due to premature atherosclerosisis 9-50 times higher than in age-matched controls. Again, kidney transplantation and chronic corticosteroid therapy may also lead to premature atherosclerosis. For these reasons, our young woman patient could suffer MI. Agresive risk factor modification must be considered in such patients.

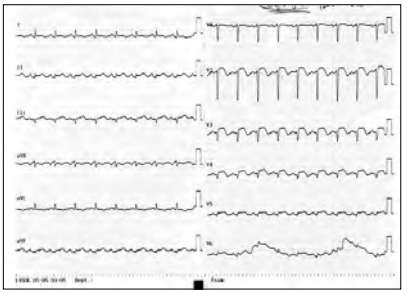

Figure 1. The initial ECG; ECG: electrogram.

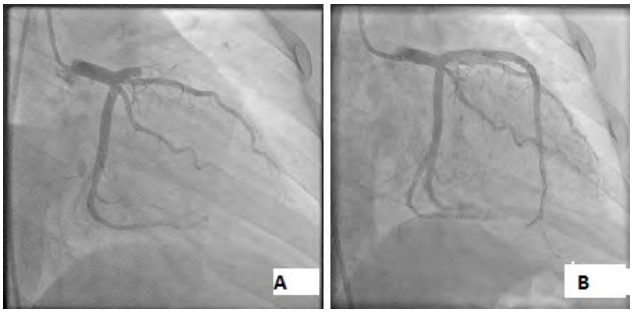

Figure 2. Coronary angiograms; coronary angiograms before and after intervention.

SO-13

Cardiac calcified amorphous tumor in a patient with end-stage renal failure

Ahmet Gürdal, Füsun Helvacı, Mutlu Çağan Sümerkan, Hakan Kilci, Şükrü Çetin, Turgun Hamit, Kadriye Kılıçkesmez

Şişli Etfal Training and Research Hospital, İstanbul

Background: Primary cardiac tumors are rare, with the most common being atriyal mixomas. However, not all cardiac masses are neoplasms. We present a very rare non-neoplasmic cardiac mass originating from atrial side of mitral valve.

Case: 48–year-old woman was referred to our hospital by doubt of infective endocarditis. She had been on hemodialysis for 5 years because of diabetic nephropathy. Transthoracic echocardiography revealed a hyperdense floating mass on atriyal side of mitral valve, and transesophageal echocardiography showed hyperdense, homogeneous mass which was measured 12x3 mm that originated from the annulus of the anterior commissure of the mitral valve and extending through the left atriyal cavity. On physical examination, there was no evidence of infectious endocarditis and laboratory data showed no evidence of an inflammatory reaction. Pre-diagnosis of the mass was Calcified Amorphous Tumor because of history of hemodialysis. The patient was scheduled for surgery, but the patient refused the operation.

Discussion: Calcified amorphous tumor of the heart is extremely rare and most commonly originates in the endocardium of the right or left ventricle. Its pathogenesis has yet to be established. It is commonly believed that these lesions develop secondary to the calcification of the intramural thrombus. Abnormal calcium metabolism due to renal dysfunction and the inflammation associated with hemodialysis may contribute to the rapid growth and pathological change. Calcified amorphous tumor is especially considered in patients with end-stage renal disease and hemodialysis.

Figure 1. Transesophageal echocardiographic scan in patient.

SO-14

Treatment approach to an incidental paracardiac mass (paraganglioma) during ST elevation myocardial infarction

Gökhan Özmen, Ahmet Tütüncü, Alper Karakuş, Burcu Çavlan, Mehmet Demir, Hasan Arı

Bursa Yüksek İhtisas Training and Research Hospital, Bursa

Case Report: 55 years old man admitted to our emergency department with typical chest pain in last 3 hours. ECG showed ST segment elevation on precordial leads and sinus tachycardia. Urgently coronary angiogram and percutaneous balloon angioplasty was performed to proximal LAD artery thrombotic oclusion and TIMI 3 flow restorated. During coronary angiography, we also detected an extracardiac structure that is supplied from left main coronary artery (Video 1). Because of possible surgical resection for the vascular structure stent was not deployed to lesion. Transesophageal echocardiography (figure 1a) depicted 3x4,5x3 cm paraaortic mass and was confirmed by BT scan (figure 1b). Cardiac surgery was carried out with left mammarian artery to LAD and mass was resected from posterolateral aspect of ascenden aorta above the sinotubular junction. Intraoperatively mass was invasing to aortic posterior wall (figure 1c). Because of strict invasion to the wall mass excised with 1 cm nondiseased assendan aortic tissue and dacron patch sutured to defect area. Any other tissue invasion was not seen macroscopically. Mass feeding arteries were ligated. Subsequently aortic clemp removed and saphen veins anostomosed to right coronary and left circumflex coronary arteries. According to histopstological assesment the mass diagnosis was paraganglioma (figure 1d).

Conclusion: Paragangliomas may be diagnosed incidentally and the histology of a paragangliomas cannot be completely confirmed via non-invasive methods. Because of the tumours invasive nature, surgical resection is the treatment of choice for paragangliomas.

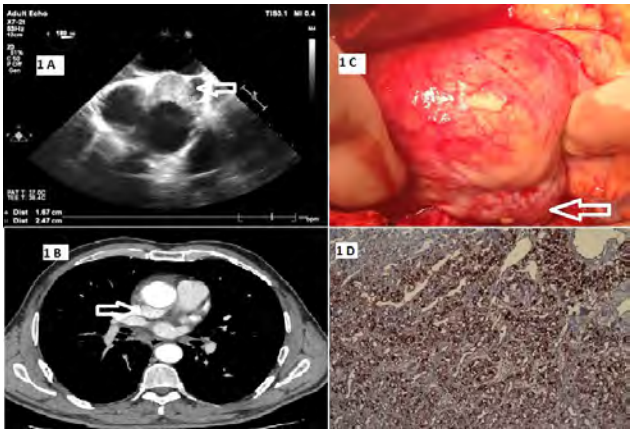

Figure 1. (A-D) Images of the mass.

SO-15

Ring-shaped calcific constrictive pericarditis

Bernas Altıntaş, Mehmet Zülküf Karahan, Derya Deniz Altıntaş, Rojhat Altındağ, Barış Yaylak, Erkan Baysal, Erol İnci, İlyas Kaya

Diyarbakır Training and Research Hospital, Diyarbakır

A foreign 72-year-old man who had Diabetes Mellitus and Hypertension was admitted to Emergency Room with severe chest pain and dyspnea. Physical examination upon arrival was found to be unremarkable with a pulse rate of 70 beats/minute and a blood pressure of 115/75 mmHg. Initial 12 lead electrocardiogram (ECG) revealed sinus rhythm minimally ST segment elevations in leads DII, DIII, aVF and V4-V6 whitout reciprocal ST segment changes. Upon emergency bedside transthoracic echocardiographic examination Inferior wall motion abnormalities were detected. The echocardiogram revealed a constrictive physiology of the mitral and tricuspid valve and pericardial thickening. The early diastolic velocity of lateral mitral annulus and that of septal annulus was not reduced in Tissue Doppler. The patient was referred to emergency coronary angiography with the diagnosis of acute coronary syndrome. The Coronary Angiography (Figure 1) showed Coronary Artery Disaese (Two Vessel Disease) and massive calcification developed along the atrioventricular groove between the atriums and ventricles. Volume Rendering Images of MultiSlice Cardiac CT (Figure 2, 3) definitely demonstrated a ring-shaped massive calcification along atrioventricular groove causing strangulation of the heart. The patient underwent an extensive pericardial resection and cardiopulmonary bypass surgery. The calcification of the atrioventricular groove was removed carefully to prevent an injury of the right coronary artery.

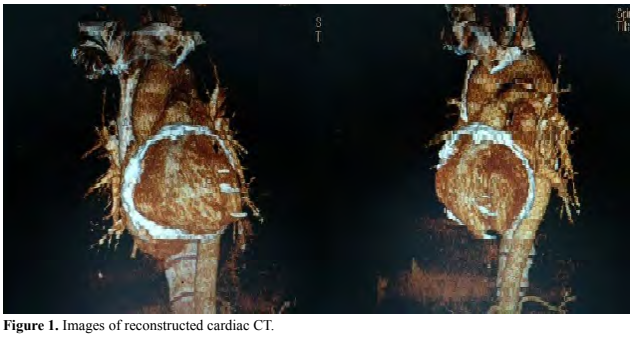

Figure 1. Images of reconstructed cardiac CT.

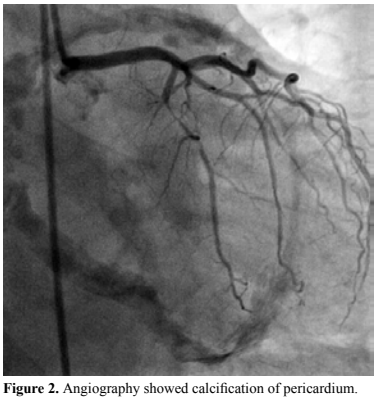

Figure 2. Angiography showed calcification of pericardium.

SO-16

The association of severe aortic stenosis and narrow aortic root in a young patient;what is the etiology: rheumatic valvulitis or lambl excrescence?

Cengiz Öztürk, Hamidullah Haqmal, Şevket Balta, Sait Demirkol, Murat Ünlü, Ali Osman Yıldırım, Mustafa Demir, Turgay Çelik, Atila İyisoy

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Aortic valve stenosis is the most common cause of left ventricular outflow obstruction in children and adults. We report a case that who had severe aortic stenosis and narrow aortic root. A 21-year old male patient was admitted to our outpatient clinic because of chest pain related exercise, exertional angina, decreased exercise tolerance, dyspnea and exertional dizziness since one year. His body mass index was 21,5 kg/m^2. Physical examination revealed a apical 4/6 systolic murmur typically with a mid-systolic ejection murmur at the aortic area with radiation to the right neck. Fourth heart sound (S4) was heard. Also carotid pulse anacrotic (parvus et tardus) was present. Systemic blood pressure was 120/80 mmhg and pulse rate was 87 beats/min. There was diffuse T wave changes and left ventricular hypertrophy on electrocardiogram. Transthoracic echocardiography (TTE) showed increased interventricular septum thickness (septal hypertrophy as 15 mm) and also showed severe aort stenosis with and moderate aortic regurgitation. There was 154 mmHg transaortic peak gradient and aortic valve area was 1.4 cm^2. Transeosophageal echocardiography (3D TEE) also showed a thickened and decreased mobility of the right coronary cuspis. There was also a membrane extending along the entire valve region like a lambl excrescence. Tomographic angiography showed normal coronary anjiogram and aort diameter at the level of the sinus valsalva level was 27.5x28 mm. Aortic annulus diameter was 17x16 mm and area was 320 mm^2 and ascending aorta diameter was 35.5 mm. Cardiac Magnatic Resonance Imaging (MRI) showed that aortic valve was tricuspid and right coronary cuspis movement was limited. According to the results, the severity of the gradient and anatomic features of the aortic root and valve were contradictory (Figure-1,Vide 01-2). Surgeons did not accept to performe valve replacement because of the small root. What what is the etiology: rheumatic valvulitis or lambl excrescence?

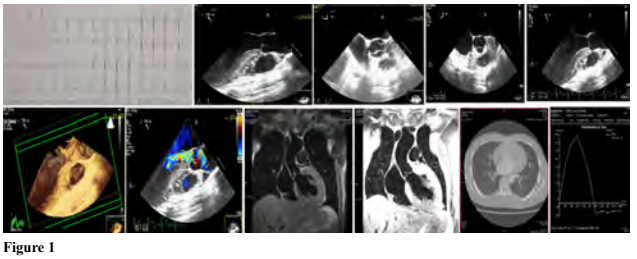

Figure 1

SO-17

Cardiac calcific amorphous tumor

Çetin Geçmen[1], Mehmet İnanır[1], Çağatay Önal[1], Gonca Gül Geçmen[2], Nihal Özdemir[1]

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62 year old male patient with a history of hypertension, dialysis dependent chronic renal failure and coronary artery bypass graft presented to our clinic with dyspnea. Transthoracic Echocardiographic assessment revealed moderate aortic stenosis (mean gradient: 32 mmHg), mild left ventricular systolic dysfunction (Global Left Ventricle Ejection Fraction: LVEF 45%) and mobile, hyperechogenic mass was measured 2.1x1.1 cm the posterior mitral leaflet (Panel A, Supplementary material online, Movie 1). For better delination, Transesophageal Echocardiography was performed. It clarified the mass was causing mild mitral regurgitation (Panel B, C). 3D Transesophageal Echocardiography confirmed the presence of a hyperechogenic mass attached to the P2 scallop of the posterior mitral leaflet (Panel D, Supplementary material online, Movie 2). On the surgery, the mass excised from the P2 scallop of the posterior mitral leaflet (Panel E) and aortic valve replacement using a mechanical valve was performed. On histologic examination; hematoxylin and eosin stained section of the 10x20 mm mass showed calcified nodules on an amorphous background of fibrin material and foreign body giant cells (Panel F). The findings consistent with cardiac calcified amorphous tumor.

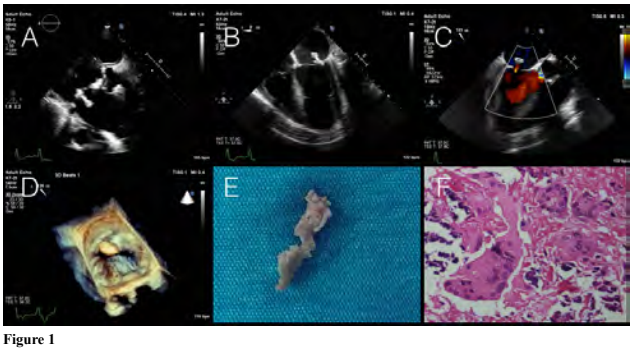

Figure 1

SO-18

3D imaging of an insidious enemy: malignant melanoma in heart with unknown primary origin

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Ege University Faculty of Medicine, İzmir

Introduction: Metastatic melanoma of unknown primary occurs in approximately 2%-5% of all cases of melanoma, and mostly seen in lymph node, lung, brain or gastrointestinal tract. Nevertheless, melanomas have propensity to metastase to the myocardium or pericardium. We represent you a case of aysmptomatic melanoma metastasis to the left atrium through pulmonary vein with unknown primary origin.

Case: A 41 year old female patient presented to the neurology outpatient clinic with headache and cranial CT revealed multiple metastatic carcinoma. PET scan showed 3.5x4.7 cm sized mass in left atrium which had a substantial fluorodeoxyglucose (FDG) uptake with a high maximum standardized uptake value (SUVmax) of 74.6 (Fig.1A). PET scan also revealed multiple lesions at liver, lung and brain without indicating a primary origin. At 3D transthoracic echocardiography a large left atrial mass was seen and transeosaphageal echocardiography (TEE) was performed for better identification of the mass. TEE revealed a 3x2.3 cm sized, mobile, pedunculated mass attached to the left atrial wall (Fig. 1B, C). A computer tomography guided tru-cut biopsy of the lung lesions was done and histopathological result was consistent with 'malignant melanoma metastasis' (Fig.1D). The patient underwent a surgical operation for the mass in the right lung, resulting with the resection of the mass inside the pericardial adipose tissue and the same histopathological diagnosis.

Discussion: Malignant melanoma of unknown primary origin is a rare condition. There is limited data about heart invasion in such cases. Nevertheless, regarding the high metastasis potantial, the importance of imaging techniques is increasing. 3D echocardiography allows better identification of masses and their relationships with cardiac chambers, their volumes and inner characteristics. In our case a large, mobile, pedunculated mass seen in left atrium was clearly defined by 3D echocardiography.

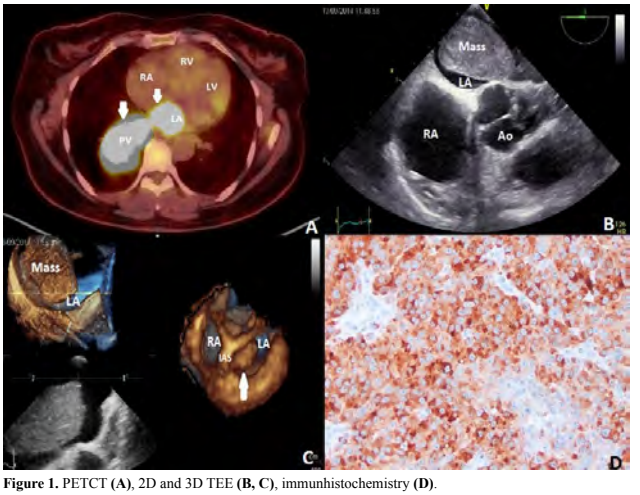

Figure 1. PETCT **(A)**, 2D and 3D TEE **(B, C)**, immunhistochemistry **(D)**.

SO-19

Multimodality imaging of a left ventricular aneurysm in a patient with normal coronary arteries: unusual localization

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Background: Left ventricular aneurysm (LVA) is rare but potentially fatal complication of miyocardial infaction. It has been strictly defined as a distinct area of abnormal left ventricular diastolic contour with systolic dyskinesia or paradoxical bulging. Diagnosis may be established by transthoracic echocardiography (TTE), left ventriculography, computed tomographic angiography, and cardiac magnetic resonance imaging.

Purpose: Here we report a case of uncommonly located LVA in an adult patient with normal coronary arteries.

Methods: A 57 year old asymptomatic female presented to our outpatient department for cardiac check-up. There was no risk factor for coronary artery disease except for hypertension. She did not have any coronary artery or peripheral vascular disease in her history. On pysical examination blood pressure was 130/65 mmHg, lung sounds were clear and there was a grade 3/6 diastolic murmur on left parasternal focus. Sinus rhythm was determined in the 12 lead surface electrocardiography. ST-segment depression and pathological Q waves were not observed on her electrocardiography. Results: TTE showed an out-pouching showing dyskinetic motion arising from left ventricular inferolateral wall, left ventricular hypertrophy and mild aortic regurgitation (Fig. 1A). Subsequently coronary angiography was performed and revealed normal coronary arteries (Fig. 1B,C). Filling pattern was observed in left ventriculography during systole and diastole (FİG. 3A). Multidedector computerized tomography (Fig. 3B) and cardiac magnetic resonance imaging (Fig. 3C) showed aneurysm formation in inferolateral wall of left ventricle. Since the patient was asymptomatic, she was discharged with medical treatment.

Conclusion: Electrocardiography, TTE, MDCT, MRI and angiography are the comlementary diagnotic tools for LVAs and to differantiate them from other causes of left ventricular outpouching. The diagnose was based on multimodality imaging in the currrent case.

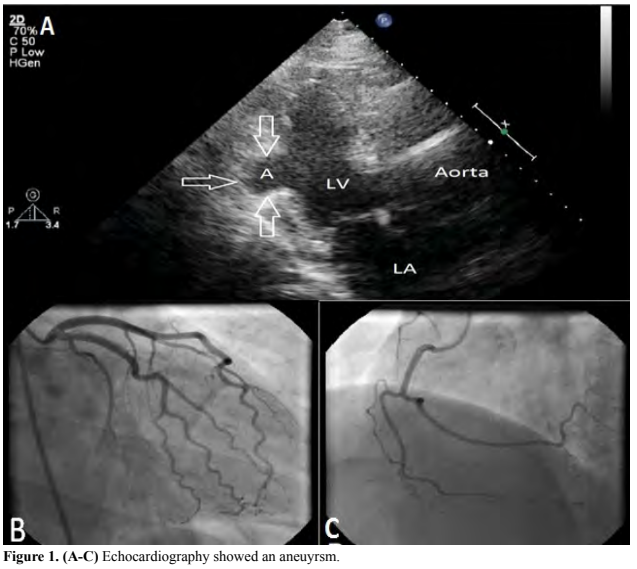

Figure 1. (A-C) Echocardiography showed an aneuyrsm.

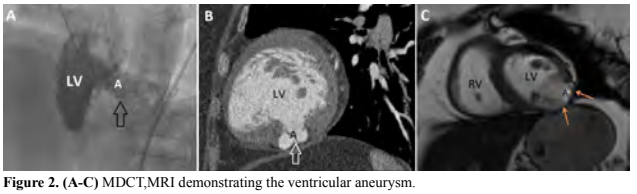

Figure 2. (A-C) MDCT,MRI demonstrating the ventricular aneurysm.

SO-20

Sol atriyal apandiste lokalize hidatik kist: vaka sunumu

Mutlu Çağan Sümerkan, Turgun Hamit, Ahmet Gürdal, Füsun Helvacı, Hakan Kilci, Şükrü Çetin, Gökhan Aksan, Serhat Sığırcı, Kudret Keskin, Süleyman Sezai Yıldız, Arzu Er, Kadriye Kılıçkesmez

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Giriş: Kist hidatik ekinokokus granülosumun neden olduğu ülkemizde Akdeniz Bölgesinde endemik olarak gözlenen parazitik enfeksiyondur. Enfeksiyon, kontamine olmuş gıda veya köpeklerle temas sonucunda bulaşır. Çoğunlukla klinik olarak asemtomatik seyretmekle birliklikte çok nadiren akut perikardit, tamponat veya kronik konstriktif perikardite, anaflaktik şok eve aritmi neden olabilir. Kardiyak kist hidatiğin tedavisi cerrahidir ve sonrası medikaldir zorunludur. Organizmalar bulaş sonrası en sık karaciğer olmak üzere, akciğere,dalak, kaslar, santral sinir sistemi ve göze yerleşebilmektedir. Çok nadir olarak kalbi tutmaktadır (%0.5-2). Kalpte en sık sol ventrikül duvarını tutar. Daha sonra sağ ventrikül ve interventriküler septum yerleşimi görülür. Literatürde kalbin birçok bölgesinin hidatik kist tarafından tutulumu yayınlanmış olmasına rağmen, atriyal apandis tutulumu gösterilmemiştir. Biz vakamızda sol atriyal apandisin hidatik kist tutulumunu göstermekteyiz.

Olgu: Altmışiki yaşında, erkek hasta ateş etyoloji araştırılması amaçlı enfeksiyon hastalıkları kliniğinde takibe alındı. Özgeçmişinde 30 yıl önce hidatik kist nedeniyle akciger operasyonu geçirmiş. Geliş kan basıncı 110/70 mmHg idi. Elektrokardiyografisinde 94 atım/dakika normal sinüs ritmi izlendi. Transtorasik ekokardiyografik incelemesinde,sağ ve sol kalp sistolik fonksiyonları normal, mitral valv prolapsusu, hafif aort, orta mitral ve trikuspid yetersizliği, orta mitral darlığı, pulmoner hipertansiton (50 mmHg), dilate sol atrium saptandı. İnfektif endokardit açısından ileri değerlendimek amaçlı üç boyutlu transözafagial ekokardiyografik incelemesinde sol atriyal apandiste genişleme (çap: 3.2 cm) ve içerinde duvara yapışık hareketli kistik yapı izlendi (Video 1). Biyokimyasal değerlendirmeler sistemik hidatik kisti destekler yönde saptandı. Hastaya operasyon önerildi ancak hasta kabul etmediğinden medikal takip kararı alındı. Hastanın bir yıllık izleminde takibinde herhangi bir patoloji izlenmedi.

Sonuç: Vakamız göstermektedir ki hidatik kist enfeksiyonunda atriyal apandis lokalize olarak tutabileceği bilinmeli, sistemik kist hidatik enfeksiyonu olan hastalarda kardiyak kist hidatiğin araştırmasında ekokardiyografik incelemede rutin görüntülemeler yanında atrial apandisinde ayrıntılı incelemesi gerekmektedir.

Kardiyak görüntüleme / Ekokardiyografi

SO-21

Noncoronary sinus to right atrium fistula in a a young patient: a rare complication of native aortic valve staphylococcus aureus endocarditis

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Infective endocarditis (IE) is the infection of the endocardial surface of the heart that may involve the heart valves most commonly but may ocur at the site of a septal defect, on chorda tendinea, or on mural endocardıum. IE has a high risk of morbidity and mortality, and prognosis is highly dependent on complications. A fistulous tract formation from the aortic sinus to the right atrium due to an infective endocarditis (IE) is very rare complication. Here, we describe a rare case of a 21-year-old refugee woman with systemic embolization (serebral and splenic infarction) and fistula between aorta and right atrium due to S. aureus IE. Patient with a known surgery history of cesarean five months ago was admitted to our clinic because of persistent fever, abdominal pain, weakness and chills. On physical examination at our hosipital revealed the following: her temperature was 37,5 C, blood pressure 90/50 mmHg, heart rate 130/min, a respiratory rate of 25/min. Her initial 12-lead electrocardiogram (ECG) on admission showed sinus tachycardia. On neurologıc and abominal examination, there was hypoestezia on her right half of face and she was tender to palpation in the left upper quadrant and defense present. Laboratory investigations revealed severe anemia, leukocytosis and increased C reactive protein level. TTE revealed a vegetative growth (−0,5X0,6) over the aortic leaflet with severe aortic valve regurgitation. Vegetations floated dynamically on the septal leaflet of tricuspit valve and the aortic valve leading to the suspicion of an aortic annular abscess. Continuous wave and color doppler echocardiography revealed a fistula between the aortic noncoronary sinus and right atrium. Abdominal CT revealed encapsulated hypodence lesion and splenomegaly which were suggestive of splenic infarction. Computarized tomography imaging of the brain revealed cerebral infarction.Patient hospitalized to coronary intensive care unit. Immediately three temporally distinct sets of blood cultures were obtained at least 1 hour interval and empirical therapy with high dose intravenous ampicillin, gentamicin were initiated. The following day, Staphylococcus aureus was found as underlying pathogen. She underwent aortic valve replacement and complex noncoronary sinus fistula pericardial patch reconstruction and repair.The patient was taken into intensive care after the surgery operation. Proceed with antibiotic therapy and coumadinized. The patient in whose transthoracic echocardiography was not aorta-atriyal shunt observed and discharged from the hospital after 2 weeks post op. In her first control after a month there were not any findings of infective endocarditis. In conclusıon we successfully treated the infective endocarditis of young refugee with combined medical and surgical treatment which is seldom seen; consisted between noncoronary cusp-right atrium,complicated with fistula and abscess formation- cause poor hygie.

SO-22

Mitral-aortic intervalvular fibrosa aneurysm with rupture into left atrium: a 3D trans-esophageal echocardiographic approach

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Introduction: Mitral-aortic intervalvular fibrosa (MAIVF) pseudo-aneurysm is detected by echocardiography as a pulsatile structure at the mitral-aortic junction. Depending upon the severity of fistulization, acute mitral deficiency like clinical condition may develop. Although Trans-Thoracic Echocardiography (TTE) and Trans-Esophageal Echocardiography (TEE) both have proven to be valuable in diagnosis and management of the condition, a 3D TEE approach has the potential to provide important clues that may prove crucial for the management of MAIVF.

Case Presentation: A 44-year-old male patient complaining of high fever and dyspnea was admitted to our emergency service department. Although vital signs were normal, a systolic murmur of grade 4/6 was detected at the apex during physical examination. A cardiology consultation was requested subsequent to detection of pericardial effusion and hepatosplenomegaly by a thoraco-abdominal computerized tomography scan. Bicuspid aortic valve and mid-eccentric aortic valve regurgitation were observed in 2D-TTE. Additionally, an aneurysm image at the MAIVF area and an eccentric turbulent flow secondary to a fistulization from the same towards the posterolateral side of the left atrium was also observed (Video 1). Using TEE, an aneurysm of dimensions 0.9x2.0 cm was detected at the MAIVF region. Using the 3D assessment, a segment bulging towards the mitral valve in diastole was also observed. There was no detectable structural defect of the anterior mitral valve leaflet (Video 2). Blood cultures were positive for Streptococcus spp. The patient subsequently underwent surgical intervention. MAIVF, from left atrium to aortic root, was restored using bovine pericardium and aortic valve replacement was also performed.

Discussion: MAIVF is more prone to trauma like infective endocarditis because of its relatively avascular structure. Although, the leading etiological causes of MAIVF are infection and surgical trauma, aortic regurgitation flow jet is also a contributor to its formation. In this patient, both infection and aortic regurgitation was considered to be responsible because diastolic MAIVF bulging in TEE images showed barotrauma to the area. 3D TEE played an important role in designing of surgical strategy for this patient because there was no effect on any segments of the anterior leaflet. In conclusion, 3D TEE has the potential to make valuable contributions in designing surgical strategies as it can accurately evaluate mitral valve involvement which is crucial in planning mitral valve interventions in MAIVF patients.

SO-23

Successful management of an unusual complication during subclavian artery stenting: incomplete stent expansion

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Case report: A 56-year-old man with a history of coronary artery by-pass surgery and left upper extremity claudication was admitted to our hospital. Selective subclavian angiography revealed 80% stenosis in the left subclavian artery (LSA). Subclavian artery stenting was planned. LSA was cannulated with the help of 8 Fr Judgkins right guiding catheter. Then, 8.0x30 mm balloon-expandable Scuba stent was deployed. However, although the balloon was inflated until 15 atm (RBP), incomplete expansion of mid to distal part of the stent was observed (Figure 1). Because of completely expanded proximal part of the stent, we could not pull back. Firstly, we progressed our guiding catheter into completely expanded proximal part of the stent to prevent its migration. Then, firstly we try to pass from the inside of the stent with the help of 7.0x50 mm Pyxis-V balloon compatible with 0.035 inch guide-wire but we couldn't succeed it. Thereafter, we took a lower profile 6.0x60 mm Glidex flex balloon, which compatible 0.018 inch guidewire, to pass across the stent. We planned to exchange guidewire. However, due to the risk of passing through the stent strut, primarily a hydrophilic 4.0 Fr STS catheter was used to pass within the stent over 0.035 inch guidewire. Over this guide wire, firstly unexpanded stent was dilated by using 6.0x60 mm Glider Flex Over the Wire balloon and complete stent expansion was achieved. After that balloon angioplasty was performed with 7.0x50 mm Pyxis-V balloon the stent was successfully implanted. The final angiogram showed no signs of remaining stenosis (Figure 2).

Discussion: Incomplete stent expansion can potentially lead to vascular occlusion, thrombosis, and limb ischemia. In this case, we want to describe an endovascular method dealing with this unusual complication during subclavian stent deployment. Our technique seems to be an easy, elegant solution and should be utilized in such an unusual complication.

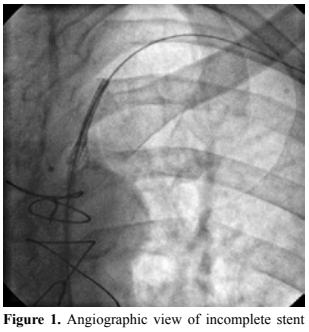

Figure 1. Angiographic view of incomplete stent expansion.

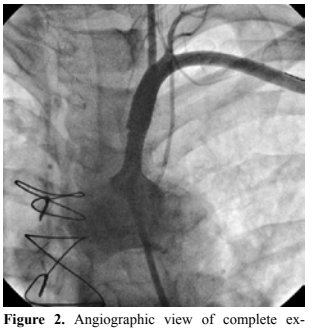

Figure 2. Angiographic view of complete expanded stent.

SO-24

Percutaneous approach to treatment of totally occluded abdominal aortic stent graft

Alptuğ Tokatlı[1], Ersan Tatlı[2], Mehmet Akif Çakar[2], Murat Aksoy[2], Harun Kılıç[2], Hüseyin Gündüz[2], Ramazan Akdemir[2]

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[2]*Sakarya Training and Research Hospital, Sakarya*

Introduction: We, herein, report a case of EVAR graft occlusion in which percutaneous angioplasty was performed.

Case: A 58-year-old female patient, admitted to our clinic with claudication and signs of ischemia in her lower extremities. The patient had ischemic rest pain without tissue loss. She had been under medical treatment with cilostazol and clopidogrel. She had absent femoral, popliteal and pedal pulses on both sides. Past medical history revealed an endovascular abdominal aortic stent graft implantation due to aortic aneurysm three years earlier. Diagnostic angiography demonstrated that the stent graft was totally occluded. As the patient denied surgery, endovascular treatment was planned. 6 Fr sheath to left brachial artery and 8 Fr sheaths to both femoral arteries were advanced. A 5 Fr 125 cm HH1 (Merit) catheter was advanced from the left brachial route and we were able to penetrate to the total occlusion site. 0,035 inch Terumo stiff glidewire (Radifocus, Terumo, Tokyo, Japan) with micro catheter (Minie 0.035 inch-135 cm/VSI-USA) were advanced to both of the common femoral arteries. For externalization of both of the guide wires coming from the brachial route, we advanced 7F right Judkins catheters via the femoral sheaths. After externalization, we went on the procedure from both of the femoral routes, brachial access was used just for imaging. Both externalized guide wires were advanced with the guiding catheters to thoracic aorta. Two 7.0x100 mm and 9.0x40 mm balloons (Pyxis-VQ/Qualimed GmbH-Germany) were placed and inflated simultaneously at 6 to 10 atmospheres. 14x60 mm (ALTOS XL/AndraTec GMBH-Germany) balloon was placed to abdominal aorta and inflated at 8 atmospheres. Control angiogram revealed that successful percutaneous treatment.

Conclusion: Although the general approach to the occluded endovascular abdominal aortic stent graft is surgery, balloon angioplasty and if necessary stenting can be successfully applied and recommended way of treatment.

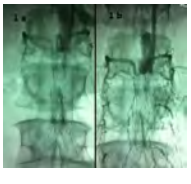


Figure 1. (A, B) Totally occluded abdominal aortic stent graft.

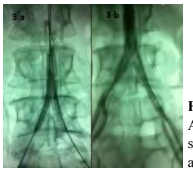


Figure 2. (A, B) Angiography shows successfull balloon angioplasty.

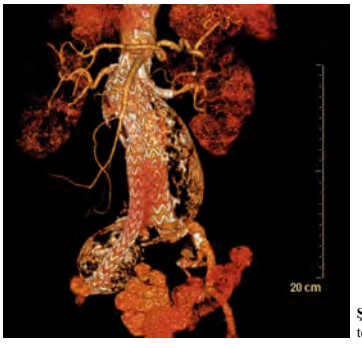
SO-25

Sol renal arteri kapsayan abdominal aort anevrizmasının Chimney Yöntemi ile tedavisi

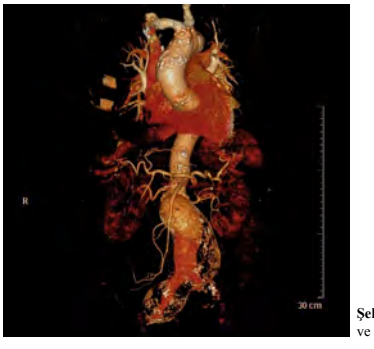
Beytullah Çakal, Sinem Çakal, Oğuz Karaca, Mehmet Onur Omaygenç, Ekrem Güler, Hacı Murat Güneş, Filiz Kızılırmak, Bilal Boztosun

İstanbul Medipol Üniversitesi, İstanbul

Yeterli boyun uzunluğunun ya da tutunma bölgesinin olmadığı abdominal aort anevrizması olgularının tedavisinde 'Chimney Metodu' aortik dal oklüzyonunu engellemek için geliştirilmiştir. Bilinen HT, geçirilmiş SVO öyküsü olan 78 yaşında erkek hasta karın ağrısı ve batında pulsatil kitle izlenmesi üzerine abdominal aort anevrizması tanısı ile tarafımıza refere edilmiş. Torakoabdominal BT anjiyografide sağ renal arterin 7 mm altında sol renal arteri de içine alan en geniş yerinde 78 mm ölçülen parsiyel tromboze anevrizma izlendi. Hastaya konvansiyonel EVAR yönteminin uygun olmaması sebebiyle Chimney yöntemi ile anevrizmanın tedavisi planlandı.Her iki femoral arterden kateter yerleştirildi. Sol brakial arterden 6 Fr Shuttle kateter suprarenal aortaya ilerletildi. 6 Fr 90 cm Destination kateteri içerisinden 4 Fr vertebral kateter sol renal artere yerleştirilerek 0,035 tel renal arter distaline gönderildi. 8x59 mm greft stent sol renal artere, 36x20x170 mm aortik uzatma greft stenti sol femoral yoldan sağ renal arterin hemen altına yerleştirildi. Önce renal stent sonra uzatma greft açıldı. Aort ve renal arterdeki stent balonları ile final kissing yapıldı. Anevrizmanın sağ iliak arteri de içine alması sebebiyle 16x13x199 mm greft stent proximaldeki uzatma grefti ile birleşecek şekilde yerleştirilerek balon ile post dilatasyon yapıldı. Son görüntülemede sol renal arterdeki stentin açık olduğu görülmekle beraber hafif tip 1A endoleak izlendi. Tekrarlayan kissing balon işlemi yapılarak son görüntüde minimal sızıntı olduğu görüldü. Her iki kasık perkütan kapama cihazı Proglide vasıtasıyla kapatıldı. Hasta yatışının 2. gününde komplikasyon olmadan taburcu edildi. Taburculuk sonrası 1. ayda çekilen kontrol BT anjiyografide işlem sonrası görülen minimal endoleakin de kaybolduğu görüldü.



Şekil 1. Chimney sonrası 3D BT; sol renal arterde Chimney greft ve aortadaki greft.



Şekil 2. Abdominal aort anevrizması; Sol renal ve sağ ana iliak artere uzanan anevrizma.

SO-26

Retrograde recanalisation of popliteal artery occlusion

Mehmet Ali Astarcıoğlu, Taner Şen, Halil İbrahim Durmuş, Basri Amasyalı

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A 71-year-old man presented with a history of intermittent claudication (Rutherford class IV) lasting two years. After an arterial duplex study revealing popliteal occlusion, endovascular therapy was pursued. Informed consent was obtained from the patient. Digital subtraction angiography was then performed and the popliteal artery was found to be completely occluded over 15 cm, with filling of distally via collaterals. Both the tibioperoneal and posterior tibial arteries had proximal stenoses with the posterior tibial artery providing dominant flow to the foot. It was decided to treat these lesions using balloon angioplasty via retrograd approach. Initially, an antegrade access site was secured with a 8-F contralateral sheath. Under road-map guidance, a 18-G needle was used to puncture the anterior tibial artery at the level of the higher dorsum of the foot. After the vessel was punctured, 5-F introducer sheath (Merit Medical Systems, USA) was positioned. The proximal anterior tibial and popliteal lesions were then crossed from below using a 0.035 inch hydrophilic nitinol guide wire (SP Medical, Denmark) and a 4-F straight catheter (Merit Medical Systems, USA). After administering 10000 units of heparin, the retrograde wire was snared and brought through the antegrade guide catheter. The rest of the procedure was then performed in a conventional manner using an antegrade common femoral approach. Balloon angioplasty of the proximal anterior tibial and popliteal lesions was performed, using 3.5*100 mm and 5.0*120 mm balloon catheters (Boston Scientific/Medi-Tech, USA) with prolonged (3–5 min) inflations at low pressure (6 atm), respectively. The proximal ATA and tibioperoneal trunk was stented with a 3.5*28 mm zotarolimus-eluting stent (Medtronic, Brescia, Italy) to optimize result. The patient's subsequent hospital stay was uneventful, with persistence of both pedal and anterior tibial pulses and immediate relief of rest pain.

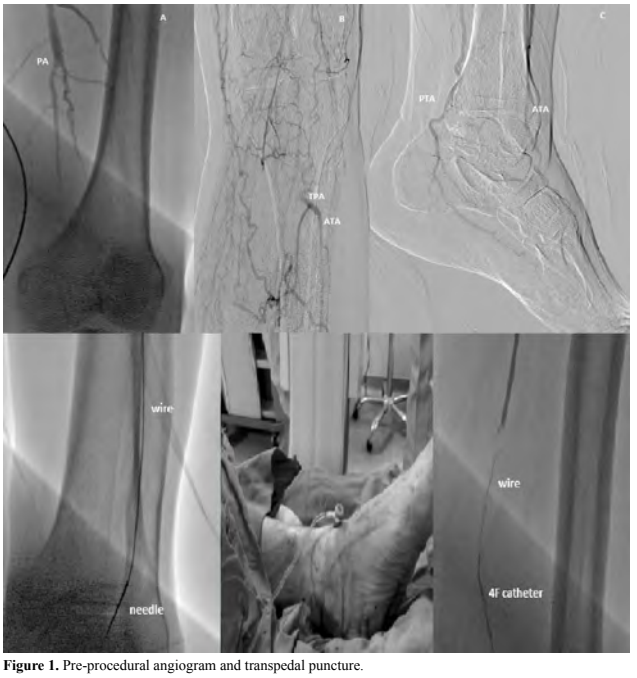

Figure 1. Pre-procedural angiogram and transpedal puncture.

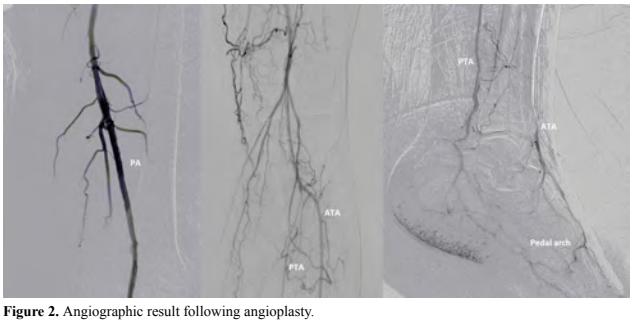

Figure 2. Angiographic result following angioplasty.

SO-27

Successful transcatheter retrieval of the embolized coronary stent during percutaneous coronary intervention

Mustafa Yurtdaş[1], Nesim Aladağ[1], Yemlihan Ceylan[1], Yalın Tolga Yaylalı[2], Yüksel Kaya[1]

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The embolization of coronary stent into peripheral circulation during percutaneous coronary intervention (PCI) may cause complications, including increased morbidity and mortality. A 52-year-old man presented to emergency department with acute inferior ST-elevation myocardial infarction (STEMI). After premedication with antiaggregant and anticoagulation, the patient emergently underwent a coronary angiography which showed thrombotic occlusion of distal part of the proximal right coronary artery (RCA). After the lesion was predilated with a 2.5x12 mm sized balloon, a zotarolimus-eluting stent (ZES) of 3.0x18 mm was implanted. Full patency was achieved, but a second lesion was found just proximal to the first stent which was considered long and significant after intracoronary nitroglycerine administration. Thus, the decision was made to stent the proximal part as well. While advancing, the stent was stuck and could neither be advanced through the lesion nor be withdrawn. Therefore, the whole system was withdrawn to the site of femoral sheath where the stent was detached from its balloon. The patient had chest pain and the right leg pain. Lower extremity angiogram displayed that the stent migrated down to the right anterior tibial artery (RATA) (Figure 1). First, the proximal part of RCA was stented with another ZES of 3.5x30 mm using a 7 F AR-1 guiding catheter without any further complication. After removel of 7 F sheath, an 8 F sheath was inserted through an antegrade puncture of the ipsilateral common femoral artery. A 4 F multi-snare set was advanced into the RATA and the stent was captured with the microsnare (Figure 2). Then, the whole system was removed from the femoral artery. Post-angiographic course was uneventful with no signs of cardiac or lower limb ischemia. This case illustrates that primary PCI can be complicated by an embolized stent resulting in acute leg ischemia and the embolized stent can be managed successfully by transcatheter technique.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

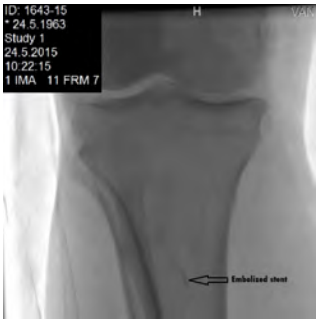

Figure 1. Embolized stent in right anterior tibial artery.

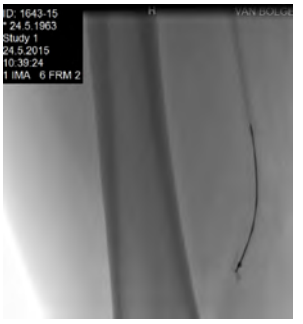

Figure 2. The embolized stent was captured with microsnare.

SO-28

Dizaltı perifer arter hastasında Revers Cart Tekniği ile her iki tibial arterin revaskülarisazyonu

Ahmet Seyfeddin Gürbüz, Müslüm Şahin, Mehmet Vefik Yazıcıoğlu, Süleyman Çağan Efe, Mehmet Fatih Yılmaz, Alev Kılıçgedik, Muhsin Türkmen, Cevat Kırma

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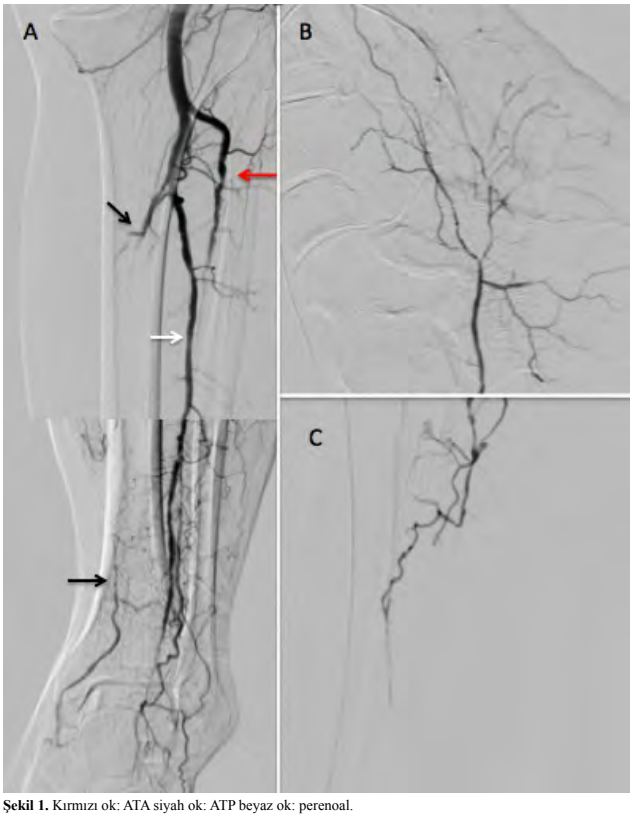
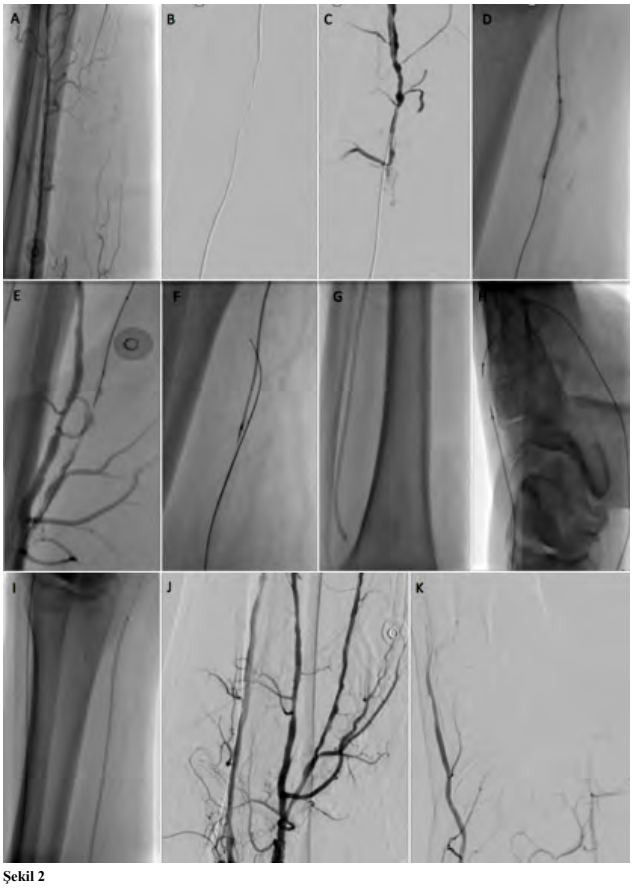
Olgu: 79 yaşında sol bacakta Fontaine Evre 3 (istirahat ağrısı) perifer arter hastalığı olan 1 yıl önce geçirilmiş CABG ve AVR öyküsü olan erkek hastaya perifer revaskülarizasyon planlandı (Şekil 1A). Bir önceki seansta antegrad yaklaşımla ATP'nin Fielder XT, Conquest Pro 9 teller ile geçilemediği bilinmesi üzerine retrograd teknik planlandı. Sol femoral antegrad ponksiyon ile, sağ femoral retrograd ponksiyon ile bilateral 7F sheat yerleştirildi. Antegrad yerleştirilen sheat içinden Minie destek kateteri (0,014 inch) ve Regalia XS 1.0 (0,014 inch) tel ile total tıkalı ATA geçildi. Regalia XS 1.0 plantar arktan ilerlememesi üzerine tel değiştirilerek Fielder XT-R ile ark geçildi. Minie kateter yardımı ile ATP retrograd görüntülendi (Şekil 1B, C).ATP'nin total oklude segmentinden retrograd Fielder XT tel ilerlememesi üzerine mikrokateter değiştirilerek Corsair mikrokateter yerşetirildi ve tel Astato XS 20 ile değiştirildi. Astato XS 20 ile proksimal lumene düşülememesi ve subintimal ilerlemesi üzerine kontralateral taraftan corsair mikrokateter ve Astato XS 20 tel ile antegrad yaklaşımla subintimal distale dogru ilerlerlendi (Şekil 2A-C) Ardından antegrad tel üzerinden 2,0x20mm balon ilerletildi. Balon dilatasyon sonrası retrograd Astato XS 20 tel ile proksimal lümene geçildi (Şekil 2D, E). Corsair desteği ile Astato XS 20 tel femoral artere kadar ilerletildi (Şekil 2F). Astato XS 20 (300cm) snare ile yakalanarak kontralateral femoral sheatten externe edildi. Astato XS 20 tel üzerinden ATA 2,0x150mm balon ile 5 dk dilate edildi (Şekil 2G). Ardından iki taraflı Corsair yerleştirildi ve içinden iki taraflı floppy wire geçildi (Şekil 2H) ATA 2,0x150 mm balon ile 5 dk dilate edildi (Şekil 2I). Diseksiyon olmaması ve akımın iyi olması nedeniyle işlem başarıyla sonlandırıldı (Şekil 2J, K). Takiplerinde hastanın istirahat ağrısı geriledi.

Sonuç: Dizaltı perifer arter girişimlerinde zor vakalarda 'revers CART' tekniği kullanılanilir.



Şekil 1. Kırmızı ok: ATA siyah ok: ATP beyaz ok: perenoal.



Şekil 2

SO-29

Percutaneous coil embolisation of an arteriovenous fistula of the lower extremity caused by penetran trauma

Mustafa Topuz, Mehmet Çoşgun, Ömer Şen, Murat Çaylı

Adana Numune Training and Research Hospital, Adana

Arteriovenous fistulas (AVFs) are unusual connections between the arterial and venous system that bypass the normal anatomic capillary beds. We present a case of an arteriovenous fistula of the lower extremity caused by a penetrating injury. Color-Doppler ultrasonography of the affected limb showed left deep femoral vein aneurysm without intraluminal thrombi, associated with an AVF of the left profunda femoris artery. The diagnostic catheterization of the affectted limb showed a high-flow AVF between the left profunda femoris artery and left femoral vein with deep femoral vein aneurysm diameter with 9x4 cm (Figure 1a). Before the procedure the patient received 80 units/kg of Unfractionated Heparin. The 6 F Judkins guiding cathether was inserted into first perforating of left profunda femoris artery. After a 0.014 inch floppy guidewire was introduced in to the aneurysm. 9 mm length Codman platinum microcoil twice the size of the fistula diameter was deployed successfully (Fig. 1b). The aim of treatment of an AVF is to isolate and close the site of arteriovenous communication.Although surgical closure of AVFs remains the gold standard, other treatment options include covered stent and coil embolization. Percutaneous embolization is an effective and minimally invasive alternative to open surgery. Although incomplete treatment of an AVF may lead to certain recurrence with a more complex architecture that is more troublesome and challenging to treat, the endovascular approach can be used safely, if the initial treatment is carefully planned and executed for a successful and durable result.

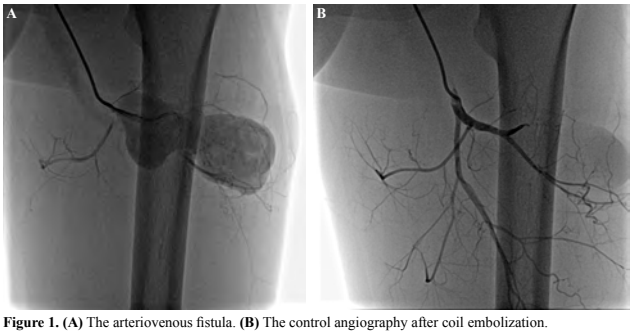


Figure 1. (A) The arteriovenous fistula. **(B)** The control angiography after coil embolization.

SO-30

Computed tomography guided percutaneous transapical closure of cardiac apex after prosthetic mitral paravalvular leak closure

Atila İyisoy, Cengiz Öztürk, Turgay Çelik, Uğur Bozlar, Şevket Balta, Mustafa Demir, Ali Osman Yıldırım, Sait Demirkol, Adem Güler

Gülhane Military Medical Academy (GMMA) Ankara, Ankara

The transapical approach (TAA) for percutaneous interventions is performed in high-risk patients with peripheral vascular disease and when transseptal attempt failed. We report a case of a successful percutaneous closure of the apical access without minithoracotomy with the computed tomography guidance. A 44 year old female was admitted to our department with severe dyspnea (NHYA classes II–III). She had hemolytic anemia and lactate dehydrogenase level was high. She had a history of mitral metallic prothesis valve replacement in 1994. Transesophageal echocardiography (TEE) showed two severe mitral paravalvular leaks (6 and 9 mm) of the mitral valve prosthesis. Firstly, we tried to close the leaks through the transseptal approach but we failed. We decided to try the TAA. Before the procedure, in order to obtain optimal puncture of the apical region of the left ventricle, a computed tomography was also performed. The radiologist marked the apex with an arrow at the sixth intercostal area and costachondral juntion with left anterior 30 degree projection (Fig 2). Under general anesthesia, TEE, CT and fluoroscopic guidance, transapical cannulation of the left ventricle from the sixth intercostal area and midclavicular region with a needle was performed and an 6 F sheath was inserted to the apical region. A Terumo hydrophilic wire was loaded on a right Judkins 6F catheter and passed through the defects. After the positioning of the catheter inside the left atrium (LA), the Terumo wire was replaced by an Amplatzer stiff wire. Then AL-1 guide the catheter to the LA. Through the guide catheter, a 10 mm x 5 mm Vascular Amplatzer plug was successfully deployed. There was no residual mitral paravalvular leak after deployment (Figs. 1). After that, the placement of the myocardial occluder (4 mm ADO-II) was performed through the delivery system, with no complication. There was also no scar on chest wall. The patient was discharged 7 days in NYHA classes I.

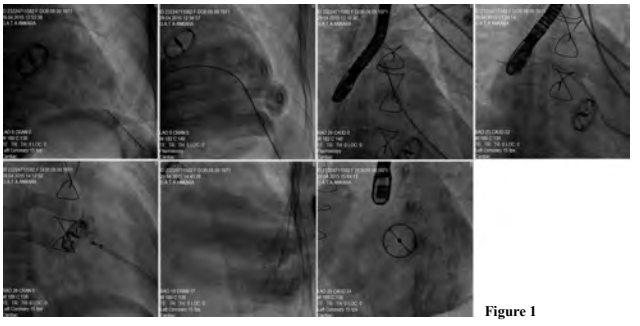


Figure 1

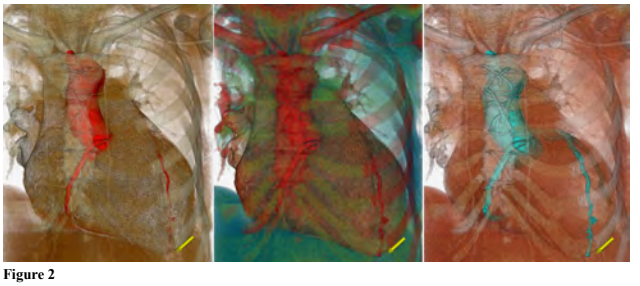


Figure 2

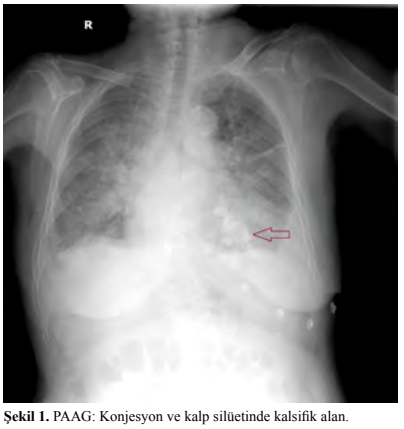
SO-31

Taş yürek; subvalvüler yapıları tutan dev mitral anüler kalsifikasyon

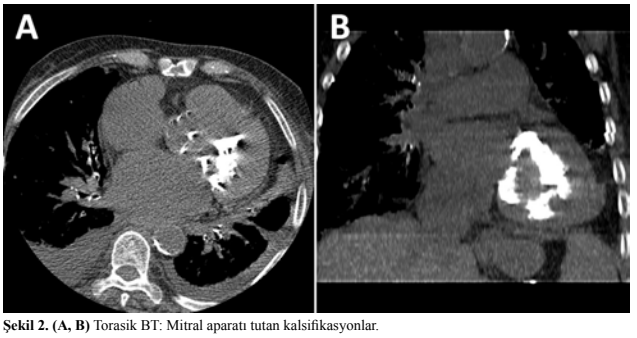
Gökhan Gökalp, Serkan Ünlü, Asife Şahinarslan, Gülten Taçoy

Gazi Üniversitesi Tıp Fakültesi, Ankara

83 yaşında bayan hasta, 2 hafta önce başlayıp, son günlerde şiddetlenen nefes darlığı yakınması ile acil servise başvurdu. Özgeçmişinde miyelodisplastik sendrom ve hipertansiyon öyküsü vardı. Fizik muayenede kan basıncı 120/60 mmHg, nabız düzensiz ve 130 atım/dk, solunum sayısı 23/dk, oksijen saturasyonu ise %87 'di. Akciğer muayenesinde bilateral skapula ortasına kadar ince ralleri ve en belirgin aort odağında 4/6 sistolik üfürümü mevcuttu. EKG sinde hızlı ventrikül yanıtlı atrial fibrilasyon saptandı. Laboratuvar bulguları Hg: 8,3 g/dl ve Na: 125 mmol/L olması dışında normaldi. Akciğer grafisinde konjesyon bulguları ile kalp silüetinde opasite artışı gösteren bir lezyon olduğu görüldü (Resim 1). Akciğer ödemi kliniğiyle hasta koroner yoğun bakım ünitesine yatırıldı. Transtorasik ekokardiyografide (TTE) sol ventrikül ejeksiyon fraksiyonu %57, orta derece aort darlığı, ciddi triküspit yetmezliği ve velositeden ölçülen sistolik pulmoner arter basıncı 65 mmHg olarak tespit edildi. Ayrıca mitral kapakta orta derece darlığa neden olan mitral anülüsü tümüyle tutan ve papiller kaslara kadar yayılım gösteren mitral anüler kalsifikasyon (MAK) izlendi (Video 1). Acil klinik durum düzeltildikten sonra mevcut patolojiyi daha iyi değerlendirmek amacıyla torasik bilgisayarlı tomografi (BT) çekildi. BT de de TTE yi destekler yönde mitral anüler bölgeden başlayıp papiller kasları tutan amorf kalsifikasyonlar saptandı (Resim 2A, B). Kardiyak kateterizasyon planlanan ancak kabul etmeyen hasta medikal tedavisi düzenlenerek taburcu edildi. MAK mitral aparatın fibrozisi ile oluşan dejeneratif ve kronik bir patolojidir. Yaşlı, kadın ve kronik böbrek yetmezliği olan hastalarda daha sık görülür. MAK da tutulum sıklıkla posterior yaprakçıkla, anülüsün posterior kesimindedir. Nadiren subvalvüler yapılar ve bitişik ventriküler duvarlara ilerleyebilir. MAK a bağlı mitral darlığı ve yetmezliği, ileti bozuklukları olabilir. Ciddi kapak disfonksiyonu oluşturmadıkça cerrahi tercih edilmez.



Şekil 1. PAAG: Konjesyon ve kalp silüetinde kalsifik alan.



Şekil 2. (A, B) Torasik BT: Mitral aparatı tutan kalsifikasyonlar.

SO-32

A case of transcatheter aortic valve implantation complication with total femoral artery thrombosis due to failure of ProStar device

İsa Öner Yüksel, Erkan Köklü, Şakir Arslan, Göksel Çağırcı, Selçuk Küçükseymen, Görkem Kuş, Murat Esin

Antalya Education and Research Hospital, Antalya

A 61-year-old man, diagnosed 2 years earlier with degenerative aortic stenosis, presented with a one-year history of increasingly labored breathing and edema in the legs. Transthoracic echocardiogram revealed severe aortic stenosis, mild aortic regurgitation, and a left ventricular ejection fraction of 0.55. The patient's calculated Society of Thoracic Surgeons risk score was 11% and at high risk for TAVI. Because of comorbid conditions, we decided to perform TAVI and not surgery. TEE showed the aortic annular size was 25 mm. Peripheral angiograms showed no tortuosity or calcification of the iliofemoral arteries but revealed severe calculus on the aortic valve. Coronary angiography revealed no significant atherosclerotic lesion and patent by-pass grafts. With use of TTE guidance and rapid temporary pacing, we performed aortic balloon valvuloplasty and transfemoral implantation of a 26-mm Edwards Sapien XT Transcatheter Heart Valve. Afterwards, TTE showed appropriate positioning of the prosthetic valve, no paravalvular aortic regurgitation, and a mean gradient of 9 mmHg. Aortic arch angiograms under fluoroscopic guidance showed patency of the right and left coronary arteries nonselectively and no aortic regurgitation (Fig. 1, video 1). After the successful valve implantation a ProStar XL® Percutaneous Vascular Surgical System was used to close the right common femoral artery percutaneously. However, despite the closure of the femoral artery ProStar device at this stage showed continued bleeding. Because ProStar failure, manual compression was applied about 10 minutes, 5.000 units protamine was administered by intravenous due to ongoing bleeding. Then, control angiography was performed and it was noticed that the right common femoral artery thrombus formation (Fig. 2, video 2). Thrombotic lesion in the right femoral artery was attempted to be passed with 0.035-inch hydrophilic wire making the crossover through the opposite femoral artery, but could not be successful. Therefore, patient was decided to treat by surgery. Thrombus was removed from the femoral artery by cardiovascular surgeons and flow was restored after vascular repair. 5 days later, the patient was discharged without any complications.

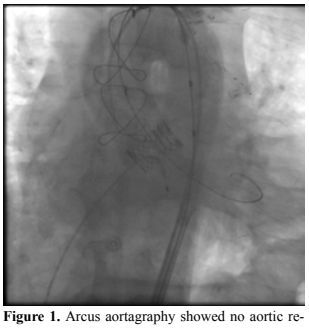


Figure 1. Arcus aortagraphy showed no aortic regurgitation.

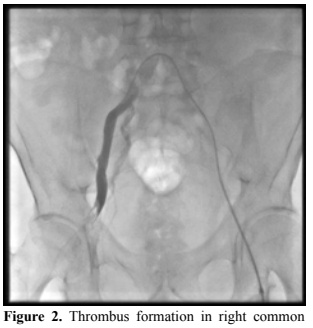


Figure 2. Thrombus formation in right common femoral artery.

SO-33

Aortic embolization of the Edwards Sapien prosthesis due to sigmoid left ventricular hypertrophy

İsa Öner Yüksel, Erkan Köklü, Şakir Arslan, Göksel Çağırcı, Selçuk Küçükseymen, Nermin Bayar, Çağın Mustafa Üreyen, Görkem Kuş, Murat Esin

Antalya Education and Research Hospital, Antalya

A 75-year-old woman diagnosed with symptomatic severe aortic stenosis was referred for TAVI. The patient had a history of diabetes, hypertension, coronary artery disease, morbid obesity, and chronic renal failure. Transthoracic echocardiography (TTE) showed sigmoid left ventricular hypertrophy and ejection fraction of 60%, Doppler echocardiographic scan revealed a mean aortic gradient of 50 mm Hg, an aortic valve area of 0.96 cm2 (Fig. 1). In transesophageal echocardiography (TEE) carried out for detailed examination, sigmoid left ventricular hypertrophy was revealed and aortic annulus measured 24 mm. The aortic annulus diameter was measured 26 x 22 mm in multi-slice computed tomography. The patient was considered to be too high risk for surgery aortic valve replacement and was referred for a TAVI by transfemoral approach. TAVI procedure practise through the right femoral artery using a 26-mm Edwards SAPIEN valve (Edwards Lifesciences, Inc., CA, USA). During balloon inflation under the rapid pacing the valve prosthesis immediately embolized into the ascending aorta (Fig. 2). The subsequent effort to position the valve in the descending aorta was unsuccessful and the bioprosthesis was re-expanded into the aortic arch between truncus brachiocephalicus and left common carotid artery. After this stage the second Edwards SAPIEN valve was successfully implanted with gradually balloon inflated. An arcus aortagraphy was carried out which showed that no aortic regurgitation and no evidence of obstruction of left common carotid artery and truncus brachiocephalicus. The patient was transferred to the intensive care unit in a stable hemodynamic condition and the patients discharged 1 week after the procedure. In our case, distal embolization has occurred due to the sigmoid left ventricular hypertrophy such as the annulus / aortic complex anatomy. In insertion of aortic valve via a percutaneous method, the key factors for proper placement and fixing them; determined to be an appropriate size choice of valve, proper alignment of the valve and placed in the correct position. Finally, the precise positioning of the valve, appropriate valve selection, and the route of administration of the procedure seems crucial for reducing the risk of valve migration.

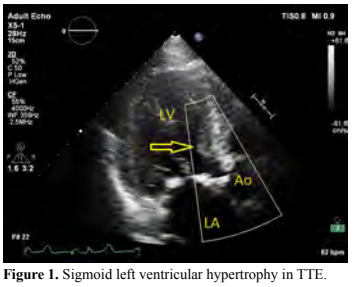


Figure 1. Sigmoid left ventricular hypertrophy in TTE.

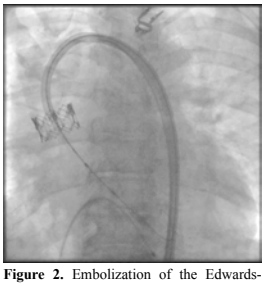


Figure 2. Embolization of the Edwards-Sapien prosthesis.

SO-34

Proximal embolization of the Edwards Sapien prosthesis in TAVI

İsa Öner Yüksel, Erkan Köklü, Şakir Arslan, Göksel Çağırcı, Selçuk Küçükseymen, Nermin Bayar, Çağın Mustafa Üreyen, Zehra Erkal

Antalya Education and Research Hospital, Antalya

A 72-year-old man diagnosed with symptomatic severe aortic stenosis was referred for TAVI. Transthoracic and colour Doppler echocardiographic scan revealed a mean aortic gradient of 42 mmHg, an aortic valve area of 0.90 cm^2, and ejection fraction of 60%. In transesophageal echocardiography (TEE) performed for detailed examination, severe aortic stenosis was revealed and aortic annulus measured 25 mm. The patient was considered to be too high risk for surgery aortic valve replacement and was referred for a TAVI by transfemoral approach. TAVI procedure practise through the right femoral artery using a 26-mm Edwards SAPIEN XT valve (Edwards Lifesciences, Inc., CA, USA). Fallowing predilatation with 23x40 mm balloon inflation under the rapid pacing the Edwards SAPHIEN XT valve successfully implanted with appropriate position. Fallowing the valve implantation an arch aongiography carried out and at that time we saw in the fluoroscopy that the Edwards SAPHIEN XT was sliding under the aortic annulus (Figure 1, video 1). TTE revealed that prosthesis valve was seen in left ventricular outflow tract, mean gradient was 14 mmHg and mild paravalvular aortic regurgitation was observed. Fortunately, the prosthetic aortic valve was stable and there were no any hemodynamic disturbances at this stage. The patient and relatives were informed about this complication and and discussed the necessity of urgent operation. However, patient refused the operation, he said he wants to stay that way, although described the risks that may occur. The patient was transferred to the intensive care unit in a stable hemodynamic condition. In intensive care unit, hemodynamics were stable. It was not observed to create obstruction because of one end of embolized valve in free, other end was holding to LVOT and opened formation in performed TTE and TEE images (Figure 2a, b). One month after discharge, mild paravalvular AR was observed and there was no valve gradient in TTE and TEE control images (Figure 3, video 2). He was began to be followed by coumadin due to prosthetic valve embolization and atrial fibrillation. Finally, the precise positioning of the valve, appropriate valve selection, and the route of administration of the procedure seems crucial for reducing the risk of valve migration.

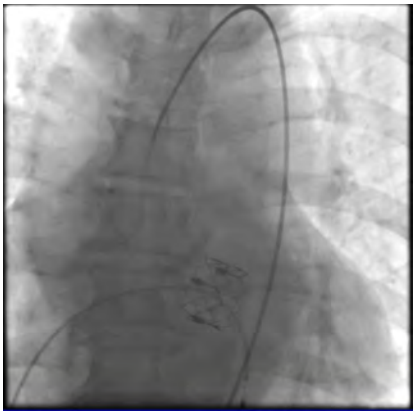


Figure 1. Proximal embolization in flouroscopy.

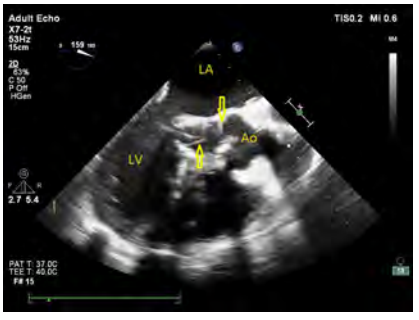


Figure 2. Ventricular embolization in TEE.

SO-35

Successful transfemoral aortic valve replacement after ipsilateral common right iliac artery stenting in the same session

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

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A 65-year-old man with the history of CABG, CRF, HT and left subclavian artery angioplasty was presented with a one-year history of exercise induced chest pain and New York Heart Association functional class IV dyspnea to our clinic. Transthoracic echocardiogram (TTE) revealed severe aortic stenosis (mean gradient: 40 mmHg and aortic valve area: 0.64 cm²) and normal left ventricular ejection fraction (65%). Coronary angiography performed 2 weeks ago in another center suggested medical follow-up. The patient's calculated STS and Logistic EuroSCORE scores were 5.2% and 24.8% respectively. After all, the heart team decided to proceed with TAVI. Multislice computed tomography showed severe stenosis on the right common iliac artery and severe tortuosity and calcification of the left ilio-femoral arteries. Due to our patient had the history of CABG and left subclavian artery angioplasty before, we were not able to use trans-subclavian TAVI. We planned to perform percutaneous transluminal angioplasty and stenting to the right CIA and then continue the TAVI procedure in the same session. Thereafter, the patient was taken to the catheterization laboratory. The peripheral angiography confirmed severe stenosis of the right CIA (Figure 1). At first, after inserting 8 French sheaths to the left and right femoral arteries, a 9x80 mm self-expandable stent was implanted to the right CIA. Then, a post-dilatation with 8x60 mm balloon was performed and stenosis was completely relieved (Figure 2). After that, aortic balloon valvuloplasty and TAVI via right femoral artery was performed successfully with a 23x40 mm balloon and 26 mm Edwards Sapien XT valve (Edwards Lifesciences Corporation; Irvine, Calif) respectively under local anesthesia and rapid pacing (Figure 3) and the patient discharged from the hospital with a good health.

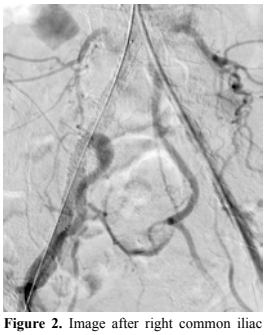

Figure 2. Image after right common iliac artery stenting.

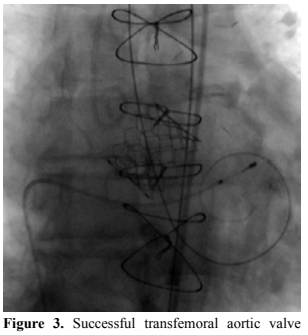

Figure 3. Successful transfemoral aortic valve implantation.

SO-36

Left ventricular side obstructive pannus formation after rheumatic mitral valve replacement with preservation of the subvalvular apparatus

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Background: In non-rheumatic mitral valve replacement (MVR), preservation of the subvalvular apparatus (SVA) is recommended to maintain annular–papillary continuity, which is known to be associated with improved left ventricular (LV) function in the early and late postoperative period. However its applicability in rheumatic valves remain controversial. The presence of intense fibrosis and calcification in rheumatic valves may promote impingement of the pannus tissue on the prosthesis and cause obstruction of the mitral inflow tract.

Methods: A 29 year old woman was admitted to our outpatient clinic with progressive dyspnea. She had undergone mitral valve replacement (25 mm St Jude Medical) due to rheumatic mitral stenosis 12 years earlier. Transthoracic (TTE), two dimensional (2D) and real-time three dimensional (RT3D) transesophageal echocardiography (TEE) were performed for the evaluation of the mitral prosthesis.

Results: TTE revealed increased mean transprosthetic gradient (15 mmHg) and decreased valve area (0.9 cm²). In parasternal long axis view, preservation of both anterior and posterior SVA was observed (Fig 1a). 2D TEE showed a hyperechogenic circular mass on the LV side of the prosthesis causing one leaflet stuck in closed position (Fig 1b). RT3D TEE confirmed the diagnosis of LV side obstructive pannus formation (Fig 1c). Since the patient was symptomatic she underwent re-do valve surgery. Postoperative pannus specimen was observed to be derived from chordal connections to the prosthetic annulus (Fig. 1d).

Conclusion: Preservation of SVA during MVR in rheumatic valve disease may provoke pannus tissue formation on the LV side of the mitral prostheses. In order to prove this association, large scaled randomized studies are needed.

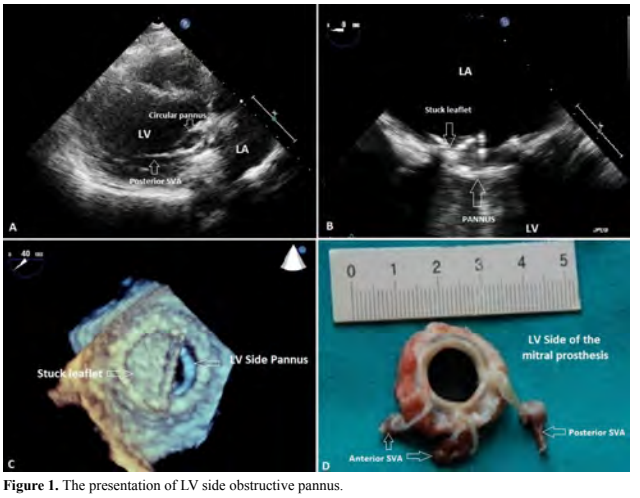

Figure 1. The presentation of LV side obstructive pannus.

SO-37

Unusual thrombolytic therapy decision in prosthetic valve thrombosis during early pregnancy

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A 29-year-old pregnant patient had a history of a mitral valve replacement (MVR) surgery 4 years prior to admission, because of rheumatic valve disease. She was on her first pregnancy and was using warfarin, INR was found to be 1.42. On transthoracic echocardiography (TTE), where restricted mobility of the anterior leaflet and an increased pressure gradient on the same valve was detected. In addition, a transesophageal echocardiography (TEE) showed that the anterior leaflet of the mechanical mitral valve did not open properly during diastole. Mean gradient was 28 mmHg and mitral valve area was found to be 0.8 cm². No clear image of a thrombus could be detected (Figure 1A, B).Because the patient was pregnant, a fluoroscopy could not be performed. The patient's last TEE had been 2 months prior to admission and had detected a functional mechanic prosthetic mitral valve with a mean mitral gradient of 6 mmHg. Judging from her symptoms, the presence of a state susceptible to thrombosis (pregnancy) and her subtherapeutic INR values.With the exception of pregnancy, the patient had no other contraindications for thrombolytic therapy and was started on a 6-hour-long 25 mg of tPA infusion. She underwent a TTE, that found no change in her transmitral gradient and given another 25 mg tPA infusion, lasting 6 hours. Another TTE and again no change in her transmitral gradient was detected. The patient was given a third 6-hour-long 25 mg tPA infusion.A TTE and a TEE were performed and the movement of the anterior mitral leaflet had returned back to normal. Mean transmitral gradient was 7 mmHg and mitral valve area was found to be 2.7 cm² (Figure 2A, B). A diagnosis of valve thrombosis should come to mind for those patients who show an evident gradient increase on their metallic valves during an echocardiographic follow-up. Pregnant patients with prosthetic valve thrombosis can be treated with prolonged infusions of low-dose tPA. Low-dose, long tPA may be given repeated doses.

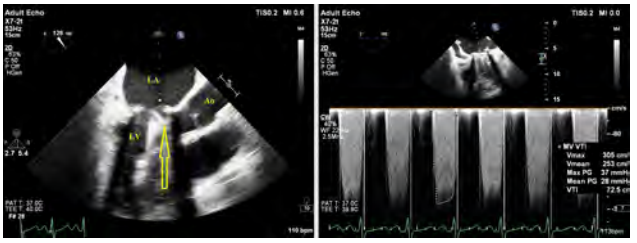

Figure 1. (A, B) Increased in transmitral gradient.

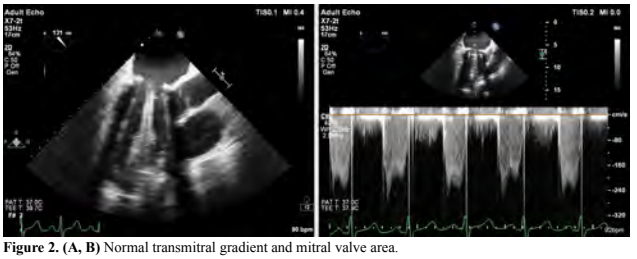

Figure 2. (A, B) Normal transmitral gradient and mitral valve area.

SO-38

Barlow sendromlu semptomatik ciddi mitral yetersizliği olan bir hastada mitraclip uygulanımı ve 12 aylık takip sonucu

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Giriş: Dejeneratif mitral kapak yetersizliği (DMY) olan hastalarda kabul edilen tedavi biçimi mitral kapak tamiri ya da mitral kapak replasmanıdır. Bununla birlikte, bu hastaların bir kısmı ileri yaş ve eşlik eden komorbiteler nedeniyle cerrahi için yüksek riskli olarak değerlendirilmekte ve gereken tedavi yapılamamaktadır.Cerrahi için yüksek riskli olan hastalarda perkütan mitral kapak tamiri (MitraClip) alternatif bir tedavi biçimi olarak son yıllarda ön plana çıkmaktadır. Bu bildirimizde MitraClip uygulanan Barlow sendrom'lu bir olguda başarılı MitraClip uygulanımı ve bir yıllık takiplerini sunmaya çalıştık.

Olgu Sunumu: Dış merkezde nefes darlığı ve kalp yetmezliği ile takip edilen 66 yaşındaki erkek hastanın yapılan tetkikleri sonucunda ileri derecede MY saptanması üzerine kliniğimize yönlendirildi. Hastanın anamnezinden 2 aydır nefes darlığı olduğu ve son 20 gündür şikayetlerinde artış olduğu öğrenildi. Ortopne ve paroksismal nokturnal dispne tarifleyen hastanın NHYA göre fonksiyonel kapasitesi sınıf 3 olarak değerlendirildi. Ek herhangi bir hastalığı olmayan hastanın kliniğimizde yapılan ekokardiyografisinde ejeksiyon fraksiyonu %65 (diyastol sonu çap: 4.5 cm, sistol sonu çap: 2.9 cm, sol atriyum: 3.8 cm) ölçüldü (Video 1, 2). Yapılan transözofajiyal ekokardiyografisinde mitral kapakta çok segmentde belirgin prolapsus, A2-P2 skalloplarında daha belirgin olmak üzere ileri derecede MY izlendi (Video 3, 4). Hastanın semptomatik MY olması nedeniyle, mitral kapağa girişim planlandı. Yapılan koroner anjiyografisinde ciddi darlık saptanmadı. Hasta Kalp Damar Cerrahisi bölümü ile birlikte değerlendirildi. Hastanın ek hastalığı olmamasına rağmen ileri derecede kaşeksisi olması nedeniyle (VKİ: 15 kg/m^2) açık kalp cerrahisinin riskli olduğu belirtilerek hastaya MitraClip uygulanmasına karar verildi. Hastaya Aralık 2013 tarihinde MitraClip uygunlandı. Mitral kapağın santral ve medial skalloplarına olmak üzere 2 klip yerleştirilerek eser MY ile işlemden çıkıldı (Video 5, 6, 7). Ortalama 5 mmHg gradient elde edildi. Hastanın takiplerinde fonksiyonel kapasitesinin belirgin biçimde düzeldiği ve KY semptomlarının kaybolduğu görüldü. 1 yıl sonra yapılan ekokardiyografisinde ortalama transmitral gradient 5 mmHg saptandı ve birinci derece MY izlendi (Video 8, 9, 10).

Sonuç: DMY özellikle batı dünyasında MY'nin en sık görülen sebebidir. Ülkemizde de yaşlı nüfusun artması ile birlikte DMY sıklığında artışın görülmesi kaçınılmazdır. DMY'liği olan hastalarda medikal tedavinin etkinliği kısıtlıdır ve mortalite üzerinde etkisi gösterilememiştir. DMY'li olan ve MitraClip uygulanan hastalarda yapılan çalışmalarda hastaların yaşam kalitesinde belirgin düzelme, hastanede yatış oranlarında ciddi düzeyde azalma saptanmıştır. DMY'liği olan olgularda cerrahi riskin yüksek olduğu öngörülüyorsa MitraClip bu hastalar için önemli bir alternatif tedavidir.Bu olgu, bildiğimiz kadarıyla ülkemizde çok segment prolabe Barlow Sendromlu bir hastada ilk kez MitraClip uygulanımıdır.

SO-39

Obstructive prostethic mitral valve thrombosis successfully thrombolysed with low-dose ultra-slow infusion of tissue plasminogen activator

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Background: Treatment modalities for prosthetic valve thrombosis (PVT) include anticoagulation with heparin, thrombolytic therapy (TT) and re-do valve surgery. TT has been recently used with successful outcomes.

Methods: A 57 year-old woman who had undergone mechanical mitral valve replacement was admitted to our institution with dyspnea. Transthoracic echocardiography (TTE) and subsequently transesophageal echocardiography (TEE) were performed for evaluation of prosthetic mitral valve. The TT protocol consisted of repeated sessions of low-dose (25 mg), ultra-slow infusion (25 hours) of tPA without bolus and six hours of heparin infusion between the sessions.

Results: TTE revealed increased mitral transvalvular gradients of 29/15 mmHg with a decreased mitral valve area of 0,9 cm^2. Subsequently 2D TEE was performed which demonstrated a giant mobile thrombus located on the mitralprosthetic valve (A) and RT-3D TEE clearly demonstrated the huge thrombus obstructing the mitral inflow (A'). Fluoroscopy revealed one leaflet fixed in closed position and the other with a restricted slight motion (A''). TT was therefore immediately started. After the first TT session repeated 2D and RT-3D TEE showed a decreased thrombus burden on the prosthesis (B and B'). There was a mild relaxation in motion of restricted leaflet but the other leaflet was still stuck on fluoroscopy (B''). Upon these findings a second session of TT was performed. The thrombus burden decreased significantly on 2D and RT-3D TEE (C and C') and there was a slight movement in the stuck leaflet on fluoroscpy (C''). Subsequently a third session of TT resulted in complete thrombolysis of the thrombus on 2D and RT-3D TEE (D and D') and nomalization of leaflet motions on fluroscopy (D'').

Conclusion: Low-dose and ultra-slow infusion of tPA is a valuable alternative treatment regimen for prosthetic heart valve thrombosis. TEE and fluoroscopy are complementary to each other for TT guidance in PVT patients.

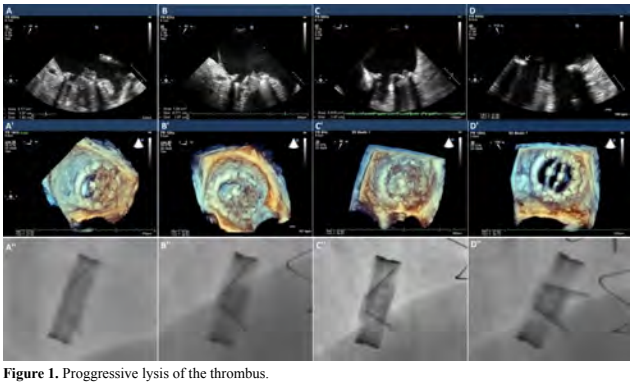

Figure 1. Proggressive lysis of the thrombus.

SO-40

The absence of the posterior mitral leaflet; like a Viking ship

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A 62-year-old woman suffering from dyspnea and palpitation was referred to our hospital. Electrocardiography (ECG) revealed left bundle branch block and atrial fibrillation. Her blood pressure was 110/60 mmHg and heart rate 130 bpm in emergency department. Cardiac examination revealed a 2/6 pansystolic regurgitant murmur at the apex radiating to the left axillary area and crepitan crackles at both basal segments of lungs. She was hospitalized for decompensated heart failure due to atrial fibrillation with rapid ventricular response. Transthoracic echocardiography revealed the absence of the posterior mitral leaflet (Figure 1A-D). Posterior myocardial wall proceed to coaptation line of the mitral valve. Chordae tendineae and papillary muscles were attached to tip of posterior myocardial wall. Subsequent transesophageal echocardiography confirmed transthoracic echocardiography findings (Figure 1E). Color coded Doppler examination revealed a mild mitral regurgitation in transesophageal echocardiography examination (Figure 1F). Congenital malformations of the posterior mitral leaflet are extremely rare and present with a wide spectrum of morphologic abnormalities. Hypoplasia of the posterior mitral valve leaflet was reported previously. Absent posterior leaflet was determined. But this anomaly that myocardial segment instead of posterior mitral leaflet was not previously reported.

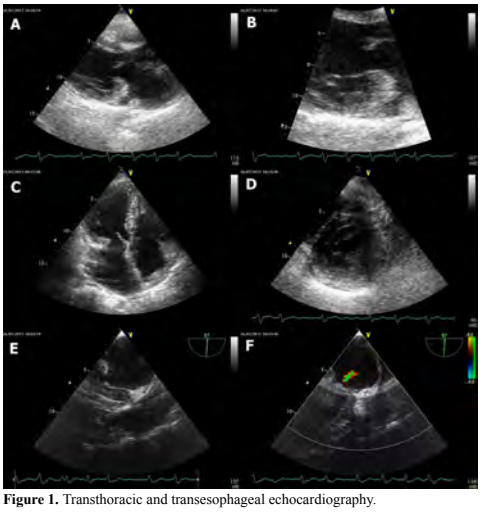

Figure 1. Transthoracic and transesophageal echocardiography.

SO-41

LAD artere diagonal kollateral yoluyla retrograt kronik total oklüzyon girişim

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Koroner arter hastalığı olan 73 yaşında erkek hasta, 09.02.2015 tarihinde kararsız angina pektoris ile kliniğe yatırıldı. Hastaya rutin tetkikleri müteakip koroner anjiyografi yapıldı. LAD Proksimal %100, CX Proksimal %100, RCA %100. AO-LAD Safen anastomaz sonrası %99 kronik total oklüzyon izlendi. RCA soldan redrograd doluyor. AO-CX Safene daha önce takılan stentte izlenen restenoza tekrar stent takıldı. AO-LAD safen iki ay sonra Kronik Total Oklüzyon (CTO) girişimi planlandı. 17.04.2015 tarihinde hasta AO LAD safen grefti için Kronik Total Oklüzyon işlemine alındı. İşlem antegrat ve diyagonalden retrograd olarak girişim uygulandı. Antegrat finecross 130cm eşliğinde sırası ile fielder FC, miracle 3 ve miracle 4,5 teller kullanılarak lezyon geçilmeye çalışıldı. Tellerin subintimal ilerlemesi üzerine LAD distalinin diagonalden dolduğu gözlendi, işleme diagonalden retrogat olarak finecross 150 cm eşliğinde sırası ile fielder FC, miracle 3 ve gaia lellerle LAD distalinden LAD proksimaline ulaşıldı. RG telin her iki ucundanda mikro kateter gönderildi. Redrograt mikrokateter ve RG geri çekilerek antegrat mikrokateterden flopy tel yollanarak işleme antegrat devam edildi. Damar açıldı, komplikasyon olmadı.

SO-42

Successful closure of mitral paravalvular leak with vascular plug – III

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59-year-old male patient was admitted with shortness of breath. In his medical history, he has been performed mitral commissurotomy due to rheumatic heart valve disease 21 years ago, mitral valve replacement operation due to the advanced development of mitral regurgitation 13 years ago, and 2 years ago paravalvular mitral regurgitation has developed. RE-DO MVR was performed. In last six months, in patient with exertional dyspnea, severe regurgitation jet was detected at localization of the posterior mitral annulus (mitral en face image on clockwise 6) on echocardiography. Paravalvular leak closure was planned. 8 F sheath was inserted via the left femoral vein way. After transseptal puncture, flexcath was placed interatrial septum. After addressed to the mitral valve posterior, 0.035 hydrophilic guidewire was passed via the leakage position. Guidewire was advanced via transaortic. In order to provide support, the snare which promoted from the left femoral artery was captured by catheter. Therefore, the closure device called as Vascular Plug-III (5x10 cm), was placed in the site of the regurgitant jet. After watching the amount of leakage is mild, the device was released.

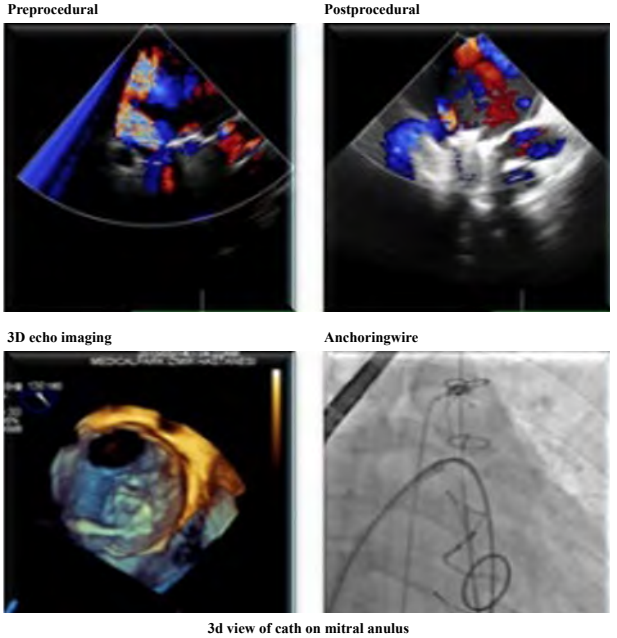

Figure 1. Pre and postprocedural images.

SO-43

Aort stenozu ve İliak arteranevrizması bulunan olguda eş zamanlı yapılan TAVİ ve EVAR olgusu

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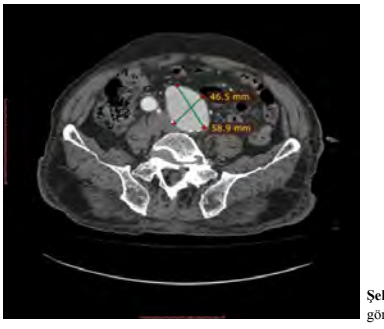
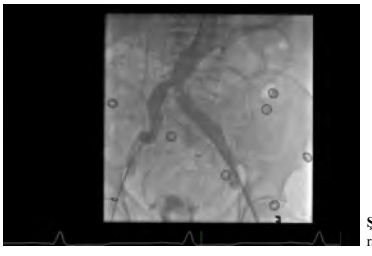
81 yaşında erkek hasta NYHA 3-4 semptom ve senkop ile hastanemize başvurdu. Hastanın tıbbı öyküsünde diyabetes mellitus, hipertansiyon ve ileri derecede kronik obstriktif akciğer hastalığı (KOAH) mevcuttu. Hastanın fizik muayenesinde sağ 2. interkostal aralıkta sistolik üfürüm ve her iki akciğer alanında yaygın wheezing haricinde patolojik bulgu saptanmadı. Transtorasik ekokardiografi ile yapılan inceleme de sol ventrikül sistolik fonksiyonları normal olarak gözlendi. Aort kapağın kalın kalsifik yapıda ve açılımının azalmış olduğu izlendi. Doppler incelemesi aort kapak üzerinde maksimum 85 mmHg, ortalama 45 mmHg gradyanın olduğunu ve aort kapak alanının ise 0.9 cm^2 olduğunu gösterdi. Hastanın yapılacak bir aort kapak cerrahi işlemi açısından Euroscore 24 olarak hesaplanması üzerine 'Heart Team' tarafından hastaya TAVI yapılması kararlaştırıldı. Hastaya işlem öncesi hazırlık amacı ile yapılan bilgisayarlı tomografide (BT) hastanın sol ana iliak damarında 46x58 mm boyutlarında anevrizma saptandı (Şekil 1). Hastaya eş zamanlı TAVI ve EVAR işlemi yapılmasına karar verildi. Hasta kateter labaratuarna alındı. Her iki femoral arter cut down ile açıldı. Yüksek frençli kılıfın sağ femoral artere yerleştirilmesini takiben aort kapak 20 mm balonla predilate edildi. Takiben 23 mm Edwards SAPIEN kapak floroskapi altında uygun pozisyonda hastaya implante edildi. İşlemin komplikasyonsuz seyretmesi üzerine EVAR işleminin yapılmasına geçildi. Endurant 26 mm greft stent ana gövde infra renal düzeyde açıldı. 120x16 mm karşı taraf bacak anevrizmatik iliak arter tarafına floroskopi altında yerleştirildi. Yapılan kontrol enjeksiyon sonrası anevrizmanın kapandığı ve vasküler bir komplikasyon olmadığının görülmesi üzerine işleme son verildi (Şekil 2). Hastane içi takiplerinde herhangi bir komplikasyon gelişmeyen hasta yatışının 5. gününde taburcu edildi.



Şekil 1. BT'de iliak arter anevrizmasının görünümü.



Şekil 2. Evar sonrası anevrizmanın angiografik görünümü.

SO-44

Dejenere aortik biyoprotezli hastaya TAVİ yöntemiyle valve-in-valve

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Amaç: Cerrahi olarak implante edilmiş aortik biyoprotezler zaman içerisinde dejenere olabilmektedir. İkinci kez açık kalp cerrahisi yapılması hastalar açısından yüksek morbidite ve mortalite riski taşımaktadır. Transkateter yöntemlerle dejenere biyoprotez kapakların içerisine yeni biyoprotez kapaklar implante edilebilir.

Olgu Sunumu: Nefes darlığı, genel durum bozukluğu ve çarpıntı şikayetleri ile polikliniğimize başvuran hasta hemodinamisinin iyi olmaması nedeniyle yoğun bakım takibine alındı. Akciğer ödemi ve pretibiyal ödemi olan olguya diüretik tedavi başlandı. Yoğun medikal tedavi ile genel durumu düzelen hasta kalp takımı konseyince değerlendirildi. Hastanın yüksek cerrahi risk nedeniyle TAVİ yöntemiyle valve-in-valve işlemi yapılmasına karar verildi. Hastanın STS skoru 14.8 ve logistic euroscore değeri 32.8 idi. Aortik gradiyentler 64/41 mmHg ve pulmoner arter basıncı 55 mmHg olarak saptandı. İleri derecede aort yetersizliği mevcuttu. Orta mitral yetersizliği ve orta triküspit yetersizliği belirlendi. Hastanın koroner anjiyografisinde kritik olmayan lezyonlar saptandı. Transözofageal ekokardiyografi (TOE) değerlendirmesinde aortik anulus 19.5 mm olarak belirlendi. Daha önceden implante edilen cerrahi kapağın 21 mm çapında olduğu öğrenildi. Tomografide aortik anulus 19.1 mm olarak saptandı. Hastanın tomografisinde 11.1 mm, LMCA ve aort kapak mesafesi olduğu görüldü. RCA ostiumunun aort kapağına mesafesi 14.8 mm olarak hesaplandı. Hastanın iliak ve femoral damarlarında hafif derecede tortuozite olduğu gözlendi. Hasta steril şartlarda anjiyografi laboratuarında işlem için hazırlandı. Sağ common femoral artere cut-down işlemi uygulandı. Geçici kalp pili sol femoral venden, pigtail ise sol femoral arterden hastaya yerleştirildi. Hastanın sağ femoral arterinden e-sheath aortaya gönderildi. AL1 kateter yardımıyla düz uçlu tel ile aort kapağı geçildi. AL1 kateter sol ventriküle (LV) ilerletildi. İçerisinden extra sertlikteki tel LV içerisine yerleştirildi. Aortaplasti ve 23 mm aortik biyoprotez implantasyonu başarılı bir şekilde yapıldı. Herhangi bir komplikasyon bulgusu saptanmadı.

Sonuç: Dejenere olmuş disfonksiyone aortik biyoprotez kapaklara başarılı bir şekilde valve-in-valve yöntemiyle yeniden aortik biyoprotez replasmanı yapılabilir.

SO-45

52 yaşında ciddi romatizmal aort ve mitral darlığı olan bir hastada aynı seansda transkateter aort kapak replasmanı ve mitral balon valvulotomi

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Giriş: Ciddi aort darlığı (AD) ve mitral darlığı (MD)'nın birlikte olduğu durumda standart tedavi yöntemi cerrahi kapak replasmanıdır. Konvansiyonel kalp cerrahisine uygun olmayan veya cerrahi için yüksek riski olan ciddi AD ve MD olan hastalar, girişimsel kardiyoloji için teknik olarak bir zorluğa yol açabilirler. Böyle hastalarda cerrahi kapak replasmanına alternatif olarak eş zamanlı perkütan ikili balon valvuloplasti başarılı bir şekilde uygulanmıştır. Ancak mitral balon valvulotominin aksine aortik balon uygulanımı palyatifdir, hastalığın gidişini değiştirmez. Son zamanlarda sık uygulanan transkateter aort kapak implantasyonu (TAVİ) konvansiyonel kalp cerrahisine uygun olmayan veya cerrahi için yüksek riski olan ciddi AD olan hastalarda yeni, ümit veren bir tedavi biçimidir. Konvansiyonel kalp cerrahisine uygun olmayan veya cerrahi için yüksek riski olan ciddi AD ve MD olan hastalar için eşzamanlı TAVİ ve mitral balon valvulotomi teknik olarak mümkün olmasına rağmen, bu tekniğin kullanımına dair literatürde herhangi bir bilgi yoktur. Olgumuzda, total perkütan yaklaşım uretral kanser nedeni ile seçildi.

Olgu Sunumu: 2 yıldır nefes darlığı yakınması olan 52 yaşındaki bayan hasta ürolojik cerrahi öncesi bize yönlendirildi. NYHA sınıf II olan hastanın romatizmal ciddi AD (ortalama aortik gradiyent:40 mm Hg) ile beraber ciddi MD (ortalama mitral gradiyent: 17 mmHg) vardı. MD'nın morfolojik eko skoru 7 idi (Şekil 1, 2, 3) (Video 1, 2). Yapılan koroner anjiyografide patoloji saptanmayan hastaya çift kapak replasmanı düşünüldü, ancak uretral kanser nedeniyle cerrahiye uygun görülmeyen hastaya genel anestezi altında, transözofajiyal ekokardiyografi eşliğinde aynı seansda önce mitral balon valvuloplasti ve vakit kaybetmeden CoreValve ile TAVİ planlandı. Mitral balon sonrası (Inoue balloon catheter, 26 mm) mitral ortalama gradiyent 5 mm Hg'a düştü (Şekil 4) (Video 3). Hemen ardından 26 mm Corevalve anulusun 4 mm kadar altında olacak şekilde implante edilmeye çalışıldı ancak, pop-out (kapak anulusden asendan aortaya doğru yer değiştirdi) gelişti. Kapak geriye alınıp tekrar yüklenerek öncekine göre biraz daha derin yerleştirildi. İşlem sonrası paravalvüler aort kaçağı gözlenmedi (Şekil 5) (Video 4, 5, 6). İşlem süresi toplam 105 dakika olarak gerçekleşti.

Sonuç: Normal koşullarda TAVİ işlemini mitral balondan önce yapmak daha mantıklıdır. Ancak TAVİ sonrası yapılacak mitral balon aortik protez kapağa zarar verilebilir (embolizasyon vs.). Bu yüzden önce mitral balon ardından TAVİ uygulandı. Ancak ciddi AD varlığında mitral balon sonrası volüm yükü nedeniyle ciddi sorunlar meydana gelebilir. Mitral balon sırasında orta derecede kan basıncı düşüşünün dışında herhangi bir sorunla karşılaşmadık. Bu olgu, başka tedavi seçeneği olmayan romatizmal ciddi AD ve MD olan hastalarda eş zamanlı mitral balon valvulotomi ve TAVİ'nin güvenli ve uygulanabilir bir tedavi olabileceğini düşündürebilir.

SO-47

A rare coronary anomaly: single coronary artery with the right coronary artery arising from septal branches of left anterior descending artery

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A 69-year-old man who is a kidney donor candidate was referred to cardiology clinic to asses before a possible kidney transplant. In anamnesis we noted the patient had a chronic hypertension for ten years and smoking 1 packet cigarette a day for 40 years. On physical examination, there was no obvious pathology. Exercise stress test could not exclude coronary artery disease which was performed cause the patient reported atypical chest pain. And then the patient underwent coronary angiography. During left coronary sinus injection, firstly we have seen a normal left main coronary artery originating from the left coronary cusp and branching into a left anterior descending artery and circumflex artery. Then we realised that right coronary artery was vizualized as anomalously originating from the septal branches of left anterior descending artery and there was no severe stenosis in coronary tree. Aortic root angiography depicted no coronary ostium orginating from the right sinus of valsalva. So, we incidentally determined a very rare anomaly which can be identified briefly single coronary artery anomaly. In order to delineate the exact anatomical course of the vessel originating from the septal branches of LAD, coronary computed tomography was performed on a 64-slice scanner. CCT, confirmed the absence of a right coronary ostium and showed the vessel is running anterior of the pulmonary trunk with moderate tortuosity. After make certain that anomalous coronary artery has no course between pulmonary trunk and aorta means that; no increased risk for ischemia or sudden cardiac death was not anticipated, the patient was discharged with medical treatment. Single coronary artery anomaly is a very rare anomaly with an incidence of 0.05 in the general population and accounts for 3.3% between coronary artery anomalies. Although it is extremely rare, this case is typical because of discovered incidentally and patient was asymptomatic.

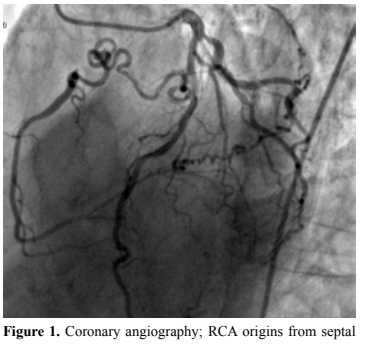

Figure 1. Coronary angiography; RCA origins from septal branches of LAD.

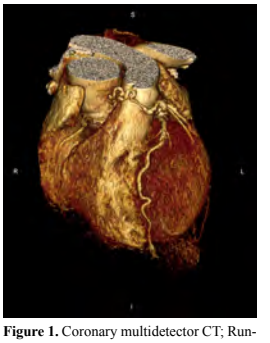

Figure 1. Coronary multidetector CT; Running anterior of the pulmonary artery.

SO-46

ALCAPA syndrome and atrial septal defect in a 68 year-old woman: an extremely rare congenital association

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A 68 year-old female patient admitted to us with shortness of breath and exertional angina which had begun fifteen days ago. Her medical history included only hypertension and denied smoking. Physical examination revealed an apical systolic murmur and second sound was fixed splitting and increased intensity. Electrocardiogram showed normal sinus rhythm with non-specific ST and T wave changes. Echocardiography revealed that ejection fraction of 60%, systolic pulmonary artery pressure of 80 mmHg, moderate tricuspid insufficiency, dilated right sided heart chamber and an atrial septal defect. Transesophageal echocardiography confirmed the diagnosis of ostium secundum type atrial septal defect. Right heart catheterization showed that mean pulmonary artery pressure was 52 mmHg. Coronary angiography (CA) revealed rudimentary left coronary system extremely tortuous, dilated right coronary artery. Furthermore, distal part of the RCA extends collateral arteries to the left anterior descending artery and some contrast was entering to the pulmonary arteries (Figure 1). Multislice computed tomography with volume rendering three dimensional reconstruction was performed that showed left main coronary artery was originated from left pulmonary artery called anomalous origin of the left coronary artery from pulmonary artery (ALCAPA) (Figure 2). The patient was treated with surgery.

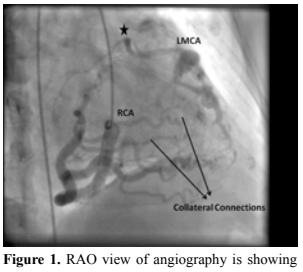

Figure 1. RAO view of angiography is showing collaterals.

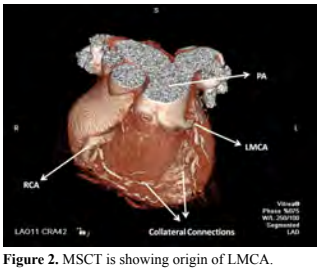

Figure 2. MSCT is showing origin of LMCA.

SO-48

Ostial coronary artery disease due to radioterapy

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A 43 year old woman who had no known cardiac risk factors, presented with exertional angina for two days. She had history of mediastinal Hodgkin's disease, for which she was successfully treated with chemotherapy and mediastinal radiotherapy two years previously. She suffered cardiac arrest at exercise stress test and underwent prompt coronary angiography which showed 90% ostial LAD stenosis and 20% LMCA stenosis. We discharged the patient after successful LAD drug eluting stent implantation, and resolution of her pain. At one year follow-up repeat coronary angiography showed patent LAD stent, slightly worsened LMCA lesion of 40% and a new 80% stenosis at RCA ostium which was normal 1 year ago. This was again treated successfully with another drug eluting stent. At 6 months follow-up control coronary angiography showed patent LAD and RCA stents and again moderate, 40% LMCA lesion. This time there was a new severe (80%) ostial Lcx stenosis. There had been no stenosis at Lcx ostium at previous two angiograms. A new drug eluting stent was placed in Lcx ostium extending to the LMCA ostium. The procedure was finished successfully following final kissing balloons in LMCA to LAD and Lcx ostia. The implantation was guided with coronary FFR measurements and Optical Coherence Tomography (OCT) imaging. The OCT imaging showed us typical radiation coronary disease. The lesion was fibrotic and contained no atherosclerotic plaque. In general, radiation induced stenosis is expected to be seen at least 5 to 10 years following radiotherapy, however our case was admitted with cardiac arrest and repetitive coronary stenosis in 2 years. Here, we would like to report a rare occurence of coronary disease in early post radiation period, in a patient with no known coronary risk factors. And also we would like to report succesful treatment of this disease with percutaneous stent implantations.

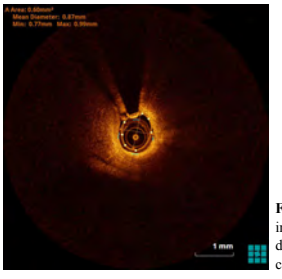

Figure 1. OCT imaging of lesion; diffuse fibrosis in coronary artery.

SO-49

Left ventricular hematoma mimicking lateral wall myocardial infarction secondary to percutaneous coronary intervention

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Introduction: Intra-myocardial hematoma is a rare disease and usually associated multiple different pathology such as myocardial infarction, chest trauma, coronary artery bypass operation, and complication of percutaneous coronary intervention (PCI).

Case: A 63-year-old woman was admitted to our clinic with complaints of chest pain. Coronary angiography was done and revealed severe stenosis (99%) in the circumflex artery (Cx). PCI was chosen as the treatment option for Cx lesion. We crossed the lesion by a 0,014 inch hydrophilic guide-wire. After pre-dilatation with coronary balloon, a 2,75x23 mm DES was implanted under nominal pressure. Final angiogram revealed acceptable results at Cx without any abnormal findings. After procedure, she suddenly complained of chest pain. The ECG showed ST elevations in D1 and aVL derivations. There was no thrombus in the stent in the second angiography. The two-dimensional echocardiography revealed a large 5.5x1.8 cm sized echolucent area with no fluid within the pericardium (figure 1, video 1). We considered this echolucent area as dissecting intramyocardial hematoma (DIH) in the lateral wall of the myocardium. On day 3 of the follow-up, the cardiac computed tomography revealed thickening of the lateral wall of the myocardium, and a radiolucent center without contrast dye (figure 2). She was discharged at 6th day of the follow-up.

Discussion: DIH can occur as a complication of myocardial infarction, PCI and cardiac surgery. DIH diagnosis can be achieved with echocardiography, magnetic resonance imaging, computed tomography, surgery, or autopsy. The conservative management of DIH is associated with a mortality as high as 90-100%. Evacuation of the DIH and surgical repair of the myocardium are the main strategies for the unstable patient. Management of DIH should be individualized integrating the patient's hemodynamic stability, the size, location, and extension of the DIH, and the development of DIH-related complications.

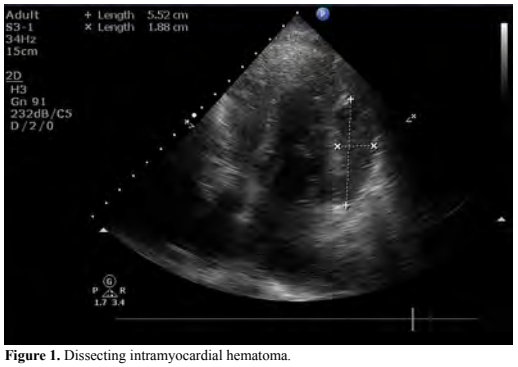

Figure 1. Dissecting intramyocardial hematoma.

Figure 2. CT view of dissecting intramyocardial hematoma.

SO-50

Koroner anevrizmaya coil uygulaması ve takip sonuçları

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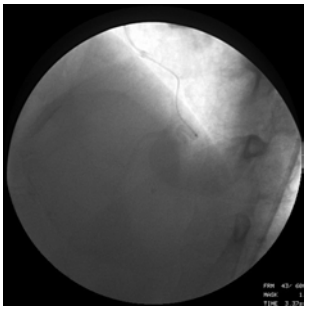
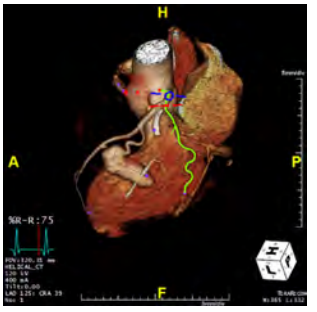
38 yaşında erkek hasta 2 aydır eforla meydana gelen tipik angina yakınması ile polikliniğimize başvurdu. Hastanın özgeçmişinde 3 yıl önce akut inferior ST elevasyonlu myokard infarktüsü ve RCA,CX-OM1 primer PTCA uygulaması mevcuttu. Hastaya koroner anjiografi (KAG) planlayarak kliniğimize interne ettik. Hastanın yapılan kardiyak ve solunum muayenesinde patolojik bulgu saptanmadı. EKG'si sinüs ritminde kalp hızı 99/dk inferİor derivasyonlarında patolojik Q saptandı. Hastaya yapılan KAG'de LMCA normal, LAD-D1 sonrası plaklı, CX-OM1 instent %30, RCA proksimal %100 instent, hastada çıkış yeri net belli olmayan koroner anevrizma saptandı. Hastanın koroner arter anevrizmasının orijini için torax BT planlandı. Hastanın koroner artere yönelik BT anjiografi incelemesinde LAD'nin sol diagonal 1 dalında lobüle konturlar ile uzanım gösteren ve büyük bir kese halinde 3x2x1,5 cm'lik bir anevrizma lobüle konturlar ile seyir gösterdiği, diagonel 2. dal çıkımında küçük bir anevrizmatik genişleme,diagonel 2 çıkışından önce LAD'nin başlangıç bölümünden yaklaşık 3,5 cm distalinde sol laterale doğru geniş ağızlı küçük bir anevrizma görülmekte idi. 3 yıl önce sağ yüzeyel femoralarter distalindeki pseudoanevrizma distal segmentine greft stent öyküsü mevcut olan hastaya koroner ile eş zamanlı periferik anjiografide çekildi. Hastanın perifer anjiografisinde sol popliteal arterde anevrizma saptandı. 3 hafta sonra hastaya yapılan popliteal arter anevrizması cerrahisinden alınan materyal patolojiye gönderildi. Hasta vaskülit ön tanısı ile ilgili kliniklere konsülte edildi. Hastada poliarteritis nodosa ve diğer orta arter hastalıkları açısından araştırılması planlandı. Hastaya 2 ay sonra koroner arter anevrizması için coil uygulanması yapıldı. 4 adet pushablecoil 1. diagonal içinde anevrizmanın proksimalinde serbestleştirildi. Ancak anevrizmanın tam olarak oklude olmadığı izlendi. Daha sonra kontrol KAG'si yapılmak üzere işlem sonlandırıldı. 6 hafta sonra yapılan kontrol KAG'de anevrizmanın tamamen oklude olduğu gözlendi.



Şekil 1. Koroner anevrizma.



Şekil 2. BT'de LAD-Diagonal 1 dalında anevrizma.

SO-51

Anterior ve posterior tibial arter ponksiyonu ile retrograd perifer kronik total oklüzyon revaskülarizasyonu

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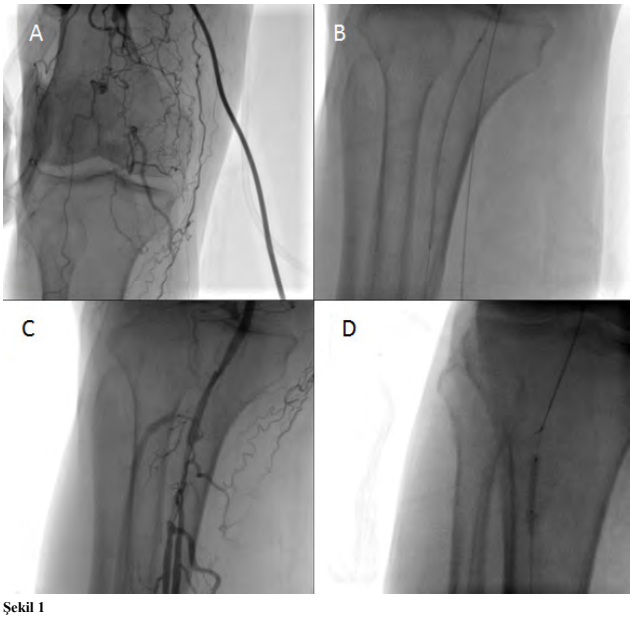
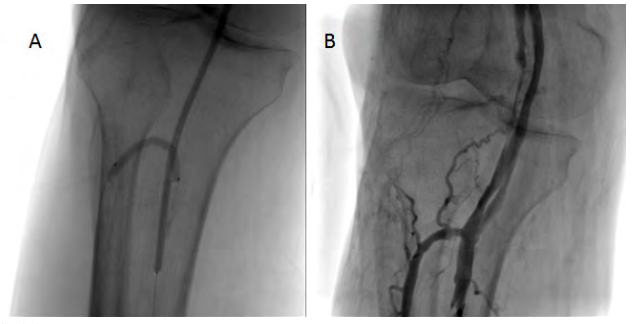
Olgu: 70 yaşında bilinen koroner arter hastalığı hipertansiyonu ve diyabeti olan erkek hasta, kliniğimize sağ alt ekstremitede progressif artış gosteren klodikasyo ve son 2 haftadır devam eden istirahat ağrısı şikayeti ile başvurdu (fontaine class 3). Yapılan doppler ultrasonografide diz altı arterial sistemde darlık ve nabızsızlık tesbit edilmesi üzerine perifer anjiyografi planlandı ve yapılan anjıyografide sağ alt ekstremite diz altı kronık total darlık tesbit edildi (Şekil 1a). Hastaya başka bir seansta antegrad yaklaşım yoluyla sağ femoralden sheat yerleştirildi, 0.035 minie kateter desteği ile 0.035 stiff body ve astato tel kullanılarak lezyon geçilemedi, bunun üzerine retrograd yol ile posterior tibial arter ponksyonu yapıldı 4F STS kateter desteği ile 0.035 stiff body kullanılarak lezyon geçildi, snare ile yakalanarak sağ femoralden externe edildi, ardından 2.0x80 ve 4.0x150 mm boyutlarında balonlar kullanılarak PTA (perkütan translüminal anjioplasti) yapıldı (4 dk) (Şekil 1b, c). Anterior tibial arterin dik açı ile çıkması nedeniyle tel ile dönülemedi öncelikle distalde balon şişirilerek geçilmeye çalışıldı başarılı olunamaması üzerine twinpass kateter desteği ile conquest 12, astato ve fielder telleri ile geçilmeye çalışıldı ancak başarılı olunamadı (Şekil 1d). Bunun üzerine dorsalis pedise sheat yerleştirildi retrograd yol ile anterior tiabial arterden popliteal artere fielder tel ile geçildi. 3.5x40 ve 3.0x120 mm boyutlarında balonlar ile kissing balon yapıldı (Şekil 2a), ardından popliteal artere 5.0x120 mm balon ile dilatasyon yapıldı ve tam açıklık sağlandı (Şekil 2b).

Sonuç: Olgumuzda 70 yaşında diz altı kronik total darlığı olan ve istirahat ağrısı tarif eden bir hastada antegrad yaklaşım ile basarısız olunması sonrası, retrograd yaklaşım yoluyla anterior ve posterior tibial arter ponksiyonu eşliğinde başarılı revaskülarizasyon tartışılmıştır.



Şekil 1



Şekil 2

SO-52

Proksimal emboli koruma cihazı (Mo.MA) ile proksimal ve distal internal karotis arter lezyonlarının aynı seansda stentlenmesi

Yakup Alsancak[1], Mehmet Bilge[2], Sina Ali[1], Ayşe Saatçi Yaşar[1], Serkan Sivri[1], Mustafa Duran[1], Zeynep Şeyma Turinay[2], Filiz Özçelik[2], Gamze Zengin[2]

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Giriş: İnme, tüm dünyada mortalite ve morbitidenin önde gelen sebeblerindendir. Özellikle karotis arterlerde bulunan darlıklar inmeye neden olan durumların başında gelmektedir. Seçilmiş hastalarda, karotis arter darlıklarının daha az invazif bir yol olan perkütan yolla tedavisinin birçok çalışma ile etkinliği ve güvenilirliği gösterilmiştir. Bu bildiride, yakın zamanda serebrovaskuler olay geçiren bir olguda aynı seansta Mo.Ma proksimal emboli koruyucu kateter sistemi ile ekstrakraniyal ve intrakraniyal karotis arter stentleme işlemi sunulmaya çalışıldı.

Olgu Sunumu: Diyabet, hipertansiyon ve koroner arter hastalığı (stent ve by-pass) tanılarıyla takipli olan 67 yaşında erkek hastada 1 ay önce meydana gelen sağ kolda uyuşma ve konuşma bozukluğu olması üzerine nöroloji bölümünde değerlendirilmiş.Hastaya yapılan selektif karotis anjiyografi sonrası sol İCA'nın ekstrakraniyal kesiminde bulunan büfirkasyonun hemen üzerindeki ciddi lezyona (Video 1) perkütan girişim kararı alındı. Hastanın kontrol görüntülenmesinde İCA'nın intrakraniyal petroz bölgesinden laceruma doğru uzanan kesiminde ciddi ve trombüslü darlık izlendi (Video 2). Hastada tekrarlayan olay ihtimali göz önüne alınarak İCA'nın intrakraniyal bölgesinde bulunan bu lezyonun perkütan yolla açılması ve ardından ekstrakraniyal bölgede bulunan darlığın açılması planlandı. 9F Mo.Ma sistemi hastanın sol İCA'sına uygun biçimde yerleştirildi. Ardından 0,014 mm hidrofilik kılavuz tel ile önce proksimalde bulunan ve ardından distalde bulunan ciddi lezyon geçildi (Video 3). Distalde bulunan trombüslü lezyona topografik görüntülere dikkat edilerek 4.0x20 mm koroner arter çıplak metal koroner stent 14 atm ile direkt olarak implante edildi (Video 4). Sonrasında 7x7x30 mm self-expandable stent proksimal darlığa implante edildi (Video 5). Arkasından 4.0x20 mm balon ile proksimal stentte postdilatasyon yapıldı (Video 6). EKA ve AKA balonlarının sırasıyla indirilmesinden sonra alınan görüntüde sonucun optimal olduğu saptandı (Video 7,8).

Tartışma: Karotis arterlerde bulunan lezyonların büyük çoğunluğu (%90-95) karotis büfirkasyon bölgesinde görülmektedir. Distal karotis arter hastalığı, karotis arter stenozu nedeniyle tedavi gerektiren olguların çok küçük bir kısmını içermektedir (%0.4). İCA'nın intrakranial bölümünde bulunan tıkanıklıklar önemli oranda inme sebebi olmasına rağmen (%10) çoğunlukla medikal tedavi tek seçenek olarak kabul edilebilmektedir. Bu gibi durumlarda intrakranial İCA darlığının giderilmeden tek başına ekstrakraniyal İCA darlığının giderilmesi tedavinin eksik kalması nedeniyle pek akıllı bir seçim gibi durmamaktadır. Mo.Ma emboli koruma cihazlarının bu yönde kullanımı giderek yaygınlaşmaktadır. Diğer taraftan Mo.Ma kullanarak aynı anda iki lezyonu da stentlemek teorik olarak mümkün ancak distal İKA lezyonuna stenti kontrast vermeden implante etmek oldukça zordur (Mo.Ma sistemi ile oklüzyon sırasında kontrast verilemez). Bu olgu Mo.Ma sistemi kullanılarak yapılan ender vakalardandır.

SO-53

The bronchial obstruction as a complication of endovascular repair of aortic pseudoaneuyrsm in Behçet's Disease

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A46 year-old man presented with hemoptysis and palpitation to the our emergency department. He had a history of Behçet's Disease (BD) for ten years and three weeks before admission he was operated for lumbar hernia. Before the operation he had discontinued of immunosuppressive medications which included steroids and azathiopirine. A contrast enhanced thorax computed tomography (CT) was performed for a possible pulmonary artery aneurysm rupture and other emergent chest disease. CT showed a 8,0x9,5 cm aortic pseudoaneurysm with contained rupture in the aortic arcus and diameter of proximal landing zone was 29 mm but aortobronchial fistula did not clearly demonstrate (figure 1 A,B). The patient was taken emergently to the interventional unit, the pseudoaneurysm was excluded with implantation of the endovascular Valiant stent graft, 30x100 mm (Medtronic, Santa Rosa, CA) via the left femoral approach under conscious anesthesia and there was no endoleak on the angiography (figure 1 C,D). Six hours later he complained about dyspnea. A chest roentgenogram was taken and showed the left lung area was completely opacified (figure 2). Chest CT was performed and demonstrated the pseudoaneurysm in endovascularly repaired aorta was growth progressively and obstructed the left main bronchus (figure 1 E). After routine preparation, a fully covered self expanding stent (18x14x14 carinal Y stent, Novotech Dumon Y stent,Boston Medical Products,Westborough,MA,USA) was successfully placed in trachea and main bronchi (figure 1 F). After this procedure a second Valiant stent graft, 34x100 mm was implanted more proximally into first endovascular graft with no endoleak. We present a case about aortic pseudoaneurysm with BD and we implicated three points; the first one is the safety and effectiveness of endovascular repair of the aortic pseudoaneurysm in patients with BD. The second one explains the importance of immunosuppressive drug continuation before and after the repair.

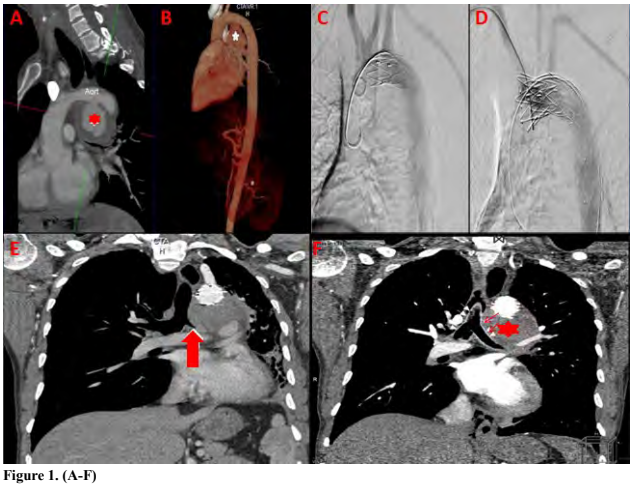

Figure 1. (A-F)

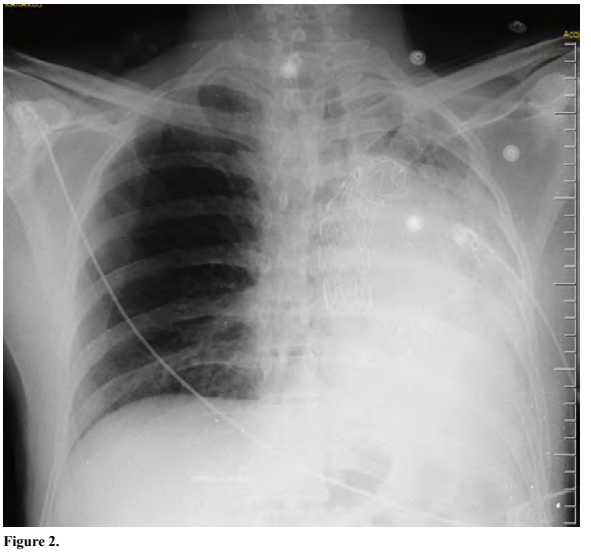

Figure 2.

Diğer

SO-54

Intramyocardial dissecting and giant haematoma: an unusual rare complication of subacute myocardial infarction

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A 69-year-old woman patient with history of diabetes mellitus, hypertension, and previous smoking was admitted the intensive care unit with complain of chest pain of 5 days duration, general malaise and cardiogenic shock. The ECG showed sinus rhythm and a large anterior Q-wave without ST-segment modification. Transthoracic echocardiography showed left ventricular dysfunction with a left ventricle ejection fraction of 25%, extensive akinesia the anterior, antero-septal and inferior-septal walls. At the apical-septal region a pulsatile cavity with systolic expansion surrounded by a thin endomyocardial border was visualized. This lesion characterized by a regular mobile hyperechoic edge and heterogeneous content with circular echo-free spaces within intermediate echodensity sludge Colour-Doppler interrogation did not demonstrate any flow within that structure. We interpreted these images as intramyocardial dissecting hematoma and underlying intramyocardial hematoma (figure 1) An intramyocardial dissecting haematoma was confirmed by computed tomography imaging, which showed an apical lesion of the left ventricle with liquid intensity separated from the left ventricular cavity by a flaccid edge (figure 2) These findings were consistent with acute ST elevation anterior myocardial infarction complicated with probable Intramyocardial dissecting haematoma (IDH).A conservative approach was taken. Due to the patient serious comorbidities and death occurred 2 days after admission. IDH is a rare and unusual form of myocardial rupture complicating acute myocardial infarction. Two types of myocardial rupture have been previously described: simple and complex (hemorrhagic dissection). Pathologic descriptions of cases with free wall rupture by Lewis. demonstrated that a linear or direct tear through the myocardium is most frequently detected (79% of cases), whereas in the second type of rupture (9% of cases) infiltration with blood within the ventricular wall is noted.

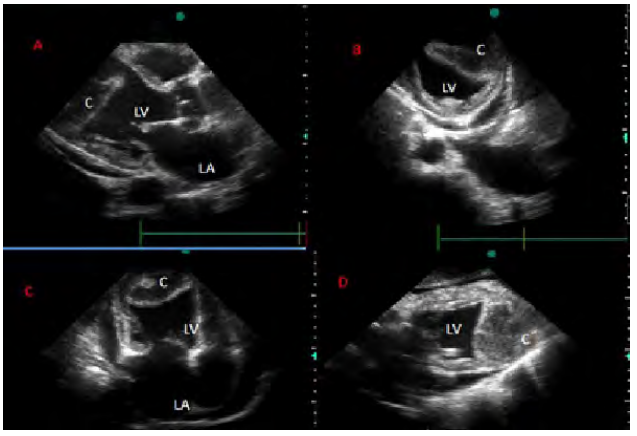

Figure 1. TTE views of intramyocardial dissecting hematoma.

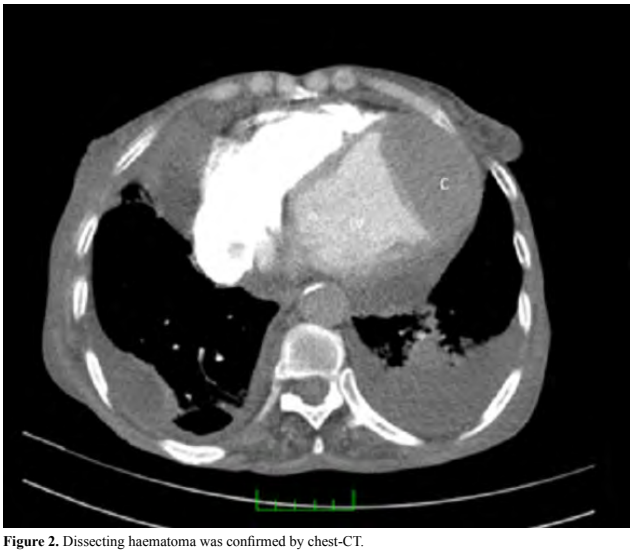

Figure 2. Dissecting haematoma was confirmed by chest-CT.

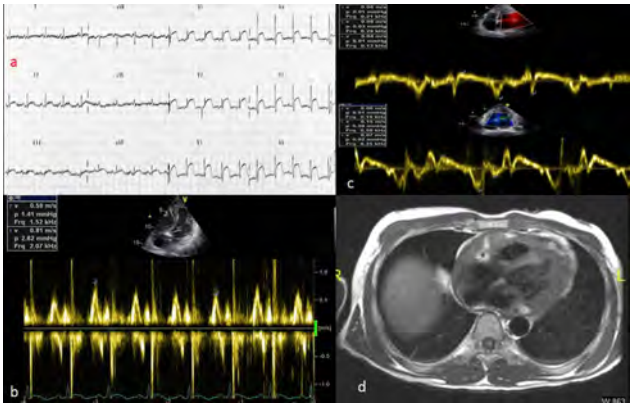

Figure 1. (A) ECG, **(B, C)** ECHO, **(D)** MRI.

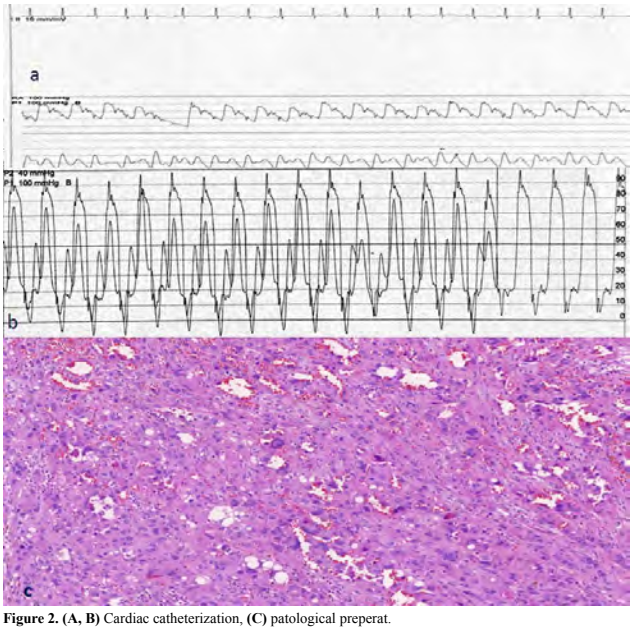

Figure 2. (A, B) Cardiac catheterization, **(C)** patological preperat.

SO-55

Constrictive pericarditis due to angiosarcoma mimicking acute ST elevation myocardial infarction

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Ankara University Faculty of Medicine, Ankara

Constrictive pericarditis(CP) is a very rare condition and its etiology is most commonly idiopathic. We report a case of CP whose ECG mimicked acute anterior myocadial infarction due to cardiac angiosarcoma.

Case: A 38 year-old male was referred to our clinic for substernal chest pain and dyspnea for the last 3 months. He had developed these symptoms after a severe upper respiratory tract infection. He had no risk factors. Physical examination revealed jugular venous distention, pericardial knock, hepatomegaly and +1 pitting pretibial edema. His ECG showed newly developed ST segment elevation on V1-6 (Figure 1a) consisted with anterior myocardial infarction. Patient was hospitalized but coronary angiography was normal. Echocardiography showed pericardial effusion with thickened pericardium, septal bounce (Video), ≥25% increase in mitral E velocity during expiration (Figure 1b), and annulusus paradoxus (Figure1c). Cardiac MRI demonstrate thickened pericardium with pericardial effusion (Figure 1d). Cardiac catheterization showed a preserved x descent, a prominent y descent on right atrial pressure trace (Figure2a) and exaggerated ventricular interaction on ventricular pressure trace (Figure 2b) that consisted with CP. Pericardiectomy and pericardiocentesis was performed. 1000cc of pericardial effusion was drained. Screening procedures for infection, vasculitis and connective tissue disease were all negative. Patological examination of pericardiectomy preparates showed angiosarcoma (Figure 2c). After the diagnosis of anjiomyosarcom had been made, the patient was reffered to oncology clinic.

Discusion: CP is a rare disease and its etiology is most commonly idiopathic. Primary cardiac angiosarcoma is exceptional tumor but is the most common primary cardiac malignant tumors in adults. Pericardium involvement may be seen in cardiac angiosarcoma but there is no literature about CP. To our knowledge, this case is the first presentation of CP due to cardiac angiosarcoma.

SO-56

Percutaneous mitral annuloplasty in a patient with coronary sinus stenosis and coronary artery compression during procedure

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As a novel alternative choice of treatment, percutaneous interventions have been developing recently such as Carillon device functional mitral regurgitation (FMR). The stenosis of the CS may seem like obstacles but it may not interfere the procedure. A severely symptomatic, 64-year-old male with ischemic heart failure and severe FMR was referred to our clinic. There were systolic dysfunction (ejection fraction 25%) and severe FMR on transthoracic echocardiography. There was a severe narrowing on proximal-mid part of the CS. Because of the high surgery risk, we decided to implant the percutaneous mitral annuloplasty(PMA) device with the Carillon system to the patient. Before starting the PMA, a drug eluting stent was implanted to distal part of the CX. After the coronary sinus angiography with marked pigtail catheter via right internal jugular vein, the distal anchor of the device was released. After the application of adequate tension, a severe compression was done on the mid-part of the CX artery. The proximal anchor of the device was not implanted and all the system was removed. This time a new device was planned to implant to the more proximal part of the CS. The coronary sinus angiography with marked pigtail catheter was done again and the distal anchor of the device was released 3 cm below the former location. After the application of adequate tension, 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 this procedure the echo parameters of FMR further improved. Vena contracta diameter of the FMR and annular dilatation were also diminished. In conclusion, the coronary sinus based annuloplasty with Carillon system for FMR can be done easily in the case of the narrowed CS. In addition, in case of CX compression, the device could be removed and replaced with a new one.

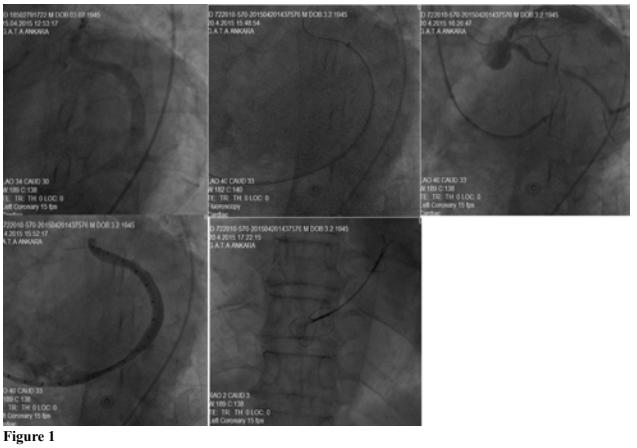

Figure 1

SO-57

Chronic traumatic aorta-caval fistula form stab wound

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İzmir KÇÜ Atatürk Training and Research Hospital, İzmir

Chronic aortocaval fistula (ACF) is a rare complication of a stab wounds to the abdomen. we report one case of traumatic ACF. Although infrequent, penetrating injuries of the aorta and vena cava are among the most lethal injuries to the intra-abdominal vessels, Gunshot or fragment wounds, stab wounds, and iatrogenic injuries account for 90% and blunt trauma accounts for the remaining 10% of all such vascular injuries. A 25-year-old man presented to our hospital with complaint of shortness of breath. Over the past one month, He could walk only short distances owing to exertional dyspnea and lower extremity edema. Four years previously, He had undergone a stab wound to abdomen. On physical examination, the patient had mild respiratory distress. His pulse rate was 130 beats per minute and his blood pressure was 110/70 mmHg.There was Jugular venous distention. The lung fields were normal. Auscultation of the heart revealed a grade 3/6 systolic murmur. A pulsatile mass in the right paraumbilical area with continuous abdominal bruit was detected. He had lower extremity edema, the peripheral pulses were all palpable. His oxygen saturation was 91% at room air.impaired. Cardiac catheterization revealed a mean right atrial pressure of 15 mmHg, a right ventricular pressure of 60/15 mmHg, and a pulmonary capillary wedge pressure of 12 mmHg. The Qp/Qs ratio was 2.7.A Coronary angiography showed normal coronary arteries. An Aortography revealed aorta-caval fistula which was in line right renal artery. It was showed that Right atrium and RV were filled with contrast agent. Computed tomography (CT) scanning was performed and showed aneurysm in line right renal artery. There was a fistula between aorta and vena cava, which was 7 mm diameter at its widest point. Chronic ACF is rare and has hemodynamically diverse pictures, and it is often misdiagnosed. The treatment of traumatic or secondary ACF is surgical closure of the fistula or endovascular therapy.

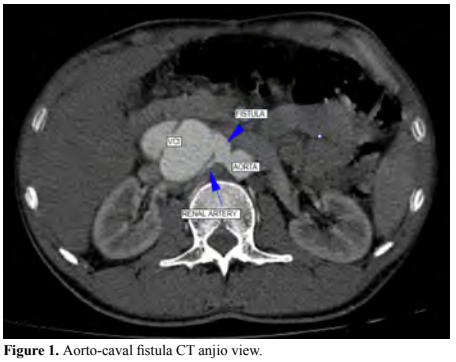

Figure 1. Aorto-caval fistula CT anjio view.

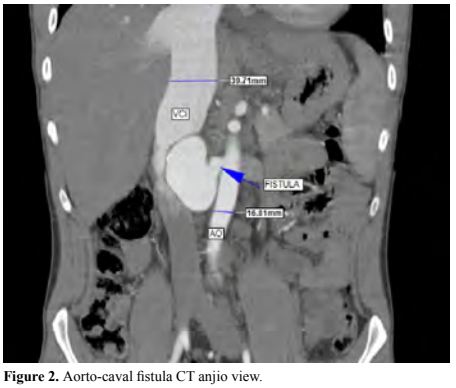

Figure 2. Aorto-caval fistula CT anjio view.

SO-58

Fulminant eosinophilic myocarditis with diffuse coronary spasm in a 18 year-old man

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

Introduction: Eosinophilic myocarditis is a clinical condition that can be mortal due to severe heart failure. Different factors such as leukemia, parasite infection, allergic disease, granumatous disease, connective tissue disease, vasculitis such as Churg-Strauss syndrome, or primary hypereosinophilic syndrome may play a role in the etiology. Certain diagnosis is based on endomyocardial biopsy.

Case presentation: A 18 year-old man was admitted to hospital because of chest pain, fatigue and syncope. Initial electrocardiography revealed ST segment elevations in lead D1, AVL, V2 trough V6 with resiprocal changes in leads D2, D3, aVF. Echocardiography showed a left ventricular ejection fraction of 20% and wall thickening with severely decreased wall motion. His first angiography showed diffuse coronary spasm in LAD and CX with a fully normal ventriculography.His general condition and left ventricular function deteriorated again so that the patient immediately received an intra-aortic balloon pump. After confirming the diagnosis of myocarditis, high-dose corticosteroids were administered. After that his control angiogram showed completely normal coronary arteries. Right ventricular endomyocardial biopsy was performed in the acute phase and showed extensive eosinophilic inflammatory cell infiltration, severe interstitial edema and moderate myocardial necrosis. After starting high-dose corticosteroids, his ventricular functions dramatically improved, and the patient made a full recovery.To best of our knowledge this is the first case of eosinophilic myocarditis with diffuse coronary spasm in literature.

Conclusion: Acute eosinophilic myocarditis is extremely rare and rapidly progressive clinical condition with poor prognosis. Although observational series suggest potential clinical benefits of corticosteroid therapy, the best strategy is to remove causative agent when known.

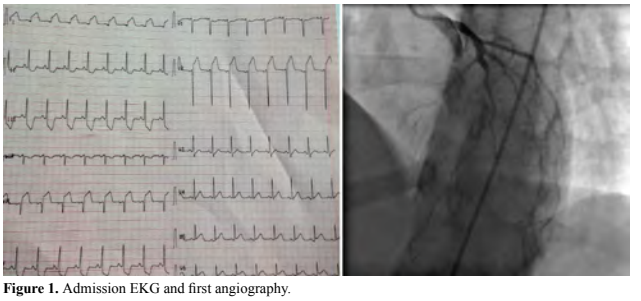

Figure 1. Admission EKG and first angiography.

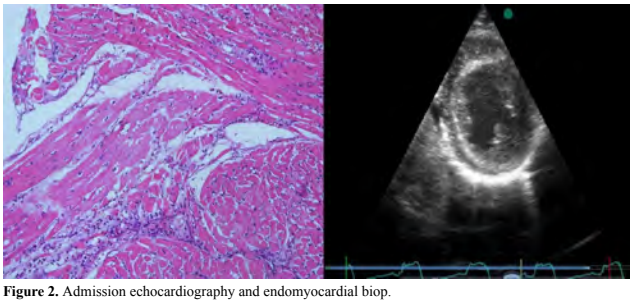

Figure 2. Admission echocardiography and endomyocardial biop.

SO-59

Rapid recovery of the myocardial systolic dysfunction in a young patient with carbon monoxide poisoning treated with hyperbaric oxygen theraphy

Ahmet Gündeş, Emrah Yeşil, Ahmet Çelik, İsmail Türkay Özcan

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Introduction: Carbon monoxide (CO) poisoning an important health problem because of its severe clinic effects and its large proportion in the rate of toxically caused deaths. The major cause of death from CO poisoning is myocardial injury and dysfunction and myocardial injury is common in moderate to severe CO poisoning. Limited data exist regarding hyperbaric oxygen theraphy (HBO2) for prevention of myocardial injury. We present a case of CO poisoning treated with HBO2 theraphy in a patient with severe myocardial systolic dysfuction.

Case: 18 year-old patient was refered to our hospital because of carbon monoxide poisoning. The etiology of the CO poisoning was using burning charcoal to heat. On admission, he had confusion and Glasgow Coma Score was 9. In his medical history, there were no history of cardiovasculer and neurological disease. His blood pressure was 100/75 mmhg and electrocardiogram showed normal sinus rhythm. On laboratory study, at first, troponin and CkMb levels were normal but they increased on follow up (troponin I=0,79 ng/mL, CkMB=8,7ng/mL). Echocardiography showed severe global left ventriculer dysfuction, ejection fraction was %36 on M-mode (figure 1). Because of consciousness, and myocardial depression, the patient was treated with hyperbaric oxygen theraphy. The HBO2 protocol was one treatment for 90 min on 100% oxygen at 2.4 atm. After one day, we observed rapidly recovery in myocardial systolic function. Echocardiogram was revealed normal ejection fraction (figure 2). The patient was discharge on day 3.

Conlusion: Myocardial injury is common in moderate to severe CO poisoning and hyperbaric oxygen theraphy can be an affective treatment for patients with myocardial systolic dysfuction.

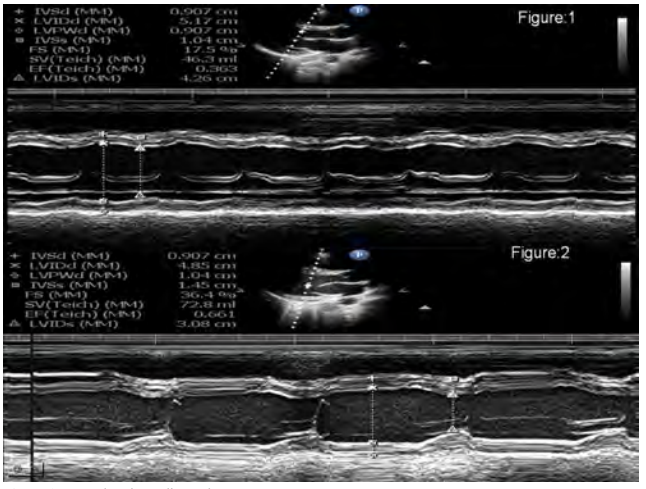

Figure 1. M-mode echocardiography.

SO-60

Massive left atrial invasion of a pulmonary metastatic tumor through left pulmonary veins

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Medical Park Hospital, Samsun

Cardiac malignancies can be of a primary or metastatic origin. While benign myxomas constitute the most of the primary tumors of the heart, sarcomas and other tumors of malign nature show a metastatic cardiac invasion. Primary or metastatic tumors of the lung may extend via pulmonary veins to the left atrium creating symptoms similar to congestive heart failure and mitral stenosis. At this time malignancy is usually in an advanced stage and palliative treatment may be the only treatment option. We present a case of a 30 year old female patient who presented at our emergency department with severe dyspnea and hematemesis. She had underwent knee operation due to osteosarcoma 6 years ago, and left pulmonary wedge resection due to pulmonary metastasis 4 years ago. She was extremely hypoxic, dyspneic and tachycardic. Chest X-ray revealed extreme bilateral infiltration and multiple calcifying lesions on both lungs, especially on the left side. Transthoracic echocardiogram showed a huge mass obliterating left atrium (Fig. 1). Aggressive diuretic and supportive medical therapy was administered in intensive care unit and a slight amelioration was observed the following hours. Beside multiple bilateral lung lesions, a giant calcification originating on the left lung extending through both of the left pulmonary veins to the left atrium was confirmed by chest CT-scan. A multidisiplinary counseling decided palliative surgery. She underwent left atrial mass excision under cardiopulmonary bypass (Fig. 2). Dramatic clinical recovery was noticed on the postoperative follow up. Pulmonary edema and hypoxia were relieved and the patient was discharged after 1 week. At the 3rd postoperative month she remains asymptomatic and under oncologic surveillance. Metastatic invasion of the heart can be seen in advanced stages of sarcomas. At this time cardiac symptoms may prevail those of malignity. Cardiac mass resection is lifesaving and necessary to decrease symptoms.

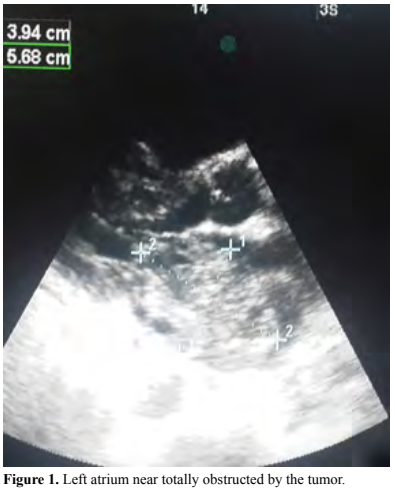

Figure 1. Left atrium near totally obstructed by the tumor.

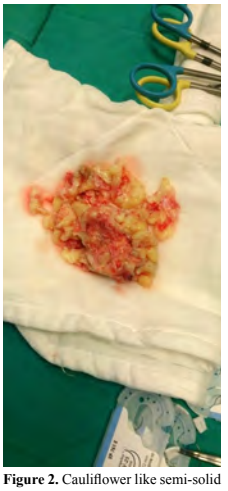

Figure 2. Cauliflower like semi-solid semi-gelatinous mass.

SO-61

A rare case of partial pulmonary venous return anomaly

Cengiz Öztürk[1], Atila İyisoy[1], Turgay Çelik[1], Zekeriya Arslan[2], Sait Demirkol[1], Murat Ünlü[1], Şevket Balta[1], Uğur Bozlar[1]

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[2]*Ankara Mevki Military Hospital, Ankara*

Partial anomalous pulmonary venous return, which describes one or more pulmonary veins (PVs) drain into a systemic vein or right atrium (RA), is a rare congenital anomaly that is seen at only 0.5-0.7% of the general population. Superior and inferior vena cavae, RA, azygous vein, portal or hepatic vein may collect the right sided PVs, while left brachiocephalic vein or coronary sinus may collect the left sided PVs. They coexist with interatrial septal (IAS) defects in about 80% of the patients; especially in asymptomatic cases. Theoretically, it is asserted that partial pulmonary venous return anomaly occurs due to a failure of connection between the common PV and the splanchnic plexus while caudal regression to the lung buds occurs in the early embryonic development phases. Transthoracic (TTE) and transesophageal echocardiography (TEE), computed tomography angiography and catheter angiography are the tools for the diagnosis. We herein present a 21 year old male patient who applied for our clinic with a complaint of exertional dyspnea. Physical examination, ECG and serum biochemistry revealed no any abnormal finding. TTE showed that the right sides of the heart were dilated, left ventricular systolic functions were normal, and the left atrium (LA) was in 40 mm in diameter. There was a mild tricuspid valvular regurgitation with a calculated pulmonary artery sistolic pressure of 33 mmHg. TEE also confirmed the intact IAS and dilated right sides. Cardiac CT showed that the left lung superior lobe vein and inferior lingula vein were merged, draining into left brachiocephalic vein (partial venous return anomaly) (Figure 1). Left lung inferior lobe PV (LLILPV) was draining into left atrium. This vein had an 50% luminal obstruction just before the joining to LA (Figure 2). A narrow sized superior lingula vein of the left lung superior lobe was draining into LA with a different orifice. Vena cava superior and the right heart chamber dilatations were also confirmed by CTA.

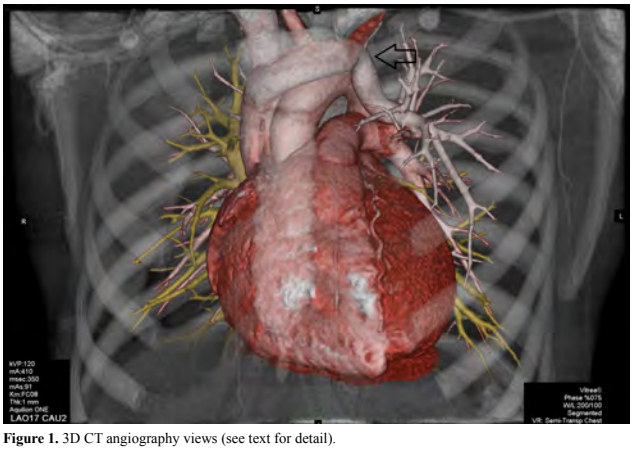

Figure 1. 3D CT angiography views (see text for detail).

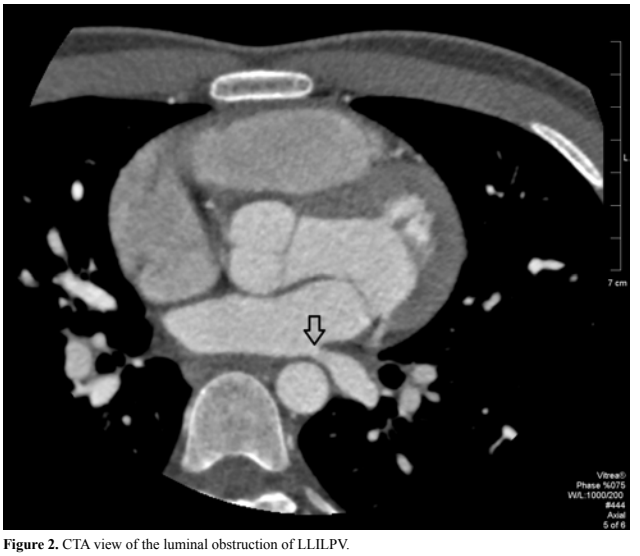

Figure 2. CTA view of the luminal obstruction of LLILPV.

SO-62

Left pulmonary artery agenesis with collateralisation from all of the major coronary arteries

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A 71 year old female patient was admitted to our clinic with symptoms of exertional dyspnea and angina and palpitations. Physical examination showed irregular beats and 2/6 pansystolic murmur audible at apical focus. Electrocardiogram revealed atrial fibrillation with a ventricular rate of 118 beats per minute. Transthoracic echocardiography showed normal systolic funtion of the left ventricle, moderate mitral and tricuspid regugitation with a systolic pulmonary artery pressure (sPAB) of 75 mmHg. Also, left atrial dilatation (57 mm) was detected. Coronary angiography was performed which revealed collateralisation from the proximal segments of each of the three coronary arteries (left anterior descending (LAD) artery, circumflex (Cx) artery and right coronary artery (RCA) into the left hypoplastic lung without any stenosis in epicardial coronary arteries. In order to define the exact anatomic structure of the heart magnetic resonance angiography (MRA) was planned. LPA agenesis was detected in MRA. Also, to define the coronary to pulmonary collateralisation, computed tomography angiography was performed and showed LPA agenesis enlarged left and right atrium and dilated right pulmonary artery. As the patient was symptomatic and the collateralisation were hemodynamically significant, patient was evaluated by a heart team for surgical intervention. As the MRA revealed LPA agenesis, distal pulmonary flow was maintained by coronary arteries via the collateralisation. For that reason it was decided that surgical intervention was not a suitable option for the patient. Therefore, outpatient follow-up was scheduled with full oral medical treatment.

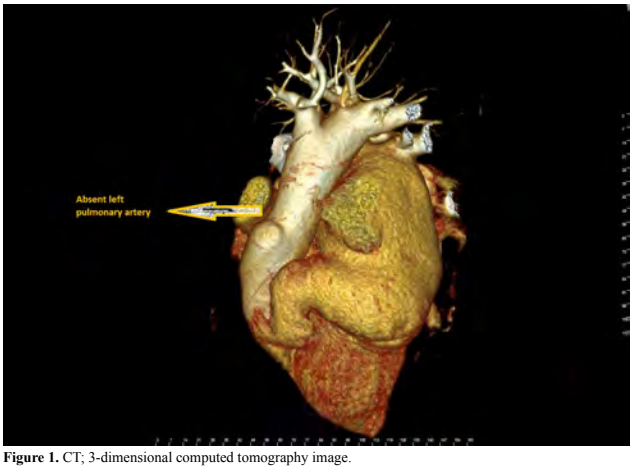

Figure 1. CT; 3-dimensional computed tomography image.

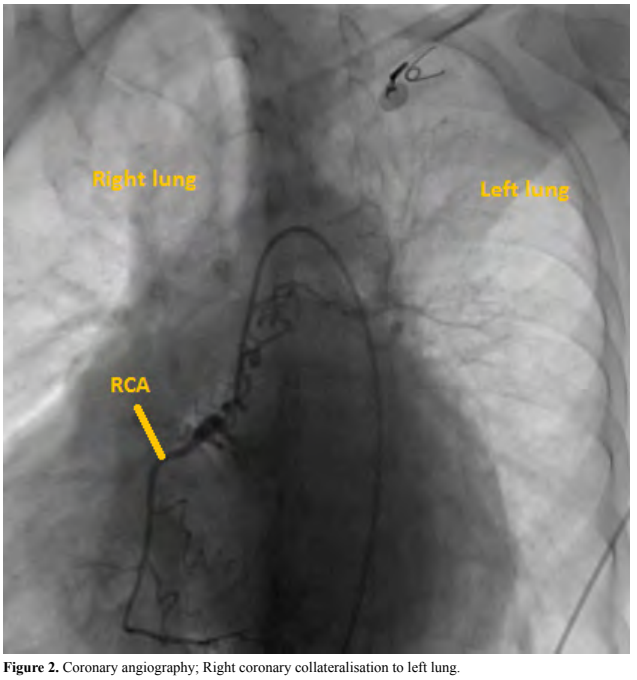

Figure 2. Coronary angiography; Right coronary collateralisation to left lung.

SO-63

Right pulmonary artery agenesis with patent ductus arteriosus and Eisenmenger syndrome

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A 30-year-old woman was admitted to our clinics because of dyspnea, and cyanosis. The left PDA with existing pulmonary arterial hypertension (PAH) and Eisenmenger's syndrome had been diagnosed already. She described repetitive respiratory infections, and episodes of hemoptysis in childhood. Our patient had central cyanosis with prominent clubbing of the toes but not of the fingers. (Figure 1) Chest X-ray revealed decreased volume of the right lung, and elevation of the right hemidiaphragm. (Figure 2) Echocardiography did not demonstrate the PDA. There was also no tricuspid regurgitation that, pulmonary artery pressure could not be estimated. However right ventricle was hypertrophied and enlarged with paradoxical septal motion. Catheterization confirmed the presence of severe PAH with systolic PAP of 155 mmHg and PVR of 32 Woods. Patent ductus arteriosus was also demonstrated with a right to left shunt and Qp/Qs of 0.79. (Figure 3). During contrast injection of pulmonary arterial trunk, it was realized that the right lung were not perfused well. During contrast injection of arcus aorta well developed MAPCA to right lung were also demonstrated. (Figure 4) Owing to these findings, unilateral absence of pulmonary artery (UAPA) was suspected. Contrast-enhanced computed tomography angiography of the chest proved the absent right pulmonary artery with dilated main and left pulmonary artery, multiple collaterals from aorta supplying right lung and a large PDA (Figure 5). Of note was the asymmetry of thoracic cavity with a smaller hemithorax ipsilateral to the affected lung. (Figure5). Accordingly, the final diagnosis was right UAPAwith PDA accompanying Eisenmenger syndrome. She underwent drug therapy to improve cardiac capacity and a heart-lung transplantation was recommended to the patient in the long term.

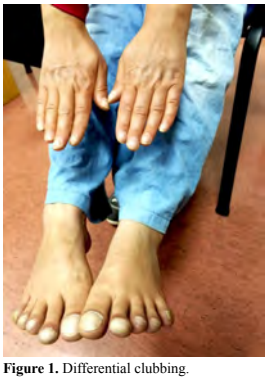


Figure 1. Differential clubbing.

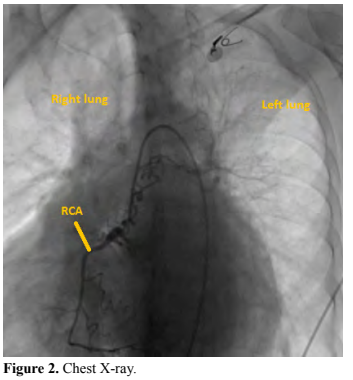
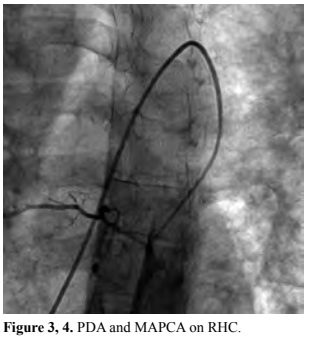


Figure 2. Chest X-ray.



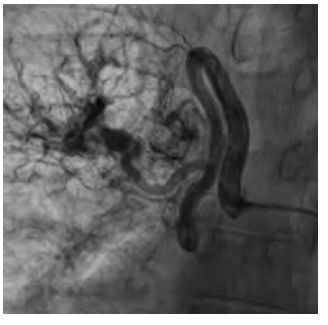


Figure 3, 4. PDA and MAPCA on RHC.

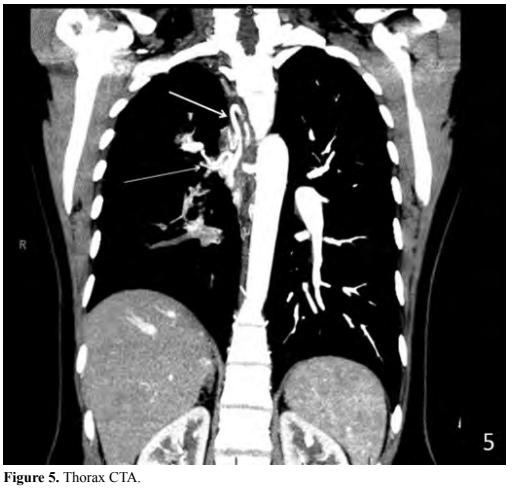


Figure 5. Thorax CTA.

SO-64

Left main artery compression by a giant pulmonary artery aneurysm associated with large atrial septal defect

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Pulmonary artery aneurysms (PAA) are uncommon. Depending on their size and location, they may lead to complications such as extrinsic compression of the left main coronary artery as we describe in our case. A 57-year-old woman presented with complaints of chest pain at rest and exertional dyspnea. On admission, a blood pressure of 100/60 mmHg. Chest X-ray showed prominent bilateral pulmonary artery enlargement. Transthoracic echocardiography demonstrated extreme dilatation of the main pulmonary artery and its branches without severe pulmonary hypertension and dilated right cardiac chambers. Transesophageal echocardiography confirmed the presence of dilated pulmonary arteries and there was no thrombus and it revealed a 2.8 cm in size prominent secundum type atrial septal defect. She was referred for invasive angiography, which revealed a 90% stenosis of left main coronary artery. At cardiac catheterization, pulmonary artery pressure 48/25 mmHg (avg; 36 mmHg). Computed tomography showed important pulmonary aneurismal dilatation of main pulmonary artery (main pulmonary arteries were measured as 70 mm) with left main coronary artery compression. PAA is a rare lesion of the thoracic cavity, with an approximate incidence of 1 in 14,000 in autopsy series. Nowadays, aneurysms are associated with congenital heart disease (PDA,VSD,ASD) in more than 50% of cases. İn our case, Behcet's disease or other connective tissue disorders and infections such as syphilis, tuberculosis were not found.

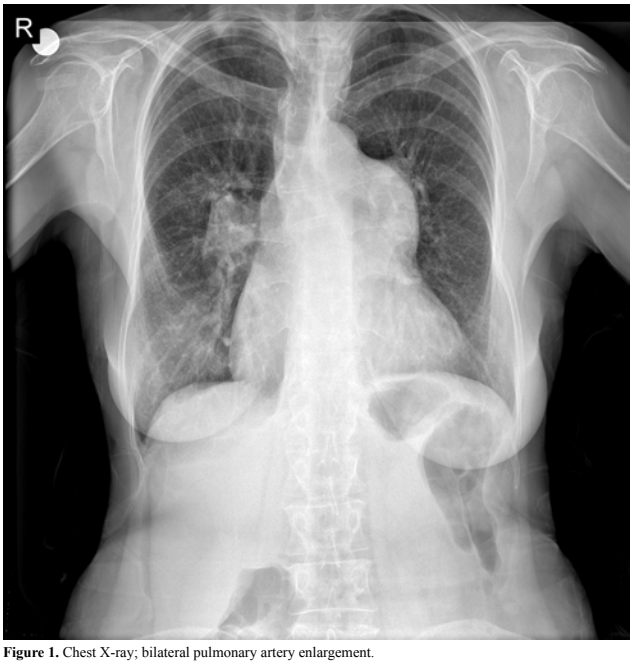

Figure 1. Chest X-ray; bilateral pulmonary artery enlargement.

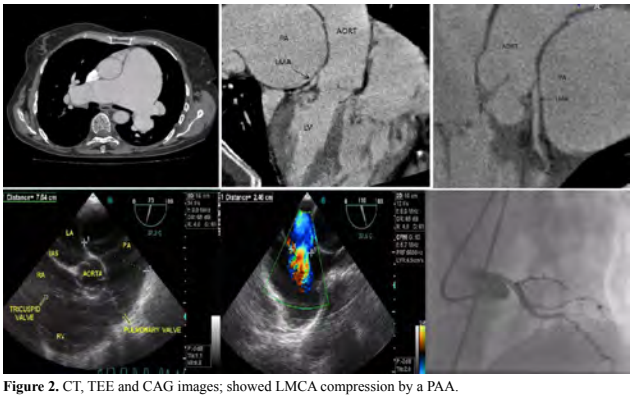

Figure 2. CT, TEE and CAG images; showed LMCA compression by a PAA.

Pulmoner hipertansiyon/ Pulmoner vasküler hastalık

SO-65

Isolated unilateral absence of pulmonary artery concomitant with pulmonary embolism diagnosed in the adulthood

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Unilateral absence of pulmonary artery(UAPA) is a rare congenital anomaly due to malformation of sixth aortic arch during embryogenesis. It can be isolated but often associated with other cardiovascular abnormalities. Patients with isolated UAPA may remain clinically silent until adulthood but they are usually symptomatic. Clinical symptoms include exercise intolerance, dyspnea, hemoptysis and recurrent pulmonary infections. Diagnosis is diffucult because of nonspecific symptoms. Based on patients complete medical history, physical examination and by using imaging tools a correct diagnosis can be made. Treatment choices are contraversial which includes; partial or total pneumonectomy, selective embolization of the systemic collaterals or primary versus staged pulmonary artery anastomosis. The overall mortality rate is 7%. Here we present 36 years old male patient with the initial diagnosis of pulmonary embolism. Recently, he has difficulty in breathing even at rest. His oxygen saturation was 98% at room air and other baseline vitals were normal. Physical examination was unremarkable except diminished respiratory sounds on the right hemithorax during pulmonary auscultation.Chest x-ray showed mediastinal shift towards the right side, absence of the right pulmonary artery shadow, right hypoplastic lung and hyperlucency of left lung. 2D transthoracic echocardiography(TTE) was performed. Ejection fraction and transpulmonary artery pressure was 40mmhg. Also, TTE revealed no structural abnormalities. Pulmonary function test revealed a restrictive pattern. Afterwards, pulmonary CT-angiography was performed and revealed absence of right main pulmonary artery, collateral supply from systemic arteries and subsegmentary pulmonary embolism in the left side. The diagnosis of UAPA concomitant with pulmonary embolism was established.

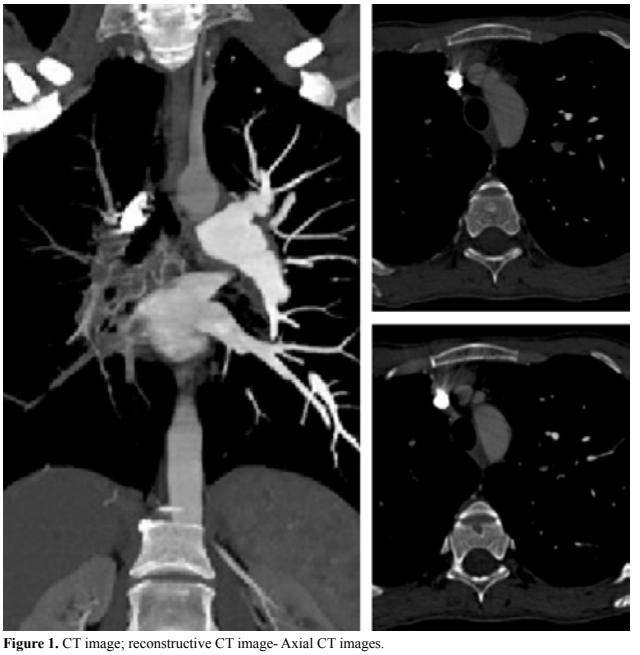

Figure 1. CT image; reconstructive CT image- Axial CT images.

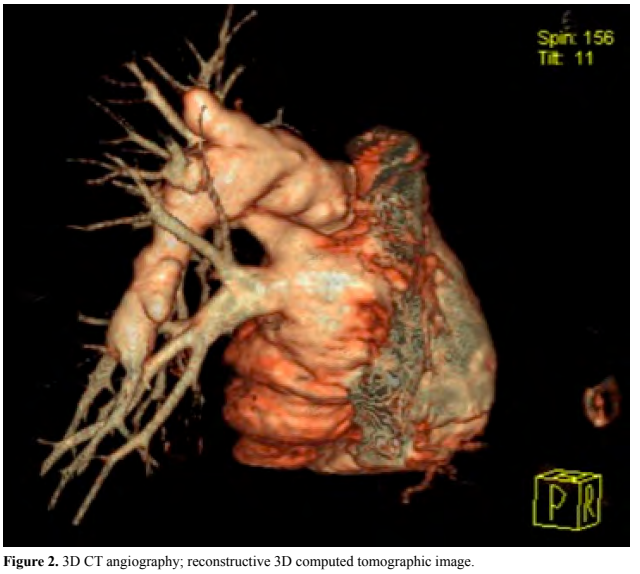

Figure 2. 3D CT angiography; reconstructive 3D computed tomographic image.

Perkütan kapak girişimi

TO-01

Aynı seansta koroner girişim artı TAVİ işlemi artı mitral balon valvüloplasti işlemi uygun mudur?

Ramazan Özdemir

İnönü Üniversitesi Tıp Fakültesi, Malatya

Girişimsel kardiyoloji / Koroner

TO-02

Radiyal anjiyografiden 9 saat sonra ancak damar dışına alınabilen radiyal kateter olgusu; çok ciddi radiyal spazmı

Mehmet Ali Elbey, Zihni Bilik

Dicle Üniversitesi Tıp Fakültesi, Diyarbakır

50 yaşında bayan aile hikayesi pozitif olan hastanın eforla göğüs ağrısı ve nefes darlığı olması üzerine yapılan efor testi pozitif olarak değerlendirildi. Miyokard perfüzyon sintigrafisinde iskemi çıkması üzerine KAG işlemine karar verildi. Sağ radiyal arterden yapılan koroner anjiyografide koroner arterler normal olarak izlendi. Tiger kateter sağ subklaviyan arterde iken çekilmeye çalışıldı ancak kateterin gelmediği izlendi. Tel geri alınarak kateter içinden defalarca nitrogliserin, diltiazem, papaverin, yapılmasına rağmen kateter geri alınamadı. Hastaya aralıklı olarak yaklaşık 15 mg dormicum yapıldı. Kateter ile yapılan görüntülemede sağ dirsek hizasından itibaren radiyal arterde akımın olmadığı izlendi. On bin heparin yapıldı ve hastaya ACT takibi yapıldı. Hasta bu haliyle YBÜ'e alındı. Yaklaşık 2 saat sonra tekrar kateter laboratuvarına alındı. Tekrar diltiazem, nitrogliserin yapıldı ancak kateter yine geri alınamadı. Hasta tekrar YBÜ'e alındı. Yaklaşık 9 saat sonra anestezi kliniği tarafından propofol ile derin sedasyon yapıldı. Hastaya diltiazem ve nitrogliserin infüzyonu başlandı. Kateter içerisinden Diltiazem ve nitrogliserin IV püşe yapıldı ve 0.032 guide wire ile ancak kateter geri alınabildi. Bir gece YBÜ de takip edildi. Hastaya bir gün sonra arteryel doppler ile radiyal arterde akımın olduğu izlendi. Hasta sorunsuz taburcu edildi.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

TO-03

Delayed manifestation of blunt abdominal trauma: infrarenal complete aortic occlusion with spontaneous coronary artery dissection

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Complete aortic occlusion related to blunt trauma is a very rare clinical condition. Most patients present with acute symptoms and signs, delayed manifestation is unusual. We describe a patient with complete abdominal aortic occlusion who presented with spontaneous coronary artery dissection, 14 years after blunt abdominal trauma. Case Report A 43-year-old man was admitted to our hospital because of chest pain and he also suffered from claudication that became progressively more intense during the last two years. He was involved in an explosion 14 years ago when he was a soldier. After the explosion he was thrown from the vehicle and felt on his face, his feet touching his head due to hyperextension of his thighs. After the event, he complained of paraplegia in his lower extremities then he gradually recovered during a period of 15 days. He remained asymptomatic in the following 12 years until he had complaints of claudication and weakness in his lower extremities. The pulses in the upper extremities were normal, however we could not detect the lower extremity pulses. He subsequently underwent coronary angiography via the brachial approach which demonstrated long spontaneous dissection from proximal to distal segment of circumflex artery. Aortography showed infrarenal complete aortic occlusion. We decided to perform percutanaeous intervention for the circumflex lesion. One month after stenting the patient underwent surgery for aortic occlusion. In conclusion, most cases of abdominal aortic injury present acutely with symptoms and signs of neurovascular deficits in the lower extremities. However, symptoms of claudication may develop slowly in the remainder of cases.

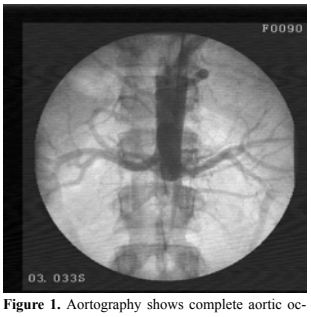

Figure 1. Aortography shows complete aortic occlusion.

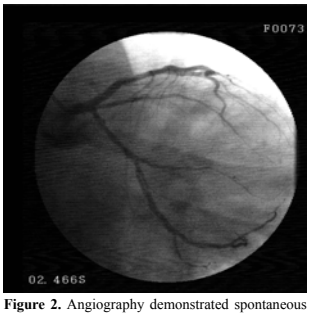

Figure 2. Angiography demonstrated spontaneous dissection.

Kapak hastalıkları

TO-04

Yeni tanı koroner arter hastalığı ve ciddi mitral yetersizliği olan bir hastada hibrid tedavi olarak MitraClip ve off-pump kalp cerrahisi

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Giriş: Mitral yetersizlik (MY) en sık görülen kapak lezyonudur. Konjestif kalp yetersizliği (KY) olan hastaların %15-30'unda orta ya da ciddi MY saptanırken, akut miyokardiyal infarktüs sonrası bu oran %12 olarak bildirilmektedir. Hastalarda gelişen MY, sonraki dönemlerde ortaya çıkacak olan KY ve ölüm riskini göstermesi bakımından önemlidir. Diğer taraftan iskemik kalp hastalığı ile birlikte ciddi MY olan hastalarda kombine by-pass ve kapak cerrahisi, hastanın operatif riskini oldukça artırmaktadır. Bu bildirimizde, perkütan yolla total revaskularizasyonun tam olarak sağlanamadığı, cerrahi riski çok yüksek olan hastalarda MY'nin perkütan tamiri ve sonrasında off-pump kalp cerrahisinin uygulanabilirliğini göstermeye çalıştık.

Olgu: 3 aydır nefes darlığı ve eforla baskı tarzında göğüs ağrısı tarifleyen 65 yaşında erkek hasta poliklinik şartlarında değerlendirilerek koroner anjiyografi planlandı. New York Heart Association sınıflamasına göre sınıf 3 olan hastanın fizik muayenesinde tüm odaklarda duyulan 3/6 şiddetinde sistolik ejeksiyon üfürümü ve akciğer orta zonlarına kadar uzanan krepitan ralleri mevcuttu. Ekokardiyografik incelemede, A2-P2 skallopundan kaynaklanan ciddi MY (Şekil 1) saptanırken ejeksiyon fraksiyonun %20 (LVEDÇ: 6.1, PAB:60 mmHg) olduğu gözlendi (Video 1, 2, 3). Yapılan koroner anjiyografisinde sol ana koronerde %40, sol ön inen koroner arterde ve dallarında ciddi darlık, sirkumfleks arterin ve sağ koroner arterin de tam tıkalı olduğu, sol sistemden retrograd olarak dolduğu izlendi (Video 4). Hastada, semptomatik ciddi koroner arter hastalığı olması üzerine koroner arter by-pass greftleme ve mitral kapak replasmanı planlandı. Kalp damar cerrahisi ile yapılan değerlendirmede, Logistic EuroSCORE'u %20 olan hastanın cerrahi açıdan yüksek riskli olduğu düşünüldü. Hasta için öncelikle MitraClip ve ardından off-pump koroner arter by-pass cerrahisi yapılmasına karar verildi. MitraClip işlemi sırasında A2-P2 skallopuna tek klip yerleştirilerek eser düzeyde MY ile işlemden çıkıldı (Video 5, 6, 7). Ortalama 4 mmHg transmitral gradient izlendi. 1 ay sonra yapılan kontrol ekokardiyografide hafif MY (Video 8) izlenen hastaya atan kalpte LAD-LİMA, diagonal-safen ve RCA-safen by-pass cerrahisi yapıldı. Desteksiz olarak operasyon sonlandırıldı ve hasta kısa süre sonra taburcu edildi.

Sonuç: Koroner arter hastalığı ve ciddi MY birlikteliği hastalarda cerrahi mortaliteyi önemli oranda artırmaktadır. Bu durum özellikle düşük EF'li hastalarda çok daha belirgindir. Ciddi MY ve orta derecede sol ventrikül sistolik disfonksiyonu ile beraber koroner arter hastalığı olan hastalarda standart tedavi koroner by-pass cerrahisine ek olarak uygulanan kapak onarımı ya da replasmanıdır. Ancak bu durum ileri derecede bozulmuş sistolik disfonksiyonlu hastalarda yüksek mortalite oranlarını beraberinde getirmektedir. Bu hastalarda eğer uygulanabilirse perkütan koroner girişim sonrası MitraClip iyi bir tedavi seçeneği olabilir.

Kalp yetersizliği

TO-05

İleri kalp yetersizliği hastasının macerası

Ahmet Ekmekçi

Dr. Siyami Ersek Hastanesi, İstanbul

Aritmi

TO-06

Antikoagülan tedavi altında hemorojik ve embolik serebrovasküler olay geçiren hastada tedavi yaklaşımları

Firdevs Ayşenur İkizler

Ankara Türkiye Yüksek İhtisas Hastanesi, Ankara

Kapak hastalıkları

TO-07

Nadir bir mikroorganizamnın neden olduğu yaygın tutulumlu enfektif endokardit vakası

İlkay Bozkurt, Metin Çoksevim, İdris Buğra Çerik, Okan Gülel, Esra Tanyel, Hakan Leblebicioğlu

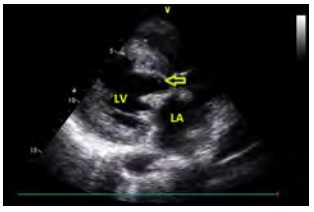
Samsun Ondokuz Mayıs Üniversitesi, Samsun

Giriş: Abiotrophia defectiva'nın etken olduğu nadir bir infektif endokardit vakası sunulmuştur.

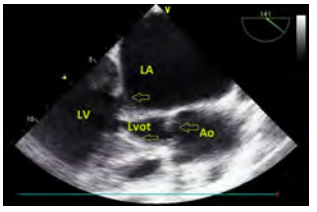
Vaka sunumu: 42 yaşında kadın hasta,1,5 aydır olan üşüme titreme ile yükselen ateş şikayeti ile başvurdu. Özgeçmişinde akut romatizmal ateş geçirdiğ ve son 5 yıla kadar penisilin profilaksisi aldığı öğrenildi. Ateş etyolojisi araştırılmak üzere enfeksiyon hastalıkları kliniğine yatırıldı. Transtorasikekokardiyografi (TTE) yapıldı.Ciddi mitral ve aort yetersizliği saptandı. Belirgin vejetasyon saptanmadı.Ampirik olarak ampisilin sulbaktam 4x2g i.v ve gentamisin 80 mg 2x1 i.v başlandı. 3. gün ateşleri düştü. Kan kültüründe A. defectiva üremesi oldu. Endokardit şüphesinin yüksek olması nedeni ile Transözefajialekokardiyografi (TEE) yapıldı. Mitral kapak atrial yüzde multipl vejetatif odak saptandı. Tedavi altında klinik ve laboratuar bulgularında düzelme gözlenen hasta,

tedavisi 28 güne tamamlanarak cerrahi kapak replasmanına yönlendirildi.Ancak hasta operasyonu kabul etmedi. Taburculuğundan 3 hafta sonra ateş yakınması nedeni ile tekrar başvurdu. TTE'de ve TEE'de mitral ve aort kapak ile sol ventrikül çıkış yolunda vejetatif kitle imajları izlendi. (Figure1-2). Antibioterapi yeniden başlandı. Alınan kan kültürlerinin hepsinde A.defectiva (Duyarlı: Eritromisin, klindamisin, penisilin, ampisilin, sefotaksim) üremesi oldu.Antibiyotik tedavisi 42 gün süre ile verildi. Kontrol kan kültürlerinde üreme olmadı, TTE'de vejetasyon saptanmadı. Tedavi sonrası hastaya mitral ve aort kapak replasmanı yapıldı. Post op 3. ayda relaps gözlenmedi.

Sonuç: Vakayı ilginç kılan özellikler, antibiyotik tedavisinden sonra 4 günden kısa sürede ateşin düşmesine, 4 hafta tedavi süresinin ardından klinik ve laboratuar yanıtı alınmasına ve kontrol kültürlerinde üreme olmamasına rağmen tekrarlamasıdır. Ancak, vakamızdaki komplikasyon potansiyeli yüksek, yaygın kardiak tutulumlu A. defectiva endokarditi uygun antibiyotik tedavisi ile hiçbir komplikasyon ve acil cerrahi gereksinim olmadan tedavi edilebilmiştir.



Şekil 1. TTE PSLAX görünütüsü; mitral ve aort kapak ile LVOT'da vejetasyonlar.



Şekil 2. TEE'de vejetatif imajlar; mitral kapakta, aort'da ve LVOT'da vejetasyonlar.

Pulmoner hipertansiyon/ Pulmoner vasküler hastalık

TO-08

The effectivity of low dose thrombolytic therapy on vanishing right atrium floating thrombus in case of pulmonary embolism

Saif Hamad, Ziya Gökalp Bilgel

Başkent Universtiy Hospital, Adana

İntroduction: Pulmonary thromboembolism (PTE) is a common clinical condition and it can be mortal unless treated. Affecting millions, a condition that is frequently encountered. The incidence of venous thromboembolism is about 1.5 per 1000 person-years.1. Treatment in massive PTE is thrombolytic theraphy and the dose of t-PA is 100 mg in two hour. But the admistiration of t-PA and the initial dose submassive pulmonary embolism is stil controversial issues.2

Case report: 54 year old female had admitted to the emergency department with nausea and vomiting, shortness of breath and once mild hemoptysis. Echocardiography examination showed large floating thrombus inside the right atrium. CT confirmed PTE. The patient undergone thrombolityc therapy which treated with 5 mg per hour rt-PA for 10 hours a total of 50 mg. After checking echocardiographic examination at the end of 10 hours the thrombus was found to be completely absent.

Discussion: In case of pulmonary embolism within two days recanalization of the occluded vessel begins with the activation of the body's endogenous thrombolytic system and is substantially complete within 10-14 days. However, only half of patients develop a complete resolution and in the other half, thrombus organized and remains residual.3-4-5. Depriving patients who have relative contraindications from thrombolytic treatment may induce thrombus organization and nonresorbable organized small thrombi will result with reduction of exercise capacity in the future. In our case we administered low dose rt-PA in case of once mild hemoptysis. At the end of 8 hours with low-dose rt-PA, thrombus was completely disappeared from the right atrium.

Conclusion: The possibility of administration low-dose t-PA in submassive pulmonary embolism in relatively contraindicated cases. Might have a beter clinical outcome comparison with not to. Futher studies need to clarify the importance of low dose thrombolytic therapy in case of PTE.

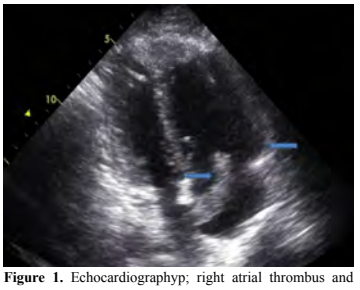


Figure 1. Echocardiographyp; right atrial thrombus and ventriculer dilatation.

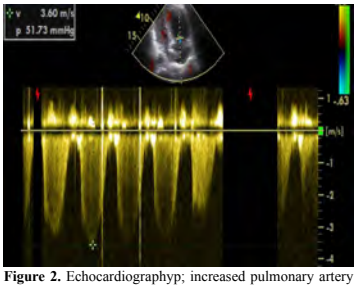


Figure 2. Echocardiographyp; increased pulmonary artery pressure.

TO-09

Successful management of methotrexate induced simultaneous thrombosis of the LMCA, LAD and RCA by systemic thrombolysis

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62-year-old male, recently diagnosed with rheumatoid arthritis treated with methotrexate for a week, was admitted to our emergency department with acute and crushing chest pain evolving since an hour. On ECG, ST segment elevation (STEMI) in anterior and inferior leads were noted. After the patient was treated with 300 mg of aspirin, 600 mg of clopidogrel, as well as a weight-adjusted (70 IU/kg) bolus of unfractionated heparin, coronary angiography was performed showing an ostial left main coronary artery (LMCA) thrombus with 90% obstruction (figure A), a total occlusion of the mid left anterior descending artery (LAD) (figure C,D) and a mid right coronary artery (RCA) thrombus with %80 obstruction (figure B). From this point on we had two possible strategies: an interventional strategy (stenting/aspiration with high risk of cerebral and peripheral embolism) and a pharmacological strategy (continuation of thrombolytic), eventually followed by the pharmacological strategy. Thus, based on the STEMI guideline of ESC; alteplase (rt-PA) 15 mg was injected intravenosus as a bolus dose and subsequently 50 mg alteplase was infused for 30 minutes and 35 mg was infused for 60 minutes. Repeated coronary angiography was performed 24 hours later, revealing that the large thrombus in ostial LMCA and mid potion of RCA was totaly dissolved (figüre E,F) but there was no flow in the distal LAD. Vascular toxicity and thrombotic effects has been reported with some antineoplastic agents. Thus, it is important for clinicians to be aware of the various rare, but potentially serious, adverse cardiac effects of these agents. In conclution, we present a case of STEMI caused by methotrexate induced simultaneous thrombosis of the left main, the left anterior descending artery and the right coronary artery was successfully managed by systemic thrombolysis. It should be kept in mind that similar cases involving LMCA thrombus can initially be managed by systemic thrombolysis.

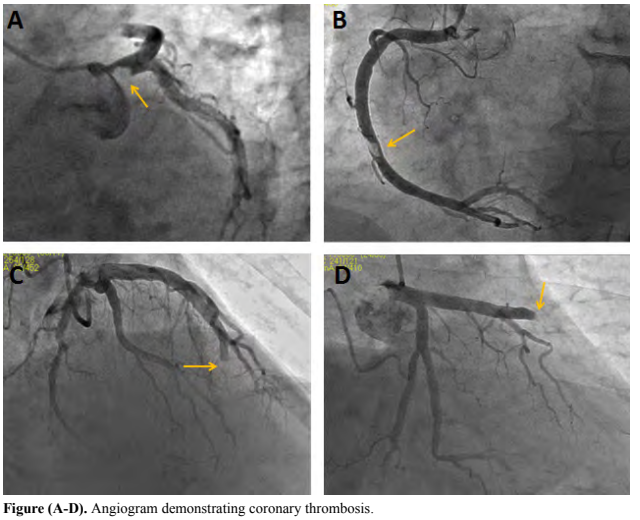

Figure (A-D). Angiogram demonstrating coronary thrombosis.

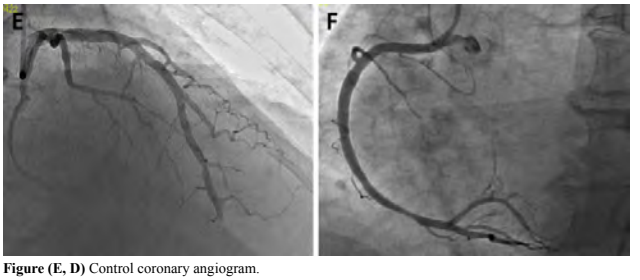

Figure (E, D) Control coronary angiogram.

TO-10

Successful Percutaneous treatment of simultaneous bilateral coronary artery occlusion during TAVI: first in literature

Hüseyin Dursun, Mohammed Abusharekh, Tuğçe Çöllüoğlu, Dayimi Kaya

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Coronary obstruction during transcatheter aortic-valve implantation (TAVI) can occur with an incidence of approximately 1%. There is no case report so far describing simultaneous occlusion of bilateral coronary artery ostium after TAVI. Herein, we present such a patient who underwent TAVI with Edwards Sapien XT valve. She was successfully treated with emergent percutaneous coronary intervention (PCI). A 75 year old female with New York Heart Association Class III dyspnea was referred to our institution for severe AS. Her past medical history revealed hypertension, diabetes mellitus, atrial fibrillation, and breast cancer. Transthoracic echocardiography showed an aortic valve area of 0.6 cm2 with a mean gradient of 52 mmHg and left ventricular ejection fraction of 50%. Coronary angiography revealed a 70% stenosis at circumflex (CX) artery. There was no other coronary lesion or peripheral artery stenosis. Her logistic EuroSCORE and STS scores were 15.05%, 1.64%. Since she had a history of radiotherapy to thorax, TAVI with transfemoral approach was decided by heart team. Multi-slice computerized tomography (CT) showed aortic annulus diameter of 23 mm and distance between aortic annulus to left coronary ostium of 12.2 and right coronary ostium of 15.8 mm. Aortic root was not severely calcified and there were no bulky calcified leaflet close to coronary ostium. Before TAVI a 4.0x18 mm sized bare metal stent was directly implanted to CX lesion. During TAVI aortic valve predilated with a 25 mm diameter balloon. Since the displacement of calcified leaflet towards left main was observed, a left coronary guiding catheter with a guidewire was placed into left coronary system before valve implantation. Then 26 mm. Edwards Sapien XT valve was implanted. Afterwards hemodynamic profile suddenly deteriorated. Angiography showed significant obstruction of left coronary ostium. 4.0x18 mm bare stent was implanted, followed by balloon post dilatation. The angiographic result was satisfactory, but the patient was still hypotensive and pacemaker dependent. So right coronary artery angiography was performed and revealed an ostial occlusion. A 4.0x20 mm sized bare stent was implanted with multiple post dilatations. The hemodynamics of the patient improved in a short time and she was extubated. During the in-hospital course no complication occurred and she was discharged home at 4th postoperative day. Obstruction of coronary arteries even bilateral coronary arteries can occur inevitably during TAVI. We demonstrated that successful PCI to both coronary arteries can be performed in such a case. To the best of our knowledge this is the first report in the literature.

TO-11

First PCSK-9 mutant in Turkey

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Introduction: Familial hypercholesterolaemia (FH) is a genetic disorder characterized with elevated serum LDL-cholesterol levels, tendon xanthomas and premature coronary heart disease (CHD). PCSK-9 gain of function mutations are known to be responsible for the development of FH.

Case: 60-year-old male patient was referred to our lipid clinic for persistently elevated LDL-cholesterol levels and premature atherosclerosis. He had had myocardial infarction at the age of 45 and underwent coronary bypass surgery. During the last 15 years, he had undergone several percutaneous revascularisation due to the restenosis. His familial medical history revealed that his sister had tendon xanthomas, meanwhile his daughters, big brother and nieces were already on statin theraphy. Physical examination showed arcus cornea existed for more than 20 years. Serum cholesterol levels were high despite aggressive antilipid therapy (80 mg/day atorvastatin). The maximum levels for total cholesterol and LDL cholesterol were 530 and 400 mg/dL, respectively The minimum levels for total and LDL cholesterol were 292 and 125 mg/dL, respectively. He was diagnosed as "Definite FH" for fulfilling Simon Broome, Dutch Lipid Clinic Network, and MEDPED criterion. In order to define 'FH' family and find new index cases, genetic analysis for single nucleotide polymorphism in PCSK 9 gene was done. Homozygous R496W mutation was detected. The family pedigree was drawn for better identification of the family (Figure 1).

Conclusion: Our case is the first declared PCSK 9 mutant in Turkey. FH caused by PCSK 9 mutant variant R496W, is characterized with extremely high LDL and total cholesterol levels, severe CHD, arcus cornea and severe family history for hypercholesterolaemia.

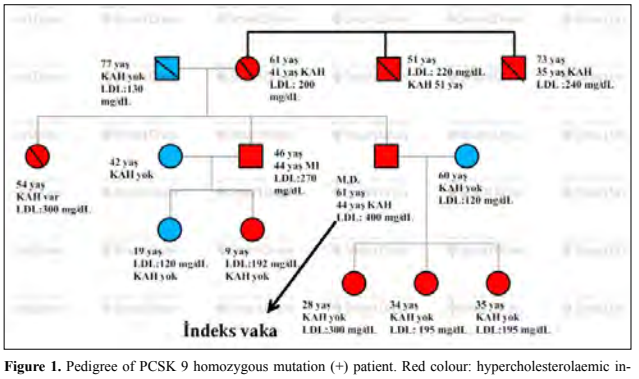

Figure 1. Pedigree of PCSK 9 homozygous mutation (+) patient. Red colour: hypercholesterolaemic individuals.

TO-12

Recurrent myocardial infarction after repetetive cannabis use: the pathophysiology of myocardial infarction after cannabis use

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

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A 28-year-old male patient admitted to the emergency department by virtue of chest pain that began 1.5 hours after cannabis use. He had no classical risk factors for atherosclerosis except smoking. His physical examination was unremarkable. Electrocar¬diogram (ECG) on admission denoted mild ST- elevations on inferior leads and V3 to V4, however, control ECG was unremarkable. Echocardiography demonstrated normal findings. Troponin level increased up to 50 ng/dl. The patient was given clopidogrel loading dose of 600 mg and aspirin 300 mg orally and he was taken to the cath-lab for coronary angiography revealing normal coronary arteries (Figure 1). After one month of discharge, the patient developed cardiac arrest and he was hospitalized to another hospital after recurrent cannabis use. After stabilisation of clinical status, he was transferred to our coronary care unit and remarkable ventricular dysfunction with an ejection fraction of 25% was demonstrated by echocardiography, hence, we decided to repeat the coronary angiography that demonstrated a large thrombus and co-existing spasm causing a total occlusion of the proximal left anterior descending artery (Figure 2). The exact mechanism of myocardial infarction after cannabis use remains controversial. There have been alternative hypotheses reported to explain the pathophysiology of MI after cannabis use. However, there is not a valid explanation of its mechanism. Our case is extremely rare and unique that recurrent myocardial infarction after one month due to repetetive cannabis use has not been reported in English literature before. Moreover, the first coronary angiography was completely normal, whereas vasospasm and thrombosis of LAD was significant in the second coronary angiogram that demonstrated the pathophysiology of myocardial infarction after cannabis use. Therefore, our case revealed that vasospasm and thrombosis even in normal coronary arteries are the main mechanisms of MI following cannabis use.

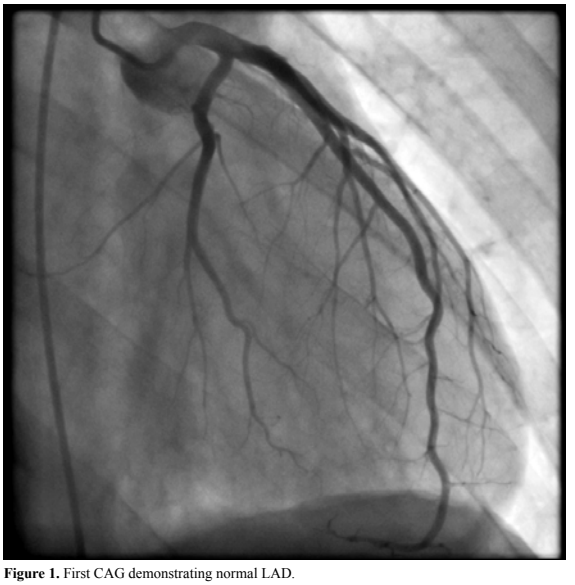

Figure 1. First CAG demonstrating normal LAD.

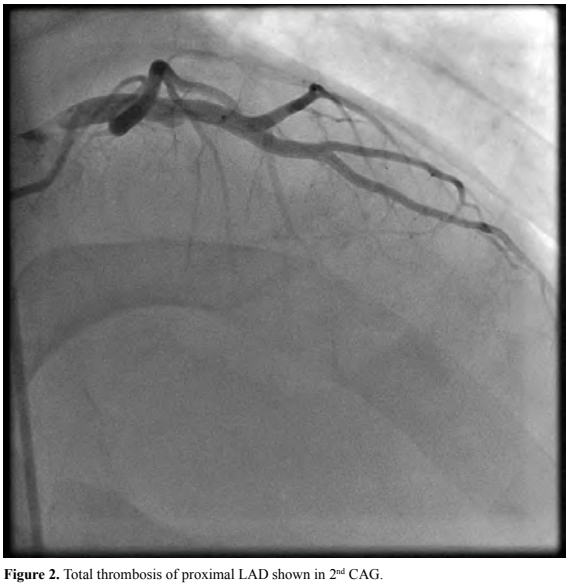

Figure 2. Total thrombosis of proximal LAD shown in 2nd CAG.

TO-13

Femoral girişim ile dejenere bioprotez trikuspit kapağa valv-in-valve transkateter trikuspit kapak implantasyonu

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Ankara Türkiye Yüksek İhtisas Hastanesi, Ankara

Transkateter aortik kapak implantasyonu ilk kez 2002 yılında uygulanmaya başlanmıştır. Günümüze kadar yaklaşık 20000 hastaya uygulanmıştır. Ciddi aort darlığı olan (mean grd> 40 mmHg) ve cerrahi riski yüksek olan (STS Sscore >10) hastalarda günümüzde standart tedavi haline geldi. Bioprotez kapaklar konjenital ve edinilmiş kardiyak hastalıklarda sıklıkla kullanılmaktadır. Fakat bu kapaklarda sıklıkla erken dejenerasyon ve kapak disfonksiyonu gözlenmekte ve bu hastalara re-operasyon gerekmektedir.TVIV prosedürü özellikle cerrahi riski yüksek, tekrar sternotomi yapılması zor, tamir şansı düşük olan hastalarda tercih edilmektedir. Günümüzde aortik pozisyonda sıklıkla kullanılan bu yöntem, trikuspit biyoprotez kapak dejenerasyonlarında da kullanılmaya başlanmıştır(yaklaşık 75 vaka). Özellikle dünyada biyoprotez kapak kullanımın arttığı, genel olarak ortalama yaşam süresinin uzadığı düşünülürse; TVIV prosedürlerinin gelecekte daha sık uygulanacağı ve rutin kullanıma gireceği düşünülmektedir.

Vaka: 49 y kadın hasta artan nefes darlığı nedeniyle kliniğimize başvurdu. 19 y akut eklem romatizması geçirmiş. 25 y (1991 yılında) ciddi AY ve ciddi MY nedeni ile aortik ve mitral metalik kapak replasmanı yapılmış. (aortik pozisyona 21 m Carbomedics, mitral pozisyona 27 mm Carbomedics kapak implante edilmiş). Ameliyat sırasında trikuspit kapak eksplore edilmiş ve 25 mm açıklık saptanıp müdahaleye gerek duyulmamış. 39 y (2005 yılında) artan nefes darlığı, ciddi TY saptanması üzerine re-operasyon yapılarak 27 no bioprotez kapak implante edilmiş. Hasta Ağustos 2015 te artan nefes darlığı nedeni ile kliniğimize başvurdu. Yapılan transtorasik ekokardiyografide (12/08/2015): Normal fonksiyone aortik ve mitral metalik kapaklar, Sol ventrikül çap ve sistolik fonksiyonları normal, Sağ boşluklar ileri derecede dilate, trikuspit bioprotez kapakta ciddi hareket kısıtlılığı, artmış gradient (25/15) ve minimal TY izlendi. Transözofageal ekokardiografide(21/08/2015): Biyolojik TVR de sıkı darlık, ileri derecede kalsifikasyon, kapak alanı <1 cm2 olarak izlendi. Hasta Kardiyoloji-KVC konseyinde triküspit kapak re-operasyonu açısından değerlendirildi. Hastanın daha önce 2 kez sternotomi öyküsü olması nedeni ile ameliyat açısından yüksek riskli olarak değerlendirildi ve hastaya Transkateter trikuspit valve-in-valve implantasyon kararı verildi. Hasta 11/08/2015 te Hybrid ameliyathane odasında işleme alındı. Genel anestezi altında entübe edilen hastaya femoral ven yolu ile 18f flex sheat takılarak girişim yolu açıldı. Öncelikli olarak trikuspit kapaktan guidewire ile geçilerek 21 no balon ile dejenere bioprotez kapağa balon dilatasyonu yapıldı. Ardından hastaya 26 no Edwards SAPIEN XT Transcatheter Heart Valve implante edildi. Başarılı implantasyon sağlandı. İşlem sonrası kontrol EKO görüntülerinde triküspit yetmezliğin olmadığı ve triküspit gradientin düştüğü görüldü. Hasta işlem sonrası ameliyathane de yaklaşık 30 dk genel anestezi sonrası extübe edildi. Girişim yerine sheat çekildikten ve kanama kontrolü sağlandıktan sonra sıkı tampon yapıldı.Hasta ardından koroner yoğun bakım ünitesinde takibe alındı. Takiplerinde nefes darlığı belirgin azalan, fonksiyonel kapasitesi düzelen hasta servise alındı. INR efektif hale gelen hasta işlem sonrası 6. gününde taburcu edildi. Bu vakada triküspit dejenere bioprotez kapağa başarılı valve-in-valve implantasyonu yapılmıştır.Özellikle cerrahi riski yüksek, reoperasyon gerektiren bu tip vakalarda uygulanabilecek yeni bir yöntemdir. Deneyimli merkezlerde doğru endikasyon ile uygulandığında mortalite ve morbidite riski düşük, hasta için konforlu, başarı oranı yüksek bir tedavi yöntemidir. Valve-in-valve tedavi yöntemi özellikle bioprotez kullanımının arttığı, transkateter bioprotez kapak kullanımının uygun hastalarda giderek standart tedavi haline geldiği günümüzde gelecek vadeden bir uygulamadır.

TO-14

Perkütan paravalvüler leak kapatma olgusu

Alev Kılıçkedik

Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, İstanbul

TO-15

Çok kısa aort kapağı-LMCA mesafeli olguya TAVI işlemi

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Amaç: Sol ana koroner (LMCA), aort kapağı mesafesi Transkateter Aort Kapak İmplantasyonu (TAVİ) işlemlerinde en önemli parametrelerden birisidir. Özellikle balonla açılabilen biyoprotez kapakların tekrar geri alınma şansı olmadığından mesafesi kısa olan hastalarda güvenlik teli ve balonu sol koroner sistemde bulundurulmalıdır.

Olgu Sunumu: İleri aort darlığı (AD) nedeniyle kliniğimize TAVİ yöntemiyle aortik biyoprotez implantasyonu yapılması amacıyla yönlendirilen olgu kalp takımı değerlendirmesinde yüksek cerrahi riskli kabul edildi. Hastanın STS skoru 11.8 ve logistic euroscore değeri 35.8 idi. Aortik gradiyentler 96/47 mmHg ve pulmoner arter basıncı 77 mmHg olarak saptandı. Hafif-orta mitral yetersizliği ve orta triküspit yetersizliği belirlendi. Hastanın koroner anjiyografisinde kritik olmayan lezyonlar saptandı. Transözofageal ekokardiyografi (TOE) değerlendirmesinde aortik anulus 20.7 mm olarak belirlendi. Hastanın tomografisinde 6.7 mm, LMCA ve aort kapak mesafesi olduğu görüldü. RCA ostiumunun aort kapağına mesafesi 8.8 mm olarak hesaplandı. Hastanın iliak ve femoral damarlarında orta derecede tortuozite olduğu gözlendi. Hastaya LMCA korumalı TAVİ operasyonu yapılması kararlaştırıldı. Hasta steril şartlarda anjiyografi laboratuarında işlem için hazırlandı. Sağ common femoral artere vasküler kapama cihazı yerleştirildi. Geçici kalp pili sol femoral venden, pigtail ise sol femoral arterden hastaya yerleştirildi. LMCA koruma işlemi için sağ radial artere vasküler kılıf yerleştirildi. Hastanın sağ femoral arterinden e-sheath aortaya gönderildi. AL1 kateter yardımıyla düz uçlu tel ile aort kapağı geçildi. AL1 kateter sol ventriküle (LV) ilerletildi. İçerisinden extra sertlikteki tel LV içerisine yerleştirildi. Hastanın sol ana koroneri JL4 kılavuz kateter ile kanüle edildi. 0.014 inch koroner girişim teli ve bunun üzerinden bir koroner balon LAD içerisine yerleştirildi. Ardından sol ana koronerdeki kılavuz kateter bir miktar geri çekildi. Tel ve balon bu şekilde bekletilerek aortaplasti ve aortik biyoprotez implantasyonu başarılı bir şekilse yapıldı. Herhangi bir koroner obstrüksiyon bulgusu saptanmadı.

Sonuç: Balon ile açılan aortik biyoprotez kapak implantasyonu için üretici firma tarafından 8.5 mm'lik LMCA ve aortik biyoprotez mesafesi önerilmektedir. Ancak LMCA koruması ile yeterince tecrübe sağlanınca daha kısa mesafelerde de TAVİ yöntemiyle balonla genişleyebilen aortik biyoprotezler implante edilebilir.

TO-16

Percutaneous transapical closure of cardiac apex with an ADO-II device after transapical transcatheter prosthetic mitral paravalvular leak closure

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Gülhane Military Medical Academy (GMMA) Ankara, Ankara

The transapical approach(TAA) for percutaneous interventions is performed in high-risk patients with peripheral vascular disease and when transseptal attempt failed. We report a case of a successful percutaneous closure of the apical access with a 4 mm ADO-II device after a standard transapical mitral paravalvular leak closure procedure without minithoracotomy. A 67 year old female was admitted to our department with severe dyspnea (NHYA classes II–III). She had hemolytic anemia and lactate dehydrogenase level was high. She had a history of mitral bioprosthesis valve replacement in 1997 and metallic mitral valve replacement in 2005. Transesophageal echocardiography (TEE) showed two severe mitral paravalvular leaks through a crescent-shaped hole located in the posterolateral portion of the mitral valve prosthesis according to the aorta. Firstly, we tried to close the leaks through the transseptal approach but we failed. We decided to try the TAA. Under general anesthesia, TEE and fluoroscopic guidance, transapical cannulation of the left ventricle from the fifth intercostal area and midclavicular region with a needle was performed and an 6 F sheath was inserted to the apical region. A Terumo hydrophilic wire was loaded on a right Judkins 6F catheter and passed through the defects. After the positioning of the catheter inside the left atrium (LA), the Terumo wire was replaced by an Amplatzer stiff wire. Then AL-1 guide the catheter to the LA. Through the guide catheter, a 10 mm x 5 mm Vascular Amplatzer plug was successfully deployed. There was no residual mitral paravalvular leak after deployment.No complications were observed (Figs. 1). After that, the placement of the myocardial occluder (4 mm ADO-II) was performed through the delivery system, with a very small amount of blood loss and an acceptable sealing of the apical tear. There was also no scar on chest wall. The patient was discharged 5 days in NYHA classes I–II. The hemolytic indices also improved.

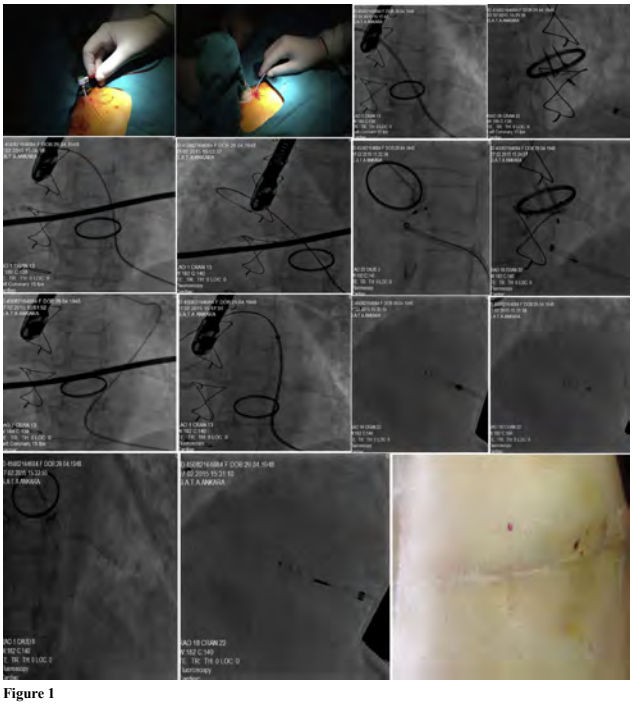

Figure 1

TO-17

Percutaneous aortic valve replacement and unprotected left main stenting in patients with multiple comorbidities; stepwise approach

Serdal Baştuğ[1], Cenk Sarı[1], Mustafa Duran[1], Abdullah Nabi Aslan[1], Ahmet Göktuğ Ertem[1], Hacı Ahmet Kasapkara[2], Hüseyin Ayhan[2], Serkan Sivri[1], Mehmet Burak Özen[1], Emine Bilen[1], Nihal Akar Bayram[2], Tahir Durmaz[2], Telat Keleş[2], Engin Bozkurt[2]

[1]*Atatürk Training and Research Hospital, Ankara*

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Introduction: Herein, we report a patient who underwent both left main artery stenting and transcatheter aortic valve replacement (TAVI) in whom remarkable improvement observed after these procedures.

Case Report: 68 year old female patient with a history of coronary artery disease (CAD),atrial fibrillation (AF), hypertension, ischemic SVO,severe aortic stenosis (AS) and congestive heart failure, was admitted to our hospital. She presented to the cardiology clinic with complaints of exercise induced angina and dsypnea. On presentation, her cardiovascular system revealed 4/6 intensity systolic murmur at aortic focus and bilateral rales in two third lower bases of lungs. Her neck veins were distended without carotid bruits. Abdominal examination revealed a palpable liver three centimeters below right costal margin, HJR+. There was a 3+ pitting edema of lower extremities to the kness. Her blood pressure was 110/70 mmHg, heart rate 110/min and oxygen saturation 92% on room air. Her electrocardiogram (ECG) showed AF 98/min beats per minute. Echocardiography examination revealed depressed ventricular function (EF %15-20) severe aortic stenosis (mean aortic gradient: 41 mm/hg, AVA: 0.41 cm^2) and Systolic pulmonary artery pressure (sPAP) was 65 mm/hg. After the symtomatic relief, we performed coronary angiography (CAG) for further evaluation. CAG revealed crucial occlusions both in ostium of left anterior descending (LAD) and circumflex arteries with distal left main (LM) involvement. Later, we discussed her condition with heart team. The STS score of the patients was 9.7, mean logistic EuroSCORE was 21.7%. Due to her prohibitive surgical risk, a stepwise approach with TAVI and percutaneous coronary intervention was planned. Firstly, we deployed a 2.5x28 mm in size drug eluting stent to LAD mid segment, afterwards 3.0x23 and 2.75x23 mm in size drug eluting stents extended to LAD ostial and circumflex artery ostial respectively. During her follow up contrast induced nephropathy occured and underwent temporary renal dialysis. Despite of all our attempts, she didn't respond medical therapies and underwent urgent TAVI. 26 mm Edwards SAPİEN XT valve deployed without any complication. After procedure urine extraction increased and vital signs returned to normal values. Two days after procedure she was transferred to cardiology clinic. Her temporary renal dialysis catheter was removed and control echocardiography showed functional bioprosthesis in aortic position with mean pressure gradient 7 mm/hg, No paravalvuler leak observed and ejection fraction was calculated as 25%. She was discharged from the hospital eleven days after first admission.

Conclusion: Combined TAVI and LM stenting is a viable option in patients who are deemed to be poor surgical candidates due to multiple comorbidites. While AVR and coronary artery bypass grafting remain the superoption, it is reasonable to offer these high-risk patients a combined percutaneous procedure for symptomatic relief.

PO-001

Dasatinib ile ilişkili pulmoner arteriyel hipertansiyon

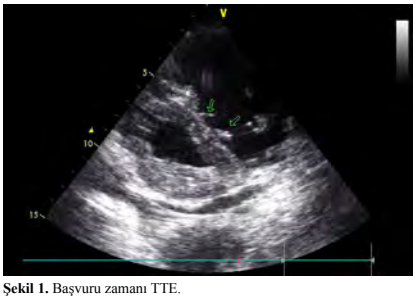
Murat Meriç, Mustafa Yenerçağ, Korhan Soylu, Serkan Yüksel, Metin Çoksevim

Ondokuz Mayıs Üniversitesi, Samsun

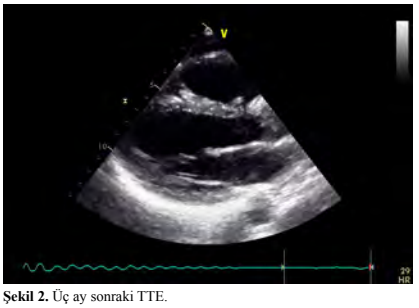
Giriş: Pulmoner arteriyel hipertansiyon (PAH) farklı etiyolojik nedenlere bağlı ortaya çıkan, pulmoner vasküler direncin yükselmesi durumudur. Dasatinib, imatinib ve nilotinib tirozin kinaz inhibitörüdür (TKİ) ve kronik myelositik lösemide (KML) kullanılmaktadır. Biz KML tedavisinde TKİ kullanımına bağlı olarak PAH gelişen ve ambrisentan tedavisi verilen olguyu sunduk.

Olgu Sunumu: 33 yaşında erkek hasta,nefes darlığı ve bacaklarda şişlik nedeniyle kliniğimize başvurdu. 3 yıldır KML tanısı ile takip edilen hasta yaklaşık üçer ay imatinib ve nilotinib tedavisi almış, myopati gelişmesi üzerine 15 aydır dasatinib tedavisi alıyormuş. Ekokardiyografisinde; sol kalp fonksiyonları normal, interventriküler septumda düzleşme (Şekil 1), sağ ventrikül bazal çapı 42 mm, sağ atriyal alan 21 cm^2 saptandı. TAPSE 15 mm, sPAB 120 mmHg ölçüldü. Hastanın 6 dk yürüme testi 75 m idi. Yapılan sağ kalp kateterizasyonunda ortalama PAB 49 mmHg, PKUB 5 mmHg, kardiyak debi 4 L/dk, PVR 21 wood ölçüldü. İloprost ile yapılan vazoreaktivite testi negatif idi. Fonksiyonel kapasitesi NYHA sınıf IV olan hastanın dasatinib tedavisi kesildi, ilaca bağlı PAH tanısıyla diüretik ve 1 hafta iv. iloprost tedavisi verildi. Takiplerimizde 6 dk yürüme tesi 275 m yükselen ve ödemleri gerileyen hastaya oral ambrisentan ve diüretik tedavi ile taburcu edildi. 3 ay sonra fonksiyonel kapasitesi NYHA sınıf II, 6 dK yürüme testi 384 metre, yapılan ekokardiyografisinde; Sağ ventrikül bazal çapı 35 mm, sağ atriyal alan 18 cm^2, TAPSE 19 mm, sPAB 80 mmHg ölçüldü (Şekil 2). Ambrisentan tedavisi altında takibimiz devam etmektedir.

Tartışma: KML tedavisinde TKİ 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. Dasatinib başta olmak üzere TKİ kullanılırken PAH yapabileceği akılda tutulmalı, ekokardiyografi ile kontrol edilmelidir.



Şekil 1. Başvuru zamanı TTE.



Şekil 2. Üç ay sonraki TTE.

PO-002

A rare cause of pulmonary arterial hypertension: atypical poems syndrome

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POEMS syndrome, with pulmonary hypertension (PH) is a rare clonal plasma cell disease which is associated with polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy and skin changes. We report a case of atypical POEMS who intially presented with severe pulmonary arterial hypertension resistant to therapy. Case: 44-year-old male patient who had a diagnosis of severe idiopathic PH was in bosentan therapy for last 3 months. Due to poor clinical response with bosentan additional hospitalization requiring and static 6-minute walk distance this was increased to combination therapy with iloprost and sildenafil. At the follow up examination splenomegaly with nonsignificant mediastinal lymphadenopathy and monoclonal gammopathy was found. Normocellular lambda light chains detected in bone marrow plazmastozis showing dominance was detected in bone marrow aspiration biopsy. 3 months after then methylprednisolone theraphy was started pulmonary artery pressure reduced mildly. Two months later, skin changes, muscle weakness uncontrolled systemic arterial hypertension, peripheral edema, sclerosing bone radiographs, polyneuropathy, organomegaly, subclinical hypothyroidism was appeared clearly. In the follow up period cardiac cirrhosis developed in 2 months followed by sudden cardiac death of patients. Discussion: Most patients with PH and organomegaly have overlooked diagnosis. POEMS syndrome is rare and usually diagnosed based on clinical and laboratory support with defined criteria. Since all of the diagnostic criteria emerge gradually and some of the present findings can be seen as associated with only PH delay in diagnosis may be experiencing. In case with PH can not be determined of PH etiology and in patients unresponsive to standard therapy, clinical course of time added to be monitored closely for new pathological findings are important in determining the secondary causes. POEMS syndrome patients with corticosteroids, chemotherapy or stem cell support is expected to decrease pH with high dose chemotherapy. The clear benefit of vasodilator therapy in these patients was not shown.

PO-003

Isolated partial anomalous pulmonary venous return; a rare cause of pulmonary hypertension in adults

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57 -year-old female patient who was followed as primary pulmonary hypertension about 7 years, admitted with increasing of shortness of breath and fatigue. On physical examination jugular venous pressure was normal. There wasn't any audible cardiac murmurs. ECG was in sinus rhythm with incomplete right bundle branch block. Transthoracic echocardiography (TTE) confirmed moderate right ventricle and right atrium enlargement with increased pulmonary artery pressure. (Fig. 1) Transesophageal echocardiography showed that superior vena cava and pulmonary artery diameter is increased without presence of any other left to right shunts like atrial septum defect (ASD) or ventricular septal defect (VSD). Three pulmonary veins were opening to the left atrium but the other pulmonary vein was not shown. (Fig. 2, Video 1-2-3) There wasn't any significant stenosis, anomalous or fistula in coronary angiography. (Fig. 3-4) Systolic pulmonary artery pressure was measured as 55 mm Hg at right heart catheterization. Oxygen saturation levels from right atrium to pulmonary artery were higher than normal and speed -up phenomenon was found between superior vena cava and right atrium. (O2 saturation levels: VCS:67%, RA: 84%, RV:84%, PA:86%). Pulmonary vascular resistance was 2 Wood units, QP/QS was calculated as 2.91. Vasoreactivity test was negative. We speculated that right superior pulmonary vein could be opened to the superior vena cava because of speed up phenomenon and enlargement of VCS. Cardiac MRI was performed to confirm the diagnosis and rule out sinus venosus ASD. It showed that right superior pulmonary vein was opening to superior vena cava. ASD wasn't accompanying. (Fig. 5) Patient was scheduled for surgery; because, QP/QS was higher and pulmonary vascular resistance was normal. Right superior pulmonary vein was anastomosed to the left atrium.

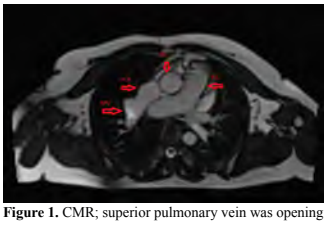

Figure 1. CMR; superior pulmonary vein was opening to svc.

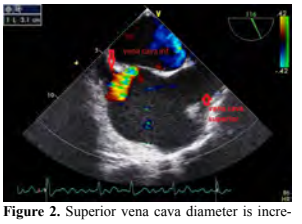

Figure 2. Superior vena cava diameter is increased.

PO-004

A rare cause of acute massive pulmonary embolism: thrombi-in-transit

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71 year-old female patient was admitted to emergency unit with severe dyspnea and chest pain for two hours. The medical history of the patient was asthma for ten years and recently diagnosed atrial fibrillation (AF). The patient was on medical treatment with bronchodilator therapy for asthma. She has not been receiving anticoagulant therapy for AF. Physical examination was normal except for tachypnea and systolic murmur on the left lower sternal border. Her heart rate was 110 beats per minute and her blood pressure was 90/55 mmHg. Arterial blood gas (ABG) showed hypocarbia (PaCO2: 30 mmHg) and hypoxia (PaO2: 75 mmHg). Pulmonary computed tomography angiography showed massive PE extending from main pulmonary trunk to right pulmonary artery. Transthoracic echocardiography showed enlargement of the right heart, increased systolic pulmonary arterial pressure (sPAB: 75 mmHg), right ventricular dysfunction (tricuspid annular plane systolic excursion: 11 mm), thrombi-in-transit in the right atrium and right ventricle and a huge thrombi particle attaching to tricuspid valves resulting in severe tricuspid regurgitation due to coaptation defect (Figure 1, 2). After diagnosis of acute massive PE, patient was transferred to coronary care unit for thrombolytic therapy. Tissue plasminogen activator (tPA) was administered at the rate of 50 mg/hour (total 100 mg) and then continued with unfractionated heparin for 24 hour (aPTT being above 2 times the normal value). The patient's complaints were dramatically recovered after thrombolytic therapy. Her blood pressure was 125/85 mmHg and peripheral oxygen saturation was 92% at room air. Low molecular weight heparin was continued for anticoagulant therapy (Enoxaparin 2x0.6 ml). After two days later, transthoracic echocardiography revealed no thrombus in right heart. The patient was discharged on the tenth day of admission with optimal warfarin therapy.

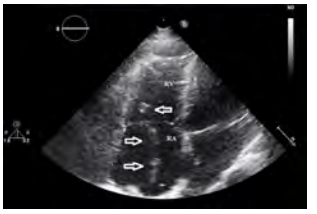

Figure 1. Thrombi-in-transit from the RA to the RV.

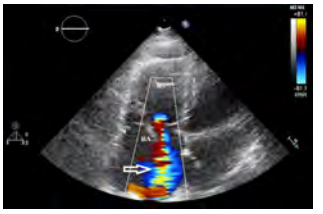

Figure 2. A huge thrombi attaching to tricuspid valve.

PO-005

Free- floating thrombus in the right ventricle causing pulmonary embolism

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A 78-year-old man admitted to the hospital with suddenly occured dyspnea and orthopnea. The patient had previous history of hypertension and chronic renal failure. He had no past history of hypercoagulability related risk factors such as immobility or malignancy. Clinical examination revealed tachycardia (a pulse rate of 140/min), tachypnea (a breath rate of 42/min), a blood pressure of 90/50 mmHg and partial oxygen saturation of 78% on room air. The electrocardiography (ECG) demonstrated sinus tachycardia. Routine laboratory examination showed high troponin I and D-dimer levels with impaired renal function tests (urea: creatinine (2.4 mg/dL). The transthoracic echocardiography revealed dilated right chambers with a highly mobile, irregular shaped mass in the inferior vena cava, which is floating toward right ventricle inflow. We decided to perform emergency surgical removal of the mass as a first therapeutic option. However, the patient was considered to be unsuitable candidate for emergency surgery because of multible comorbidities. Thrombolytic therapy with recombinant tissue plasminogen activator (Actilyse) was immediately administered by intravenous infusion of 100 mg over 2 hours, followed by 800 units/hour unfarctional heparin infusion. Within a few minutes after the thrombolytic therapy, his clinical condition was gradually improved. The control transthoracic echocardiography showed complete disappearance of the mass.

Figure 1. Beforethrombolytictherapy; demonstrating right ventrıcle thrombus.

Figure 2. After thrombolytic therap; disapperance of right ventricle thrombus.

PO-006

Idiopathic pulmonary artery aneurysm

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It is unusual to encounter pulmonary vascular diseases, pulmonary artery aneurysm is found 1/14000 in autopsy series. Description of pulmonary artery aneurysms holded up to 1700s. Idiopathic pulmonary artery aneurysm is more rare when all reasons thought. Most of the patients with pulmonary aneurysm are asymptomatic. Here we present 60 years old man patient admitted to the emergency department with chest pain. His physical examination was totally normal except bilateral ronchi was heard in pulmonary auscultation. Apparent pulmonary artery was detected in chest X-ray. In transthoracic echocardiography there was no functional abnormality in the right ventricle, nevertheless severe expansion in the pulmonary arteries were seen. The systolic pulmonary artery pressure in the tricuspid valve was 30 mmhg. The patient was investigated for systemic disease or vasculitis, all the biochemical tests were negative even patergy test was used to eliminate Behçet disease. Pulmonary arterial computerized tomography was used to understand if there was any thrombus. In the multisection pulmonary tomography the main pulmonary artery was 44mm. He was taken to close follow-up from outpatient clinic to watch the pulmonary artery closely. Operation will be thought in the folow-up if the expansion of the pulmonary arteries continue.

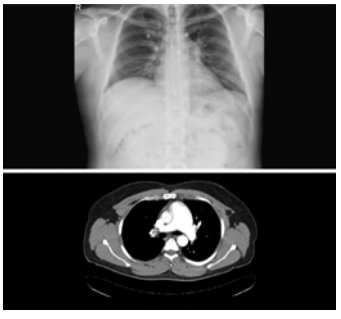

Figure 1. Pulmonary apparent in chest X-ray, pulmonary CT.

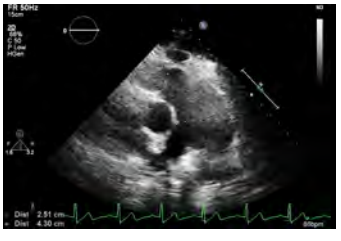

Figure 2. Parasternal short axis view in TTE.

PO-007

We perform vasoreactivity test in pulmonary hypertension, but reproducibility? A case presentation

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Sarcoidosis is a chronic, granulomatous disease that it can affect all organs. Pulmonary hypertension (PH) is one of the most important complications of sarcoidosis and indicates poor prognosis. PH is determined by right cardiac catheterization and mean pulmonary artery pressure is ≥25 mmHg at rest. Vasoreactivity test is recommended to this patients, to determine vasodilator drugs to response of pulmonary vascular bed. Calcium channel blockers are used in patients with a positive test vasoreactivity. Case report: A 32 years old women who had previously diagnosed pulmonary sarcoidosis and PH, was admitted to department of cardiology of our hospital because of fatigue, weakness, increased dyspnea. She was 11 years old with a diagnosis of pulmonary hypertension. She consulted to other hospital seven months due to advanced dyspnea. Echocardiography was detected elevated systolic pulmonary artery pressure and pulmonary hypertension diagnosis was confirmed by right cardiac catheterization. Vasoreaktivit test was performed by using inhaled iloprost and the test was found positive. Thus, She took calcium channel blocker. However, dyspnea did not decrease and fatigue, weakness occurred following this treatment. So she was admitted to our clinic. There was no significant a disease exception pulmonary sarcoidosis. When she came to our clinic, diltiazam 600 mg / day, methotrexate 15 mg / week and oxygen was using. Physical examination showed that blood pressure of 100/68 mm Hg, pulse rate of 98/min, and temperature of 37.0°C. There were decreased breath sounds, prolonged expration and 2/6 systolic murmur in the tricuspid focus. Electrocardiography demonstrated a normal sinus rhythm pattern, p pulmonale, inverted T waves at D3, aVF. Echocardiography showed severe pulmonary hypertension with dilatation of the right heart chambers, normal ejection fraction (65%), severe tricuspid regurgitation. The systolic pulmonary arterial pressure was 50-55 mm Hg. Tricuspid annular plane systolic excursion (TAPSE) was 2.3 cm. 6-minute walking distance (6MWD) was 340 meter. Diltiazem therapy was terminated because her complaints increased following this drug. Right heart catheterization and vasoreactivity test was repeated. Mean pulmonary artery pressure and pulmonary capillary wedge pressure were 38 mmHg and 7 mmHg, respectively. Vasoreaktivity test was performed with adenosine, the drug dose could be increased 200 µg/ kg / min due to hypersensitivity reaction. Mean pulmonary artery pressure was 37 mmHg following 200 µg/ kg/min adenosine. Bosentan was started because vasoreactivity test was negative. Her semptoms have tended to improve and 6MWD has 375 meter 12 weeks later. Conclussion: Patients who are vasoreactivity test positive and treated with calcium channel blockers should be closely monitored. High doses of calcium channel blockers does not seem to be well tolerated by patients. Bosentan looks like a good option for treatment of Sarcoidosis-associated PH.

PO-008

Saddle pulmoner emboli

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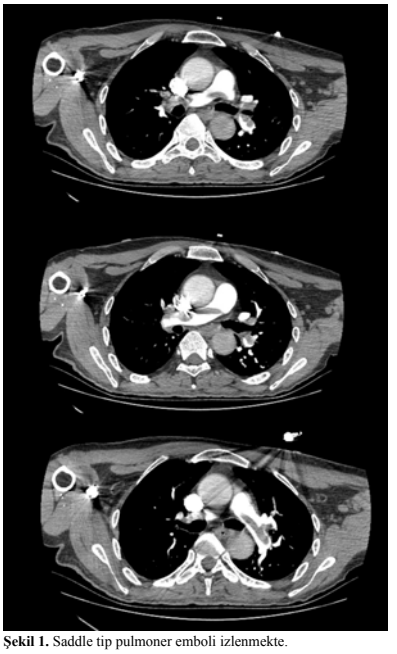
55 yaşında erkek hasta göğüs ağrısı ile acil servise getirilmişti. Hastanın immobilazyon öyküsü mevcuttu. Kan basıncı 110/70 mmHg, nabız ise 105 atım/dk idi. Elektrokardiyografisi; sinüs taşikardisi ve komplet olmayan sağ dal bloğu mevcuttu. Hastaya ait kan tetkiklerinde troponin + olması üzerine hasta değerlendirildi. Transtorasik ekokardiyografik değerlendirmede sol ventrikül sistolik fonksiyonlar ve segmenter duvar hareket kusuru izlenmedi. Sağ boşluklar dilate ve pulmoner arter basıncının artmış (50 mmHg) olması üzerine pulmoner bilgisayarlı tomografik anjiografi çekildi ve saddle tipte pulmoner emboli saptandı.



Şekil 1. Saddle tip pulmoner emboli izlenmekte.

PO-009

The coronary artery dissection due to falling from a truck in the left anterior descending artery leads myocardial infarction

Atila İyisoy, Cengiz Öztürk, Turgay Çelik, Zekeriya Arslan, Şevket Balta, Mustafa Demir, Ali Osman Yıldırım, Murat Ünlü

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A 54 year old man who presented with acute chest pain for 6 h was after falling from a truck admitted to our coronary care unit. He had no previous trauma or emotional stress. There was significant elevation of the troponin T level. There was ST segment elevation on precordial derivations on electrocardiogram. The patient was diagnosed to have a anterolateral ST-elevation myocardial infarction (STEMI). The patient was transferred to the catheter laboratory. Dual anti-platelet therapy (acetylsalicylic acid and ticagrelor) was initiated. Invasive coronary angiography showed a totally occlusion of the left anterior descending artery (LAD). At first intervention the lesion was not passed with a 0.014 inches wire. After 6 hours the lesion was passed but an adequate flow was not obtained. There was severe dissection on proximal LAD. In order to avoid extending the dissected segment, intravascular ultrasonography (IVUS) was not performed. Heparin and tirofiban infusions were started for 24 hours. LVEF was 20% on transthoracic echocardiography. Medical therapy was applied. After 7 days, coronary angiography was done again with IVUS. The flow was better but a intramural hematoma was observed. A serial balloon inflations were performed and 3.5x12 mm metal stent was implanted to the proximal LAD. The flow and dissection were relatively good. After 6 months, the flow and anatomies of the the proximal LAD was so good but distal was not. The dissected and stenotic segment were also seen at the same levels of the vessel. LVEF was increased to the 45% and the patient was referred to the surgery. The patient had no severe atherosclerotic lesions in the remainder of the arteriel system, left circumflex and the right coronary arteries. The patient had no atherosclerotic risk factors with only smoking. He had history of the falling from a truck. In this patient,totally occlusion of the LAD was related a coronary dissection due to the trauma.

Figure 1

PO-010

Successful thrombolytic therapy for ST-elevation acute myocardial infarction in a patient with immune thrombocytopenic purpura

Erkan Köklü, Görkem Kuş, İsa Öner Yüksel, Selçuk Küçükseymen, Murat Esin, Göksel Cağırcı, Şakir Arslan

Antalya Education and Research Hospital, Antalya

Immune thrombocytopenic purpura (ITP) is an autoimmune disorder presenting with thrombocytopenia. A 44-years old male patient admitted to non-primary percutaneus coronary intervention capable center, 2 hours after onset of pressure type chest pain localized to the retrosternal region. Electrocardiography (ECG) showed ST segment elevation in leads V1-V3 (Figure 1) and the patient was diagnosed with STEMI. Distance between the center patient had referred to and the nearest primary percutaneus coronary intervention capable center would take more than 120 minutes. Thrombolytic therapy was given intravenously to the patient who had permenant chest pain and ST segment elevation on surface ECG. Laboratory results were as follows: hemoglobin 13.4 g/dL, thrombocyte count 46.000 µI, troponin I 25 ng/mL (normal value <0.06 ng/mL). Results of standard coagulation tests were in normal limits. Protein C or protein S deficiency was not identified. Antiphospholipid antibodies and homocysteinemia were not detected. Echocardiography did not show any pathological finding except for mild hypokinesis of the anterior wall. The patient had been diagnosed with ITP before and he was not receiving any medical therapy for ITP as he was in remission. The patient was evaluated together with hematology unit. Coronary angiography of the patient was performed 12 hours after onset of chest pain.Coronary angiography showed a plaque causing 30% stenosis in the left descending coronary artery; whereas the circumflex artery and right coronary arteries were normal (Figure 2). Thrombocyte count was monitored daily and the patient was discharged after 5 days of hospitalization. Primary percutaneous coronary intervention should be the first choice of treatment in ITP patients representing with ST-elevation myocardial infarction. However, thrombolytic therapy as demonstrated in the present case may also be given when primary percutaneous coronary intervention is not feasible.

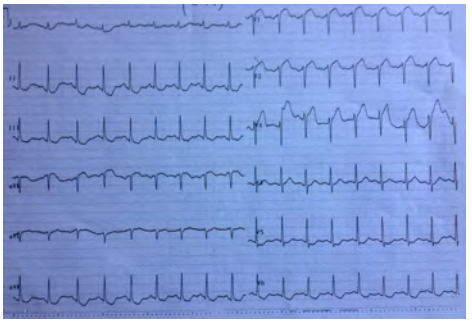

Figure 1. V1-V3 ST segment elevation before TPA.

Koroner arter hastalığı / Akut koroner sendrom

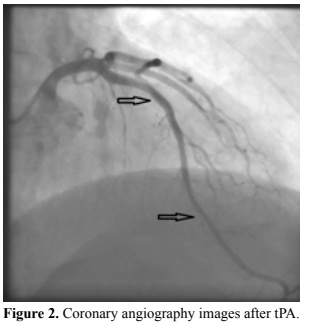

Figure 2. Coronary angiography images after tPA.

PO-011

An handicap in takotsubo cardiomyopathy: dynamic outflow obstruction

Muhammed Keskin, Veysel Ozan Tanık, Ayça Gümüşdağ, Mert İlker Hayıroğlu, Edibe Betül Börklü, Özlem Yıldırımtürk

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Takotsubo cardiomyopathy (TCM) is an unusual form of acute cardiomyopathy showing left ventricular apical ballooning. In patients with hypotension and cardiogenic shock, dynamical left ventricular outflow obstruction (LVOT) should be kept in mind. A 77-years-old female without known history of coronary artery disease was admitted to emergencey department with complaining of rapid onset of severe chest pain after an argument with her family. The character of the pain was retrosternal squeezing sensation and radiating to jaw and left shoulder. In medical history, she had not a traditional risk factor for coronary artery disease. Her electrocardiogram showed ST segment elevations in leads V1-6, II, III, aVF (Figure 1) that was consistent with acute ischemia. Due to severe hypotension (73/45 mm Hg via intra-arterial measure) dopamine infusion was started considering of cardiogenic shock and noradrenaline infusion was added because of resistant hypotension. Her coronary angiogram revealed patent coronary arteries with minor irregularities and TIMI-III flow. Echocardiography showed hypokinesis of apical mid-ventricular walls and a left ventricular ejection fraction of 35% that was consistent with TCM (Video 1). Systolic anterior motion of the mitral valve and moderate mitral regurgitation were obtained. The echo-derived peak and mean LVOT gradient was 137/61 mmHg respectively (Figure 2). After obtaining of dynamical LVOT obstruction, inotropic agents were stopped and 500 cc bolus of %0.9 NaCI and 5 mg bolus of intravenous metoprolol treatment were given by reason of deterioration of hemodynamic status. After a while the patient's tachycardia and hypotension was resolved. Oral metoprolol treatment was initiated. The patient's subsequent course was uneventful. A repeat echocardiography after 3 days showed no LVOT gradient but apical hypokinesis.

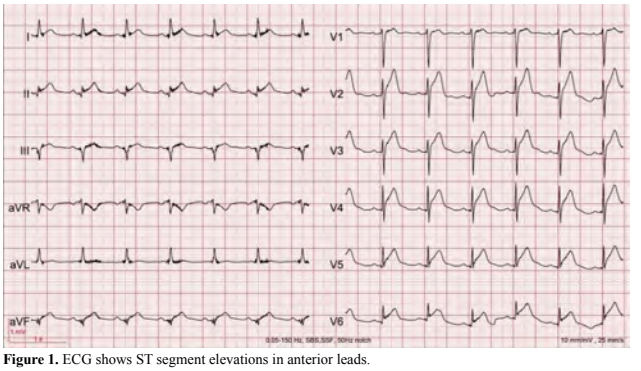

Figure 1. ECG shows ST segment elevations in anterior leads.

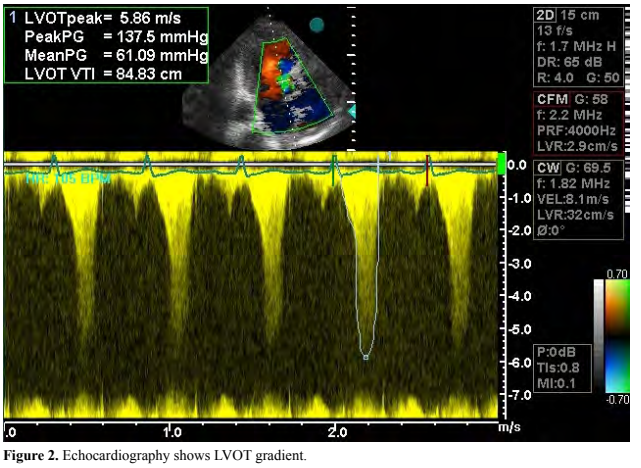

Figure 2. Echocardiography shows LVOT gradient.

PO-012

Huge myocardial bridging, hypertrophic cardiomyopathy and severe mitral regurgitation in a patient presenting with acute coronary syndrome

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Hypertrophic Cardiomyopathy (HCM) is characterized by a thickened but nondilated left ventricle in the absence of other cardiac or systemic conditions (e.g., aortic valve stenosis, systemic hypertension, and some expressions of physiologic athlete's heart) capable of producing the magnitude of left ventricular (LV) hypertrophy evident. It causes symptoms such as typical or atypical angina pectoris, syncope and heart failure symptoms like dyspnea. Myocardial bridging is defined as a segment of a major epicardial coronary artery, the 'tunneled artery,' that goes intramullary through the myocardium beneath the muscle bridge. This case is about rare coexitence of HCM, muscular bridge and severe mitral regurgitation. A 38-year-old caucassian man brought to the emergency department due to aggreviation of his chest pain. He was diagnosed with unstable angina and coronary angiography revealed long and critical myocardial bridge causing 90-99% stenosis in the proximal segment of left anterior descending artery. Echocardiographic assesment revealed hypertrophic cardiomyopathy causing severe mitral regurgitation. Left hearth catheterisation and ventriculography also confirmed the diagnosis. After counseling with cardiovascular surgeants septal myectomy, mitral valve replacement and coronary artery by-pass grafting surgery is planed. The presence of myocardial bridge and hypertrophic cardiomyopathy is rarely defined. Myocardial bridges causing ischemia is also rare. Structural changes in HCM (more extensive distribution of hypertrophy) may be delayed until later in midlife. In adults with hypertrophic cardiomyopathy and myocardial bridge, an increase in the ischemia due to stenosis can be related to the increase in left ventricular hypertrophy in midlife.

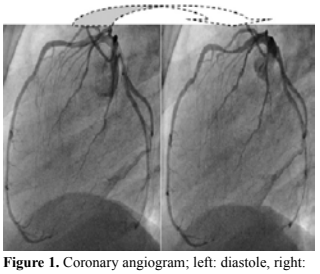

Figure 1. Coronary angiogram; left: diastole, right: systole, myocardial bridge.

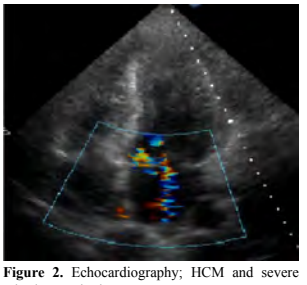

Figure 2. Echocardiography; HCM and severe mitral regurgitation.

Koroner arter hastalığı / Akut koroner sendrom

PO-014

Nadir birliktelik, koroner arterlerde dev anevrizmalar ve aort anevrizması

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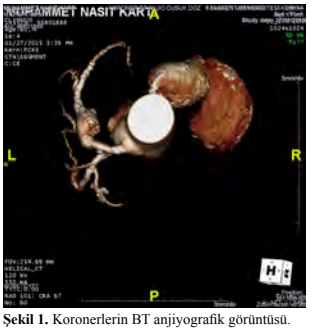
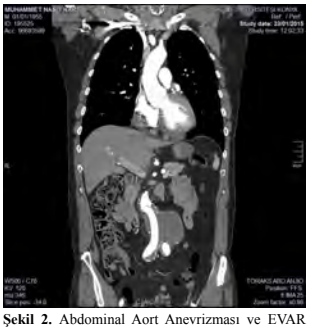
Giriş: Dev koroner arter anevrizması(KAA) tanımı çapı 8 mm üzerindeki koroner arterler için kullanılmaktadır. Yaklaşık insidansları %0,02-0,2 arasındadır. KAA'larının büyük kısmı asemptomatiktir ancak miyokard infarktüsü, ani ölüm, perikardiyal tamponad, fistülizasyon ve bası bulgularına yol açabilirler. Abdominal aort anevrizmasıyla birliktelikleri ise literatürde mevcut değildir.

Vaka Takdimi: Altmış yaşında erkek hastanın akut miyokard infarktüsü nedeniyle yapılan koroner anjiyografisinde, sol ön inen arterin(LAD) tromboze tam tıkalı olduğu bölgede damarla birlikte hareket eden yaklaşık 25 mm çaplı, kenarları kalsifik bir yapı izlendi. Sağ koroner arterde de (RCA) lümen çapının 10 mm olduğu anevrizmatik genişleme ve kenarı kalsifik, 35 mm ölçülen bir yapı izlendi. Kalsifik yapıların tanımlanması için bilgisayarlı tomografi (BT) ile anjiyografi çekildi. Çekilen BT 'de rastlantısal olarak abdominal aortada da, renal arterlerin hemen altından başlayan 7,1 cm'lik sakküler anevrizma kesesi saptandı (Şekil 1, 2). Tedavi için değerlendirmesinde, RCA'nın distale kadar anevrizmatik olması sebebiyle inoperabıl olduğu, LAD'nin de rüptür ihtimalinin düşük olduğu düşünülerek cerrahi müdahale yerine BT ile seri takibin uygun olacağı kararı alındı. Abdominal aort anevrizması için endovasküler aort tamiri (EVAR) yapıldı. EVAR sonrası BT ile stabil olarak takip edilen hasta 3. yılında ani kardiyak ölüm ile kaybedildi.

Tartışma: Dev KAA'lar en sık sağ koroner arterde lokalize olurken azalan sıklıkla LAD ve CX'de gözlenirler. Konjenital, ateroskleroz, ilaç salınımlı stent kullanımı, göğüs travmaları, bağ dokusu hastalıkları, Kawasaki hastalığı en sık sebeblerdir. Vaka serilerinde beş yıllık yaşam beklentisi %71 olarak rapor edilmektedir. Tedavide medikal takip, kaplı stentler ve cerrahi gibi seçenekler vardır. Ancak hangi anevrizma çapına veya şekline hangi tedavi yapılacağı net değildir.



Şekil 1. Koronerlerin BT anjiyografik görüntüsü.



Şekil 2. Abdominal Aort Anevrizması ve EVAR stenti.

PO-013

Acute myocardial infarction in a young adult due to massive coronary thrombus burden - do not underestimate the power of genes

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Case presentation: A 31-years-old male patient was seen at the outpatient clinic who presented with the complaints of severe exertional dyspnea (NYHA III) and atypical angina. He had had an acute anterior ST-segment elevation myocardial infarction (STEMI) 2 years earlier. He had undergone emergency coronary angiography which had revealed a non-critical lesion in the LAD, and a massive thrombus extending from the ostium to the mid portion of the LAD, and a stent was implanted. Despite the intervention, he had low left ventricular ejection fraction (LVEF=32%). He was on a standard regimen with ASA 100 mg/day, atorvastatin 10 mg/day, carvedilol 12.5 mg/day, and perindopiril 5 mg/day. A repeat cardiac evaluation was performed: ECG was consistent with sinus rhythm and evidence of prior anterior wall MI. Echocardiography revealed severe wall motion abnormality of the septum, anterior wall, and the apex, and a LVEF of 33%. Repeat coronary angiography performed due to anginal complaints revealed a patent stent but slow flow in the LAD and otherwise normal coronary arteries. Dyspnea subsided after the addition of i.v. furosemide. İvabradine was also added, which further improved the NYHA class during follow-up. We thought that a genetic analysis for thrombogenic mutations might also be relevant, considering the STEMI at at a young age, due to a massive coronary thrombus. An initial work-up including the levels of protein C, protein S, and anti-thrombin III was normal. Genetic analysis for thrombogenic mutations was normal except methylene tetrahydro folate reductase (MTHFR) gene, which displayed homozygous allele mutation for the C677T polymorphism. Based on this finding, folic acid was added to the treatment regimen. As the patient refused to receive warfarin, antiplatelet therapy was intensified with the addition of clopidogrel 75 mg/day, in order to minimize the risk of recurrent thrombotic events. The patient is now under close surveillance, and free of symptoms except mild to moderate exertional dyspnea (NYHA II). Discussion: Thrombotic events including acute MI and stroke are not very rare in young subjects and should always raise suspicion of an underlying genetic mutation. The most common of these thrombogenic mutations are Factor V Leiden, prothrombin 20210, and the MTHFR mutations.The influence of the latter may sometimes be underestimated when compared to the first two, but literature provides evidence that particularly the mutations for the C677T polymorphism are related to clinical thrombotic events, especially when in the homozygous form. A busy clinician may sometimes focus on medical and interventional therapy, and unintentionally neglect the genetic aspect. Nevertheless, as our case demonstrates, thrombogenic mutations should always be sought especially in young subjects presenting with acute STEMI, as their detection may potentially alter the treatment regimens and future care for these patients.

PO-015

Simultaneous st elevation myocardial infarction of left anterior and right coronary artery after cannabis use

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A 39-year-old female patient admitted to emergency department due to chest pain that began 2 hours after cannabis use. She had classical risk factors for atherosclerosis as diabetes mellitus, tobacco smoking, hypertension and obesity. On physical examination S3 was present with a 3/6 pansystolic murmur. Electrocar¬diogram denoted ST elevations on the inferior and anterior leads. The echocardiography revealed advanced systolic dysfunction with an ejection fraction of 20% and moderate to severe mitral regurgitation. The patient was given clopidogrel loading dose of 600 mg and aspirin 300 mg orally. Immediately she was taken to the cath-lab for coronary angiography where we confirmed total occlusion of right coronary artery (RCA) and left anterior descending coronary artery (LAD). PCI of the proximal LAD with 2.75x18 mm DES and 3.0x24 mm stenting of the right coronary artery were performed successfully (Figure 1,2). Remarkable thrombus load and vasospasm in these coronary arteries were noteworthy. The exact mechanism of myocardial infarction following cannabis use remains controversial. The majority of patients reported in literature having acute coronary syndrome after cannabis use were smokers. Coronary vasospasm and coronary thrombosis were both reported in case reports in literature. One can argue that coronary spasm triggered by cannabis use can result in endothel damage giving rise to acute thrombosis at the position of a vulnerable atherosclerotic plaque. There are case reports of ACS after cannabis use with one vessel occlusion and moreover, there is a case report of a patient having simultaneous thrombosis of LAD and RCA who was crystal methamphetamine abuser. However, to the best of our knowledge, our patient is the first reported case having synchronous anterior and inferior myocardial infarction after cannabis use.

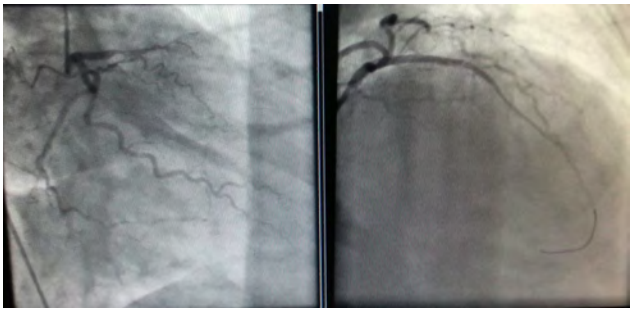

Figure 1. LAD before and after stenting.

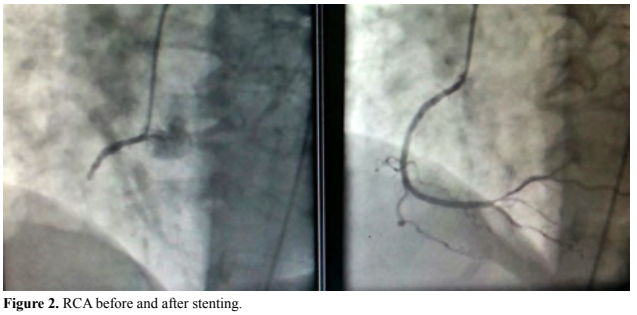

Figure 2. RCA before and after stenting.

PO-016

Kounis syndrome after intervention to a non-ruptured hydatid cyst

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

[1]*Antalya Education and Research Hospital, Antalya*

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A 34-year-old male patient had anaphylactic shock during hydatid cyst aspiration being executed by nonvascular interventional radiologists. Even after detailed evaluation, any leak or rupture of cyst could not be found. The patient was taken to intensive care unit due to hypotension (60/30 mmhg), ST depression on the monitor (D2 derivation) and chest pain. Noradrenalin infusion was administered as well as diphenhydramine 40 mg IV and prednisolone 100 mg IV, however, the patient was intubated owing to impending larengeal edema and subcutenous adrenalin was applied. During follow-up, arterial tension recovered to 110/60 mmhg and larengeal edema improved. The patient was extubated, however, troponin I level increased up to 37 ng/ml and acetylsalicylic acid and clopidogrel were administered. On the third day of hospitalization, coronary angiography revealed normal coronary arteries. Anaphylaxis is a hypersensivity reaction that is rapid in onset and may have lethal consequences. Mediators are released from mast cells and basophils via IgE mediation. These mediators are histamine, neutral proteases, thromboxane, leukotriens and oxidative enzymes. These mediators can induce platelet activation and cause coronary artery spasm. Acute coronary events by virtue of allergens or hypersensitivity reactions are described as Kounis syndrome. Its remedy involves both suppression of immune system and treatment of acute coronary event. There are reported cases of anaphylaxis due to ruptured hydatid cysts, however, Kounis syndrome after intervention to hydatid cyst without rupture is extremely rare. To the best of our knowledge, our case is the third reported article demonstrating acute coronary syndrome (ACS) after intervention to a non-ruptured hydatid cyst. Moreover, our article supports that ACS and anaphylactic shock in patients without macroscopic rupture of cyst wall may not be a coincidence. This potential complication should be noticed by physicians executing intervention to hydatid cyst.

PO-017

The implantation of percutaneous transvenous mitral annuloplasty device in serious mitral regurjitation

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Percutaneous transvenous mitral annuloplasty device system may be a good option for patients with severe mitral insufficiency(MI) patients in case of high surgical mortality risk. A 67-year-old female symptomatic patient with non-ischemic dilate cardiomyopathy was admitted to our clinic despite optimal medical treatment. She had increasing of shortness of breath (NYHA Class-III) since one year. On transthoracic echocardiography, LVEF was 23% and serious mitral-tricuspid regurgitation and biatrial dilatation were detected. Left ventricular diastolic and systolic diameters were 58/51 mm, mitral annulus diameter was 50mm, left atrium was 58 mm, pulmonary artery systolic pressure was 45 mmHg and vena contracta diameter of mitral regurgitation was 9 mm. 9F sheath was implanted to the right internal jugular vein. Coronary angiography was performed to evaluate whether there was a compression to coronary arteries and it was also evaluated the

anatomy of coronary sinus before and during the implantation procedure. Under flouroscopic guidance, 0.035 inches hydrophilic gliding wire and a multipurpose catheter were inserted via a 9F guiding catheter to the distal part of the coronary sinus (Fig-1). Then, the gliding wire and the multipurpose catheter were removed and the stiff wire was inserted to the distal point. Marked pigtail catheter was pushed over guiding catheter and after giving contrast agent, the proximal-distal diameter and extension distance were calculated (Fig-1). The distal and proximal anchor of the device were released to the distal and proximal portion of coronary sinus (Fig-1). During the implantation procedure, coronary angiography was performed simultaneously and there wasn't any compression to circumflex artery (Figure-1) The procedure was terminated successfully. On transthoracic echocardiography after the procedure vena contracta diameter of MI was regressed to 4 mm and the degree of mitral regurgitation was also diminished.

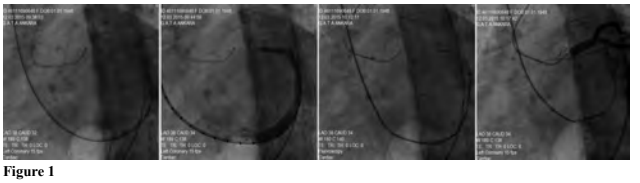

Figure 1

PO-018

Dermatologic findings may indicate severe cardiac involvement

Örsan Deniz Urgun, Duygu Genç, Burçak Kılıçkıran Avcı, Barış İkitimur, Zeki Öngen

İstanbul University Cerrahpaşa Faculty of Medicine, İstanbul

58 years old male, without any known disease, presented to our outpatient department with progressive dyspnea (NYHA 4) and leg edema. Physical examination revealed third heart sound, +++ lower extremity edema and diminished breath sounds on both lower lung zones. The ECG showed atrial fibrillation with low QRS voltage (Figure 1). The chest X-ray showed bilateral pleural effusion. Echocardiography demonstrated marked LV hypertrophy (Figure 1) with severe diastolic dysfunction, global hypocontractility (LVEF 35%) and increased PASP (55 mmHg). Multiple mediastinal lymphadenopathies were found in chest CT. On the 3rd day after his admission, spontaneous periorbital ecchymosis was appeared (figure 2). In last 2 years the patient had noticed recurrent ecchymotic lesions on both periorbital regions and body, which resolved spontaneously in 1 to 3 weeks. Although he asked medical attention several times, no diagnosis was established. Punch biopsy of these lesions, serum immunoelectrophoresis and urine analysis revealed multiple myeloma with systemic primary amyloidosis (prA). He received IV diuretics and inotropic support. Chemotherapy was initiated. Nevertheless his dyspnea didn't respond to therapy. Thoracentesis was done to relieve symptoms however his dyspnea persisted. By the 40th day he died because of sudden respiratory arrest.

Discussion: Pleural effusion occurs in prA, usually due to restrictive cardiomyopathy or nephrotic syndrome. In this case since it was refractory to aggressive diuresis, pleural amyloid infiltration was the most probable cause for its development. Periorbital hemorrhages (so-called raccoon eyes) occur spontaneously due to deposition of amyloid in the capillary walls, leading to fragility of capillaries. Early recognition of amyloidosis-induced ecchymosis may lead to early diagnosis and prevents additional myocardial damage. However our patient was diagnosed on the late stages of the disease and he did not respond any therapy.

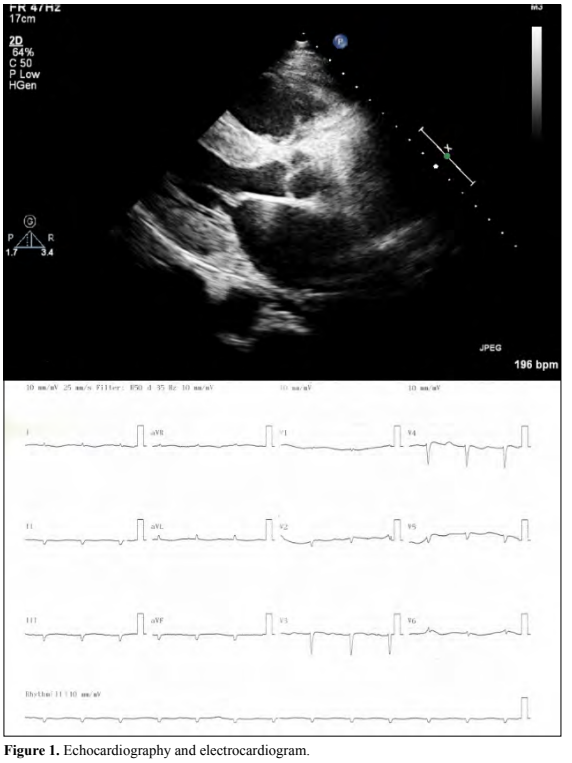

Figure 1. Echocardiography and electrocardiogram.

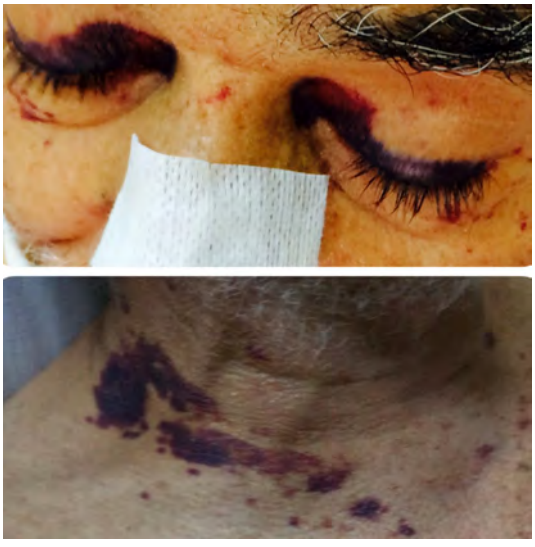

Figure 2. Bilateral periorbital and neck ecchymosis.

PO-019

A very rare cause of heart failure and significant improvement by short-term treatment: cardiac beri beri

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Dokuz Eylül University, Faculty of Medicine, İzmir

Cardiac beriberi has been reported for centuries, however it is very rare in the modern era. It is commonly missed without a high index of suspicion. Here we present a case of cardiac beriberi presenting as heart failure which improved dramatically after thiamine administration. A 57 year old male admitted to our department with New York Heart Class III dyspnea. His past medical history revealed smoking and alcohol use. On physical examination blood pressure was 100/60 mmHg, respiration rate was 26 per minute, heart rate was 120 beats per minute, temperature was 36.5°C and oxygen saturation was 93% in room air. A third heard sound was audible on cardiac auscultation, and respiratory sounds were diminished on both lower lung fields. Hemoglobin level was 10.5 g/dl, white blood cell count was 8.5x103 cells/ml (neutrophils 73.3%, lymphocytes 15.9%), and platelet count was 252x103 cells/µl on laboratory investigation. The biochemical profile showed: total protein 6.2 g/dl, albumin 3.2 g/dl, blood urea nitrogen 31.5 mg/dl, creatinine 1.08 mg/dl, CK-MB/troponin I 3.8/0.11 ng/ml, brain natriuretic peptide 795.0 pg/ml, sodium/potassium 126/4.1 mmol/l. His chest X-ray showed cardiomegaly with mild pulmonary congestion, and pleural effusion. Electrocardiogram (ECG) demonstrated nonspecific ST-segment changes in lead III, aVF and V2–V6. Diuretic treatment with supplemental oxygen was administered with the diagnosis of heart failure. Echocardiography showed left ventricular ejection fraction (LVEF) of 50%, with an end-systolic diameter of 4.1cm, and end-diastolic diameter of 5.4 cm, second degree tricuspid regurgitation with a pulmonary arterial systolic pressure (sPAP) of 60 mmHg. Despite of the treatment with supplemental oxygen, beta blocker, and diuretic the patients functional class did not improved significantly. Coronary angiography was performed for the suspicion of coronary artery disease but there was no significant atherosclerotic lesion. But left and right heart catheterization revealed a cardiac output of 6.9 L/min, elevated pulmonary capillary wedge pressure of 18 mmHg. These findings with the history of chronic alcohol use gave the suspicion of high output heart failure due to thiamine deficiency and we began oral thiamine supplement to the patient. The patient improved in a short-period of time with this treatment and discharged home. At the 2nd month control functional status of the patient was NYHA 1. Echocardiography showed LVEF of 61%, end-systolic diameter of 2.8 cm, end-diastolic diameter of 4.4 cm and minimal tricuspid regurgitation with a normal sPAP. And biochemical profile showed Na/K: 142/4.35 mmol/L, brain natriuretic peptide 63.0 pg/ml. Although it is now relatively rare, clinician should take into account cardiac beriberi due to thiamine deficiency as one of the differential diagnoses in patients with heart failure. Dramatical improvement usually come out with short term treatment.

PO-020

Diüretik tedaviye dirençli kalp yetersizlikli bir hastada tolvaptan ile hiponatreminin düzeltilmesi ve ultrafiltrasyonun kesilmesi

Saim Sağ, Ercan Çeğilli, Abdülmecit Yıldız, Bülent Özdemir, Ali Aydınlar

Uludağ Üniversitesi Tıp Fakültesi, Bursa

Vaka: 60 yaşında erkek hasta nefes darlığı ve yaygın ödem ile acil servise başvurdu. 20 yıldır diyabet ve hipertansiyon nedeni ile takipte olan hasta 2003 yılında anteriyor miyokard enfarktüsü geçirmiş ve sonrasında baypass operasyonu olmuş. 2012 yılında yapılan koroner anjiografide baypas greftleri açık saptanıp medikal tedavi kararı verilmiş. Hastanın son 1 yılda 10 kez dekompanse kalp yetersizliği ile hastaneye yatış öyküsü mevcut. Son 3 yatışında diüretik tedaviye yanıt alınamayan ve derin hiponatremi gelişen hastaya ultrafiltrasyon tedavisi uygulanmış. Hastanın başvuru esnasında karvedilol 25 mg/gün, ramipril 10 mg/gün, furosemid 80 mg/gün,İnsulin glargine 20 IU/gün, spironolactone 25 mg/gün, aspirin 100 mg/gün ve metformin 2000 mg/gün kullanıyordu. Hastanın başvuru sırasında kan basıncı 100/50 mmHg, nabız ritmik, 95 /dakika, dispneik, taşipneik, solunum dakika sayısı 24 /dakika, +++/+++ pretibial ödem, karın distandü, yaygın asit ve bileteral masif plevral efüzyonu mevcuttu. Yapılan ekokardiyografide EF %25, apikal anevrizma, anteriyor akinezi, biatrial dilatasyon, orta-ileri mitral yetersizliği ve ciddi triküspit yetersizliğine eşlik eden 70 mmHg pulmoner arter basıncı mevcuttu. Laboratuvar değerlerinde Hemoglobin 10.5 gr/dl, üre 120 mg/dl, kreatinin 1.5 mg/dl, serum potasyum 4.1 mmol/L, sodyum (Na) 128 mmol/L ve pro-BNP 1423 ng/ml saptandı. Hastaya sıvı kısıtlamasına (500 mL/gün) ek olarak intravenöz devamlı infüzyon olarak 500 mg/gün furosemid ve pozitif inotropik tedavi başlandı. Günlük 2500-3000 mL diürez sağlanmasına rağmen hastanın serum Na değerleri giderek düştü ve 110 mmol/L düzeyine inince hipertonik sodyum uygulaması başlandı. Hipertonik sodyuml ile artan dispne ve akciğer ödemi gelişince hastaya diyaliz kateteri açılarak ultrafiltrasyon tedavisi başlandı. Ultrafiltrasyon ile genel durumu ve ödemi rahatlayan hastada 10. günde kateter enfeksiyonu gelişince kateter çekildi ve diüretik tedavi artırıldı. Diüretik tedavisi alırken hastada tekrar derin hiponatremi gelişince (Na <100 mmol/L) tolvaptan 15 mg/gün tedavisi başlandı. Tolvaptan tedavisi ile hastanın serum Na değerleri 3 gün içinde normal seviyeye çıktı. Hasta tolvaptan 15 mg/gün ve furosemide 80 mg/gün tedavisi ile taburcu edildi. Hastanın 3 ay içinde tekrar hastaneye yatışı gerektiren semptomu olmadı. Hastanın haftalık takiplerinde serum kreatinin ve Na değerleri normal düzeylerde seyretti. Tartışma: Tolvaptan ile mortalitede iyileşme olmasa da konjestif semptomları iyileştirmede ve tekrarlayan hastaneye yatışta azalma mevcuttur. Ayrıca, pahalı ve kompikasyonu yüksek olan ultrafiltrasyona göre göre daha iyi bir seçenek olabilir.

PO-021

Pheochromocytoma presenting as dilated cardiomyopathy: a case report

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Pheochromocytoma is a tumor of adrenal glands originating from chromaffin tissue. Episodic hypertension, tachycardia, sweating, palpitation are common presenting signs and symptoms due to catecholamine excess in circulation. Cardiovascular complications have also been documented in pheochromocytoma and include arrhythmias, myocardial infarction, left ventricular hypertrophy and catecholamine-induced cardiomyopathy presenting as dilated cardiomyopathy or Takotsubo cardiomyopathy. We present a case of pheochromocytoma diagnosed while investigating dilated cardiomyopathy and showed reversal of cardiomyopathy following surgical treatment. A 35-year-old male patient admitted to emergency service with acute progression of dyspnea. The patient had a history of palpitation, dyspnea, sweating, vomiting for 3 months and his complaints showed progression. Pulmonary edema was detected and the patient was given diuretic treatment in the emergency service. Bedside echocardiogram showed low ejection fraction and the patient was referred to cardiology clinic after relief of dyspnea. In cardiology clinic, transthoracic echocardiogram showed an ejection fraction of 27%. No ischemia and infarction were detected in myocardial perfusion scintigraphy and cardiac magnetic resonance imaging showed midwall late gadolinium enhancement in midventricular segments. The patient was diagnosed as dilated cardiomyopathy and started therapy including a beta blocking agent. Following therapy, complaints showed progression, suggesting the differential diagnosis of pheochromocytoma. Urinary excretions of metanephrine and normetanephrine were found to be abnormally high and abdominal computerised tomography showed right side adrenal mass of 4.0x3.5x3.5 cm, and the patient was referred to surgery. One month after the surgery of adrenal mass, the complaints of patient were relieved, NT-pro BNP showed regression to normal level from preoperative level of 25.800 pg/ml, ejection fraction increased to 41% from 27%, and the dimension of left ventricule cavity was dramatically reduced. The pathological evaluation of the adrenal mass showed malignant pheochromocytoma and the patient was diagnosed as dilated cardiomyopathy secondary to pheochromocytoma. Pheochromocytoma is a rare cause of dilated cardiomyopathy, but it is important to keep in mind before initiation of therapy for cardiomyopathy because beta blockade may worsen symptoms and surgical treatment of pheochromocytoma may improve cardiac functions. It is usually hard to diagnose and careful attention must be paid to the history of the patient. This case report highlights the symptomatic and echocardiographic improvement in dilated cardiomyopathy secondary to pheochromocytoma, following surgical excision of tumor.

PO-022

Long term usage of low molecular weight heparin in a patient with left ventricular assist device

Kamil Gülşen, Cenk Conkbayır, Levent Cerit, Barçın Özcem, Hamza Duygu, Onur Akpınar, İlhan Sanisoğlu

Near East University Faculty of Medicine, Turkish Republicg of Northern Cyprus

Introduction: Left ventricular assist device (LVAD) has become a wide spread treatment modality in advanced heart failure patients. As the number of the patient with LVAD increase, device related problems become more frequent. Bleeding, thrombosis, infection, right heart failure are the common complications. Anticoagulant management of the patient with LVAD who has a history of recurrent bleeding remains controversial. Herein we present a LVAD recipient who use low molecular weight heparin (LMWH) as an anticoagulant for 18 months.

Case: A 74-year-old mail patient admitted to our hospital cause of refractory heart failure symptoms. The patient' s status was evaluated as grade -3 in accordance to INTERMACS scale and a LVAD (Heartmate-II) was implanted to the patient (Fig. 1). After the procedure, he had abdominal bleeding which the bleeding region could not been explored in laparotomy. His antiplatelet and warfarin therapy was stopped and LMVH (enoxiparine) was started. The enoxiparine dosage was titrated considering factor Xa level. However, he had intramural hematom in rectus abdominis muscle where the enoxaparin was injected (Fig. 2). The injection side was changed first, than, cause of recurrent gluteal hematoma, enoxiparine was changed to tinzaparine. LVAD parameters remained stabil during to this time and there has been no any finding about device thrombosis under LMVH therapy for 18 months.

Discussion: This particular case also first LVAD implanted patient in Northern Cyprus and to the best of our knowledge oldest patient among to Turkish LVAD recipients. Bleeding is a serious problem in patients with LVAD. Although there are studies and case reports with regard to short term usage of LMWH instead of warfarin, data is scarce about long term anticoagulant management of a LVAD recipient with recurrent bleeding. We suggest that, our case could help the clinicians to making a decision in such challenging condition.

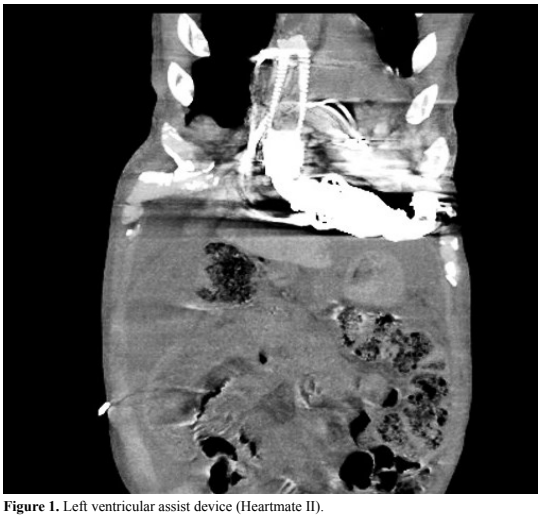

Figure 1. Left ventricular assist device (Heartmate II).

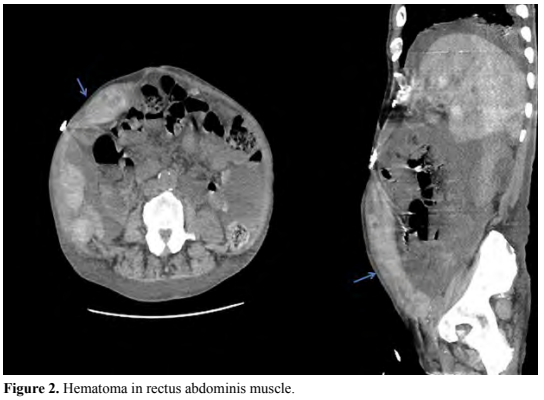

Figure 2. Hematoma in rectus abdominis muscle.

PO-023

Myocardial infarction due to spontenous coronary arter dissection in a patient witg left venticular noncompaction: a case report

Selahattin Aydın[1], Mevlüt Serdar Kuyumcu[1], İlker Duman[2], Meryem Kara[1], Sefa Ünal[1], Elif Hande Özcan Çetin[1], Özcan Özeke[1], Omaç Tüfekçioğlu[1]

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Introduction: Isolated left ventricular noncompaction (IVNC) is a rare congenital form of cardiomyopathy. Myocardial noncompaction occurs when the early, normal intrauterine process of endomyocardial morphogenesis is arrested, resulting in abnormal segmental hypertrabeculation (spongy myocardium) of the left ventricle (LV), and particularly at the apex. Echocardiography showing recesses in the wall, especially around the apex, is diagnostic for this type of cardiomyopathy, though magnetic resonance imaging (MRI) is useful when no echocardiogram is available. Nevertheless, LVNC can be mistaken for other conditions, such as dilated, hypertrophic, and apical hypertrophic cardiomyopathies,6 or infiltrative disease such as cardiac amyloidosis, and LV opacification might help to confirm the diagnosis. The incidence of LVNC is uncertain, but has been estimated at 0.12 per 100,000 in children younger than 10 years. The main causes of mortality and morbidity in this patient group are heart failure (HF), embolic events and lethal arrhythmias. Thus, patients are likely to complain of breathlessness, fatigue, and peripheral edema. Electrocardiogram features include extreme QRS voltages, isolated T waves, and evidence of Wolff–Parkinson–White syndrome, with premature contraction of both atria and ventricles.ST-segment elevation myocardial infarction (STEMI) is a rather unexpected complication in these patients and rarely reported in the literature. Mostly, this combination is a result of coexistent LVNC and atherosclerotic coronary disease, rather than a direct causal relationship. In some instances, however, STEMI could be directly caused by thromboembolic material dislodged from the LV Cavity or by microvascular dysfunction. In this case, a 43 years old woman admitted with STEMI, risk factors were cigarette and family history. On coronary angiogram, there was a total occlusion with spontaneous dissection of the RCA. This case hadn't be published to date in the literature.

Case Report: A 43- year- old female patient was admitted with typical cheast pain and diaphoresis that had begun an hour before admission. Her family history was remarkable. She has smoken for 20 years and 2 packages in a day. No notable findings were found during physical examination. Cardiac biomarkers were significantly elevated, with troponin I level of 50 ng/dL. Electrocardiogram demonstrated ST elevation >1 mm in D2, D3, AVF and resiprocal ST depression in D1-AVL leads. We decided to perform coronary angiography immediately, which showed total occlusion of the RCA with spontane dissected lesion. Firstly we couldn't passed to the true lumen with 0,014 inches floppy wire so that dissected lesion extended to proximal. After that we managed to pass true lumen and implanted to dissected lesion graft stent. Thus TIMI 1-2 flow occured in the RCA. We carried the patient to coronary care unit. She was treated with anticoagulants, glycoprotein 2b-3a inhibitor(tirofiban) infusion.

PO-024

Aortic greft tortion

Regayip Zehir, Mert İlker Hayıroğlu, Tolga Sinan Güvenç, Altuğ Ösken, Abdullah Kemal Tuygun, Şennur Ünal Dayı

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

Ascending aortic surgeries have been doing for years in order to protect patients from death due to aortic dissection. Many procedures put forward in course of time; composite valve and greft implantation such as Bentall, Buton and Cabrol technics, only greft implantation such as David and aortic remodelling technics. Here we discuss 34 years-old man operated three months ago with Bentall technic (aortic valve replacement (AVR-25 carbomedics)+supracoronary no. 30 tube greft ascending aort replacement+ coronary artery bypass greft x 1) presented to emergency with dyspnea and diffuse edema. He was evaluated in the emergency deparment and internated to hospital with congestive heart failure. Transthoracic echocardiography(TTE) showed ejection fraction (EF) 20% (globally hypokinesia), increased gradient in the tube greft (maximally 62 mmHg) in parasternal long axis view.Thoracoabdominal computed tomographic angiography and aortography were performed in the purpose of explaining the gradient. Surprisingly, aortic tortion was seen and the decrease in the ejection fraction thought to be secondary to greft tortion. Due to high risk of the re-operation, endovascular aneurysma repair (EVAR) technic was used successfully to decrease the gradient.

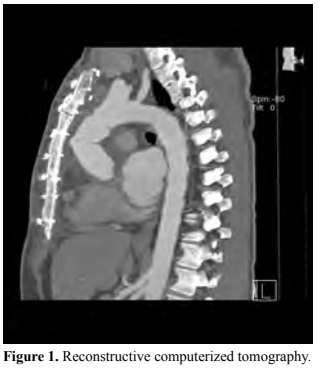

Figure 1. Reconstructive computerized tomography.

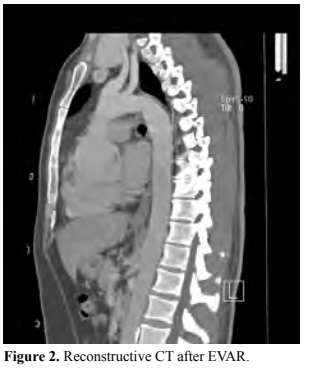

Figure 2. Reconstructive CT after EVAR.

PO-025

Sol ana iliaka arterde valsalva sonrası gelişen diseksiyonun balon ile açılan stentle başarılı tedavisi

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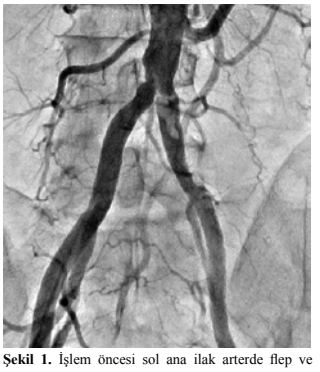
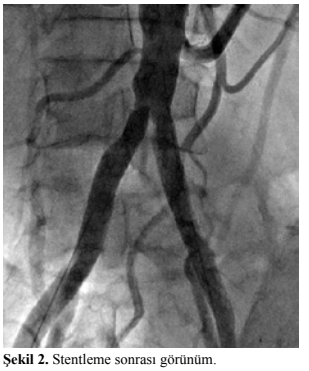
Gazi Üniversitesi Tıp Fakültesi, Ankara

46 yaşında erkek hastanın 2 ay önce 100 lt'lik varili kaldırırken zorlanma hissi ve sonrasında sol bacağında kladikasyo şikayeti başlamış. 50 m yürümekle ağrısı olan hasta bu yakınması nedeniyle kliniğimize başvurdu. Kardiyak muayenesinde özellik saptanmayan hastanın sol alt ekstremitesinde nabızlar alınamamaktaydı. 25 paket/yıl sigara kullanınmı olan hastanın kan biyokimyasında ve ekg sinde özellik yoktu. Alt ekstremiteye yönelik yapılan usg de sol ana iliak arter başlangıcında 2.5 cm'lik segmentte diseksiyon ve buna bağlı %90'lık darlık raporlandı. Hasta görüntüleme ve girişim açısından kateter laboratuarına alındı. Alt ekstremite anjiyografisinde sol ana iliak arter başında diseksiyon flebi ile buna bağlı ciddi darlık ve sağ ana iliak arter başında %50 darlık saptandı. Her iki femoral artere 6F ve sağ brakial artere 6F sheat yerleştirildi. Sağ brakial arterden 6F JR4 kılavuz kateter yardımı ile sol ana iliak arterdeki diseke lezyon 0.035 inç terumo hidrofilik tel ile geçildi. Sonrasında sol femoral arterden snare yardımı ile hidrofilik tel eksternalize edildi. Sağ femoral arterden de 0.035 hidrofilik tel ilerletilerek aortaya ilerlendi. Sağ ana iliak artere 7.0/39 mm ve sol iliak artere 7.0/79 mm balon ile açılan cordis genesis marka stent ilerletilerek her iki ana iliak artere V stent tekniği ile yerleştirildi. Kontrol anjiyografide her iki iliak arterde akımın iyi olduğu ve sol ana iliak diseksiyonun kapandığı izlendi. Hasta ertesi gün komplikasyonsuz olarak taburcu edildi. 2. ay kontrolünde hastanın asemptomatik olduğu görüldü.



Şekil 1. İşlem öncesi sol ana ilak arterde flep ve darlık.



Şekil 2. Stentleme sonrası görünüm.

PO-026

Improvement of heart failure secondary to Takayasu Arteritis following thoracic endovascular aortic repair procedure: a case report

Mustafa Kurşun[1], Cemil Gürgün[2], İnan Mutlu[2], Halil Bozkaya[2], Eren Mutlu[2]

[1]*Tepecik Training and Research Hospital, İzmir*
[2]*Ege University Faculty of Medicine, İzmir*

Takayasu Arteritis is a rare, chronic vasculitis of large vessels; predominantly affecting aorta, its main branches and pulmonary arteries. Aortic involvement may rarely cause left ventricular dilatation and systolic dysfunction secondary to increased afterload. We present a case report of a Takayasu Arteritis patient presenting with heart failure and showing improvement following thoracic endovascular aortic repair procedure. The patient was admitted to cardiology clinic with dyspnea, fatigue and left arm pain. By physical examination, pretibial edema and pulselessness in left radial artery were detected. Echocardiogram showed marked left ventricular dilatation and left ventricular ejection fraction of 27%. Thoracic CT angiography showed radiological findings of Takayasu Arteritis such as thrombosis of pulmonary arteries and left subclavian artery, medial hypertrophy in aorta and critical stenosis in the distal segment of thoracic aorta. For etiological evaluation of heart failure, myocardial perfusion scintigraphy was performed and no significant ischemia was detected. The patient was diagnosed as Takayasu Arteritis and heart failure secondary to increased afterload due to arteritis of aorta. Anticoagulation, heart failure and antiinflammatory therapies were initiated and after remission of arteritis, thoracic endovascular aortic repair procedure was performed. 3 months after the procedure, echocardiogram showed left ventricular ejection fraction of 40% and regression of left ventricular dilatation, and the patient was suffering less dyspnea ad fatigue. Thoracic endovascular aortic repair procedure is a promising treatment of Takayasu Arteritis, especially in patients with secondary heart failure. This procedure may provide relief of symptoms by improvement of cardiac functions and should be considered in such patients. This case report highlights the importance of thoracic endovascular aortic repair procedure in Takayasu Arteritis patients with heart failure.

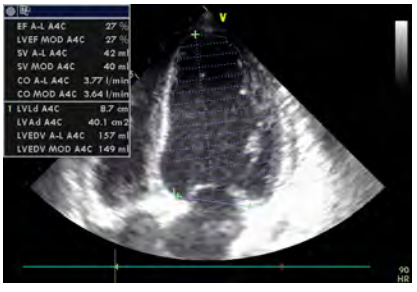


Figure 1. Low ejection fraction before TEVAR.

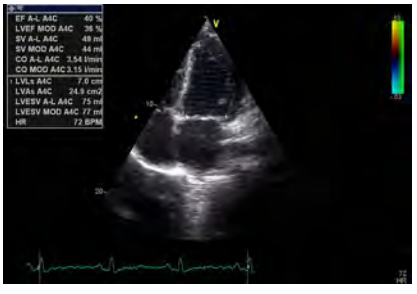


Figure 2. Improvement of ejection fraction after TEVAR.

PO-027

Exposed to hyperperfusion while avoiding from hypoperfusion: a very rare case of cerebral hyperperfusion syndrome

Erkan Köklü, İsa Öner Yüksel, Şakir Arslan, Göksel Cağırcı, Görkem Kuş, Murat Esin, Selcuk Küçükseymen

Antalya Education and Research Hospital, Antalya

A 54-year-old woman patient was investigated for the history of amourosis fugax and was found to have atherosclerotic lesions causing 90% stenosis in right internal carotid artery (ICA) (Figure 1A), 95% stenosis in left subclavian artery (Figure 1B). She had left subclavian steal syndrome. A 9.0x29 mm balloon expandable stent (Omnilink Elite, Abbott Vascular) was placed to 95% stenotic lesion in left subclavian artery via anterograde approach. Blood flow in left vertebral artery was changed to anterograde (Figure 1C). After the procedure, intraarterial blood pressure from femoral artery was 150/90 mmHg. The patient had severe left parieto-occipital headache right after the procedure in angiography room, upon which she started vomiting.Her neurological examination was normal and cranial diffuse magnetic resonance imaging (MRI) was negative for ischemia (Figure 2). Cranial computerized tomography did not demonstrate any bleeding or cerebral edema. CHS was suspected and patient was transferred to intensive care unit for intraarterial blood pressure monitorization and intravenous esmolol infusion. Control cranial diffusion MRI didnot shown any signs of pathology. After 5 days of follow-up, she was discharged. Possible mechanisms leading to CHS include cerebral autoregulation disorders, hypertension, ischemia-reperfusion injury, production of reactive oxygen species, baroreceptor dysfunction, and intraprocedural ischemia. Symptoms of hyperperfusion syndrome are ipsilateral headache, seizures, focal neurological signs and intracerebral hemorrhage. An increase in peak blood flow velocity of >100% is predictive of postinterventional hyperperfusion. CHS might be seen after subclavian artery stent placement in addition to carotid and vertebral artery interventions. Rapid diagnosis of hyperperfusion after symptom onset and early regulation of blood pressure are improtant to preprevent fatal complications.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

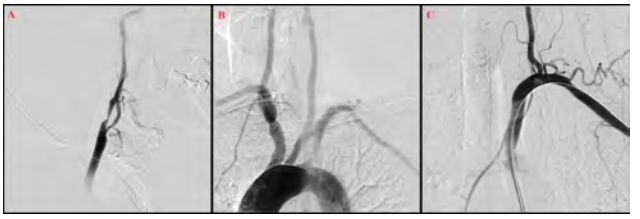


Figure 1. 90% stenotic lesions and Antegrade flow.

Figure 2. Diffusion magnetic resonance imaging of cranium.

PO-028

A successful case of percutaneous mechanical thrombectomy together with the sandwich technique for acute carotid artery in-stent thrombosis

Abdullah İçli, Kurtluş Özdemir, Ahmet Lütfü Sertdemir

Necmettin Erbakan University Meram Faculty of Medicine, Konya

A successful case of percutaneous mechanical thrombectomy together with the sandwich technique for acute carotid artery in-stent thrombosis Abdullah içli md, Ahmet Lütfü Sertdemir MD, Kurtuluş Özdemir MD Carotid in-stent acute thrombosis can cause thromboembolic events although it is a rare complication of carotid artery stenting CAS). We present a successful case of percutaneous mechanical thrombectomy together with the sandwich technique for acute in-stent thrombosis. A 68-year-old man was hospitalized for the treatment of a cerebral infarction presenting with dysarthria and left upper extremity weakness. Following sufficient medical management including dual anti-platelet therapy, CAS was performed because cerebral angiograms showed severe and long-segment left internal carotid artery ICA stenosis (A). In the immediate aftermath, after closed self expandable carotis stent placed we observed massive thrombus in instent (B_C). Emergency cerebral angiograms revealed left ICA occlusion due to in-stent thrombosis (C;D;E). Rapid revascularization with percutaneous mechanical thrombectomy of the in-stent occlusion was performed and then was replaced with a second closed-cell self expandable carotis stent sandwich technique (F;G). Post-procedural angiogram revealed recanalization of the left ICA (H), and the patient was free from neurological events after the last procedure. Stent was found to be patency in carotid CT angiography done after 6 months The evaluation and treatment of in-procedural in-stent thrombosis following CAS must be prompt and aggressive for prevention of catastrophic events. Percutaneous mechanical thrombectomy together with the sandwich technique is a useful tool for rapid treatment of acute in-stent thrombosis after CAS.

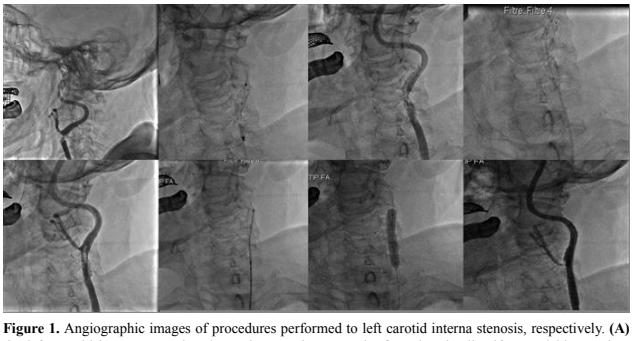


Figure 1. Angiographic images of procedures performed to left carotid interna stenosis, respectively. **(A)** the left carotid interna stenosis prior to intervention. **(B)** The first closed-cell self-expandable stenting. **(C)** in-stent acute massive thrombus after stent implantation **(D)** aspiration catheter application to in-stent intensive thrombosis. **(E)** After aspiration catheters application persistent intense thrombus **(F)** Placing a second closed-cell self-expandable stent in-stent "sandwich technique". **(G)** Postdilation. **(H)** successfully patency the left carotid interna stenosis.

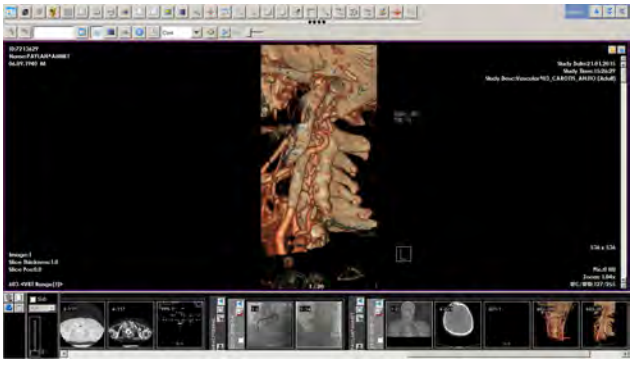


Figure 2. After 6 months the left carotid artery stenting were found to patency by carotid tomography imaging.

PO-029

De novo renal artery stenosis after renal sympathetic denervation

İbrahim Etem Çelik[1], Burak Açar[2], Alparslan Kurtul[1], Sani Namık Murat[1]

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59 year-old male patient was referred to our hospital for resistant hypertension. He had been diagnosed with hypertension six years ago and medical history was unremarkable except hypertension, no diabetes nor cardiovascular disease. His daily drug treatment was valsartan/hydrochlorothiazide 320/25 mg, amlodipin 10 mg, nebivolol 5 mg and spironolactone 25 mg. His blood pressure was well controlled until one year ago. Numerous drug combinations had been used for controlling hypertension, but none of them were effective. Serology and renal artery duplex revealed normal renal function and no renal artery stenosis (RAS). After exclusion the secondary causes of hypertension, renal sympathetic denervation (RDN) was planned. The procedure was performed under local anesthesia that 6 Fr introducer sheath was placed right femoral artery. Before performing renal denervation, renal artery angiography revealed absence of renovascular disease. SymplicityTM (Medtronic Ardian, Mountain, California) catheter was used for renal denervation. His blood pressure was under control after the procedure and he was discharged with taking valsartan 320 mg, hydrochlorothiazide 25 mg and nebivolol 5 mg. After the five months, he was admitted to us with the complaints of headache, palpitation and significant elevation of blood pressure despite the drug therapy. Laboratory measures were in normal range, renal arterial Doppler ultrasonography showed increased velocities at the origin of left renal artery (peak systolic velocity >300 cm/s). The patient underwent renal angiography according to these findings that showed left RAS of 90% which was not resolved with nitroglycerin (Figure 1). The stenosis was treated with 6.0x18 mm stent (RX Herculink Elite Renal Stent System, Abbott Laboratories, Santa Clara, USA) ve balloon angioplasty (Figure 2) and received dual antiplatelet therapy for one month. His blood pressure fell under 160/100 and his complaints were resolved after one week.

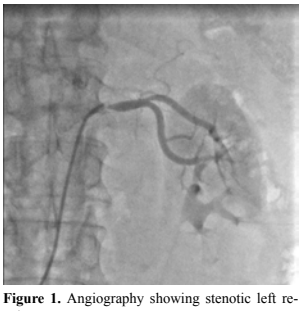

Figure 1. Angiography showing stenotic left renal artery.

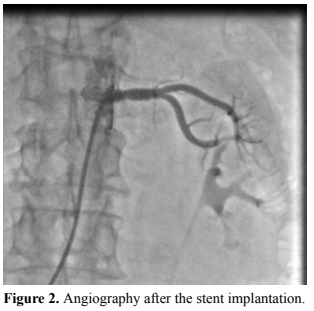

Figure 2. Angiography after the stent implantation.

PO-030

A case of a new anomaly of the left anterior descending artery: type X dual LAD

Turgay Çelik, Cengiz Öztürk, Uğur Bozlar, Samet Verim, Mustafa Demir, Sait Demirkol, Atila İyisoy

Gülhane Military Medical Academy (GMMA) Ankara, Ankara

Dual left anterior descending artery (LAD) is rarely encountered congenital coronary artery anomaly. In the current case we present a novel case of dual LAD anomally demonstrated by coronary computed tomographic angiography (CCTA). Up to date it has been reported 9 types of dual LAD variants detected not only in catheterization laboratory but also in CCTA. A 39 –year- old lady admitted to emergency service with the complaints of palpitation and dyspnea on exertion (NYHA Classs-III). Cardiac auscultation revealed a loud first heart sound and an opening snap in early diastole followed by a holodiastolic decrescendo rumbling murmur and loud holosystolic murmur. Morevover second pulmonary sound was found to be louder. Also she had fine crackles on basillary segments of both lungs. Resting ECG showed atrial fibrillation accompanied with biatrial abnormality and mild right axis deviation. Transthoracic echocardiography showed tight mitral stenosis, severe mitral and tricuspid insufficency combined with moderate to severe pulmonary hypertension. After stabilization of her medical condition it was consulted with cardiothoracic surgeons regarding to mitral valve replacement. Classical coronary angiography demonstrated that aberrant LAD originating from the right coronary sinus without evidence of epicardial coronary artery disease. Colored 3D volume rendered CCTA image showed both the long LAD and right coronary artery (RCA) originating from the right coronary sinus with different ostia on 320-row MDCT scanner. Short LAD originated from the LMCA and terminated in the proximal AIS. However it was noticed that long LAD originated from the right coronary sinus with separate ostium and followed an anomalous prepulmonic course anterior to the RVOT, and entered mid to distal AIS. She underwent succesfull robotically assisted mechanical mitral valve replacement. In conclusion we showed a novel type of dual LAD anomaly in the current case (Figure 1).

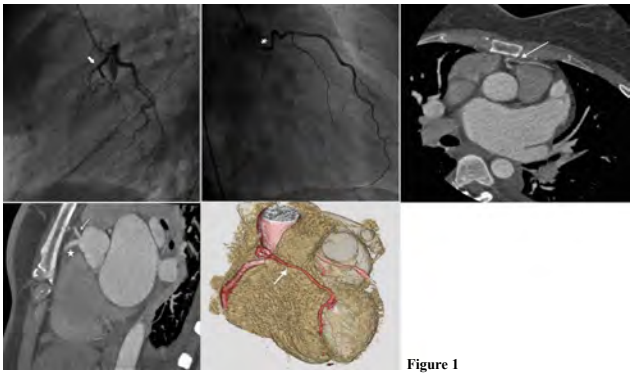

Figure 1

PO-031

Isolated right ventricular noncompaction —a case report

Mustafa Beyazıt Alkan[1], Murat Bilgin[2], Ahmet Sayın[3], İlker Gül[4], Burcu Zihni[5], Sanem Nalbantgil[6]

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[2]*Dışkapı Yıldırım Beyazıt Research and Educational Hospital, Ankara*
[3]*Tepecik Training and Research Hospital, İzmir*
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[6]*Ege University Faculty of Medicine, İzmir*

Isolated involvement of right ventricular noncompaction (RVNC) is very rare congenital cardiomyopathy. The left ventricule is the usual site of involvement, but involvement of both ventricles and rarely isolated RVNC can be seen. The main diagnostic criterion of left VNC; end-systolic ratio of noncompacted to compacted myocardium thickness >2, presence prominent trabeculations and deep intertrabecular recesses, absence of any coexisting cardiac anomalies, colour flow imaging revealed direct blood flow within the deep intertrabecular recesses. But, no specific criterion has been proposed for the diagnosis of right VNC. The clinical manifestations of VNC are highly variable, ranging from asymptomatic course to disabling congestive heart failure, systemic thromboemboli, arrhythmias and sudden death. In this case, we present 44 years old male patient presented with exertional dyspnea, who had a right VNC. Cardiac examinatiosn was normal. The 12-lead electrocardiogram showed sinus rhythm. Transthoracic two-dimensional echocardiography showed prominent trabeculations, with deep intertrabecular recesses in apex and free wall of the right ventricle. Colour Doppler demonstrated flow from RV cavity into the trabecular recesses. The right ventricular and atrium was mildly dilated. Right ventricular area changes fraction was %54. The pulmonary artherial pressure was normally. The coronary angiography was performed to exclude the possibility of a coronary artery disease. Epicardial arteries were normal. Cardiac magnetic resonance imaging (CMRI) was performed. CMRI confirmed the diagnosis of RVNC. Standard medical therapy, cardiac resynchronization therapy, implantable cardiac defibrillators and cardiac transplantation has been used for these patients. Our patient was treated with diuretics, angiotensin converting enzyme inhibitors, and beta-blocker.

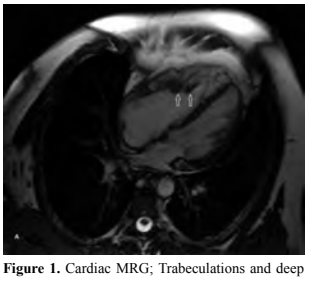

Figure 1. Cardiac MRG; Trabeculations and deep intertrabecular recesses.

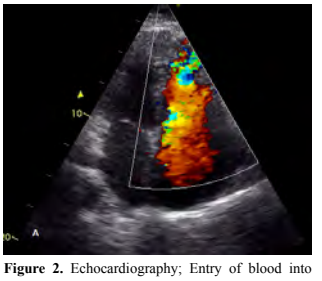

Figure 2. Echocardiography; Entry of blood into intertrabecular recesses.

PO-032

Ebstein's anomaly without tricuspid regurgitation in a sportsman

Zekeriya Arslan[1], Murat Ünlü[2], Cengiz Öztürk[2], Sait Demirkol[2], Uğur Küçük[3], Turgay Çelik[2], Atila İyisoy[2]

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Ebstein's anomaly is a rare congenital pathology defined by Wilhelm Ebstein in 1866 as displacement of the leaflet of the tricuspid valve through the apex at the right ventricle (RV). In general, it is characterized with a significant tricuspid valve insufficiency leading to RV failure and the right atrial dilation. Arrhythmias originating from this overload also can appear frequently. Ebstein's anomaly is seen in one twenty thousandth and only 5% of them can survive until the fifth decade. We herein present an asymptomatic 23 year-old male patient who applied for our Cardiology outpatient clinic for screening examinations. Previously, he accomplished the selection tests in the first place, for example, 400 m running test in 67 s which was very well success. He had no family history and no any risk factor but smoking for two years. Physical findings were normal, blood pressure and the heart rate were 124/82 mmHg and 64/rpm. ECG showed a sinus rhythm and fragmented QRS in V1-2 and inferior leads. Routine echocardiographic examination revealed that the tricuspid valve posterior leaflet was attached to the lateral wall of the right ventricle (Figure 1a), the septal leaflet originated from the mid portion of the interventricular septum (Figure 1b), and the anterior leaflet was rudimentary (Figure 1c). Interestingly, the tricuspid valve was completely able to close and there was no any insufficiency in Doppler images (Figure 1d). Interventricular septum basal portion was inclined towards the left ventricle (Figure 1e). Although there was not tricuspid regurgitation, the right atrium was dilated (Figure 1b). The patient didn't complain any palpitation and there was no any dysrhythmia during the short examination period. Although the Ebstein's anomaly is expected to be with tricuspid insufficiency, the function of the valve seems to be normal in rare circumstances like this case. We anyway recommended to this patient not to participate in sporting activities.

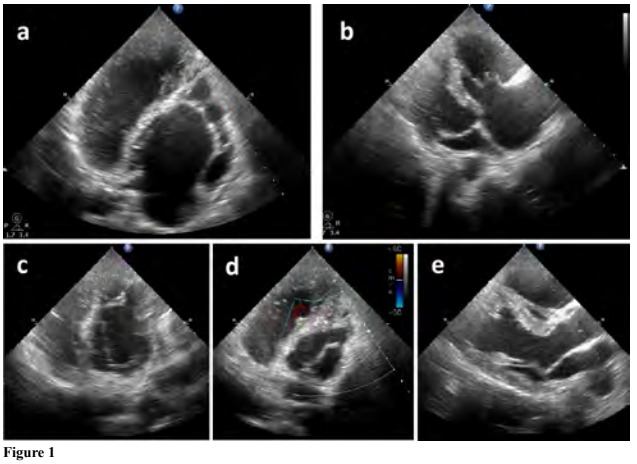

Figure 1

PO-033

Corrected transposition of great arteries and pulmonary stenosis in a patient with dextrocardia

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A 70-year-old man with no prior medical history was admitted to the out-patient cardiology clinic with the complaint of New York Heart Association functional class III dyspnea. He had no chest pain or palpitations. Family history and physical examination was unremarkable apart from pallor. The electrocardiogram showed a normal sinus rhythm with a heart rate of 60 beats per minutes and signs of biventricular hypertrophy. Echocardiography showed a pulmonary stenosis and pulmonary regurgitation. We detected a congenitally corrected transposition of the great thoracic vessels. Transesophageal echocardiography demonstrated that the ventricle of the right side was hypertrophic with decreased trabeculation and a typical left ventricular morphology. This ventricle appeared with normal function. The outflow vessel of this ventricle appeared as a pulmonary artery or pulmonary trunk with a low-grade stenosis (peak velocity 2.7 m/sec). Additively, mild pulmonary regurgitation and post-stenotic dilatation of the pulmonary artery was observed (Figure 1). The ventricle on the left side of the septum appeared as a morphological original right ventricle with increased trabeculation. This ventricle's systolic function was impaired. For further examination, cardiac magnetic resonance imaging (CMRI) was performed. CMRI calculated a mildly impaired anatomical right but functional left ventricular ejection fraction as 40% and revealed the switched anatomy (Figure 2). It was seen that an anteriorly positioned aorta on the left side, with steeply ascending aortic arch to the dorsal side and the pulmonary artery was located on the right side related to the aorta and preceded dorsal. The patient was evaluated in the council of clinics of cardiology and cardiothoracic surgery and discharged with conservative medical therapy.

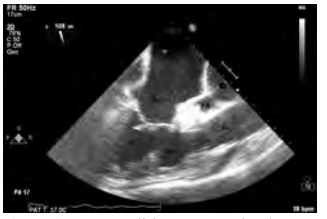

Figure 1. TEE: Parallel anatomy and pulmonary dilatation (*).

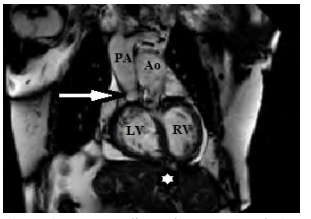

Figure 2. MRI revealing pulmonary stenosis (arrow).

PO-034

A patient with double inlet left ventricle reaching adulthood without surgery

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Introduction: Double inlet left ventricle is a rare congenital heart disease which is usually diagnosed soon after the birth. It is considered to carry a poor prognosis if untreated. The number of patients who have survived up to adulthood without surgery is quite low.

Case: A 45-year-old female patient was admitted to our hospital with complaints of exertional dyspnea, excessive fatigue and palpitations. Her history was double inlet left ventricle (DILV) and the transposition of the great vessels (TGA). Pulmonary artery banding (PAB) procedure was performed, when she was 5 months of age. Jugular venous distension and cyanosis of mucosa were detected on her physical examination. On admission electrocardiogram showed atrial fibrillation with high ventricular rate, right axis deviation and left ventricular hypertrophy. Transthoracic echocardiography (TTE) was performed and it was showed an enlarged left ventricle, hypoplastic right ventricle and a large ventricular septal defect. The apical 5 chamber TTE views showed that the aorta and pulmonary artery runs parallel to each other (TGA) (video 1). Single ventricle and the absence of interventricular septum was demonstrated in the apical 4 chamber TTE view and also both atrioventricular valves were connected to the left ventricle (double inlet morphology)(video 2). The pulmonary valvular gradient was measured as 29 mm Hg due to pulmonary artery banding. The patient was considered as a low ejection fraction heart failure because of the left ventricular ejection fraction was calculated as 35%, and the medical therapy was directed toward this. Spironolactone, furosemide and ramipril were added to her ongoing medical treatment with metoprolol, which is used beacuse of atrial fibrillation. In addition, digoxin was added to the treatment to reach targeted heart rate. Anticoagulation therapy was newly started, because the patient had refused to use anticoagulation before. Her symptoms improved after treatment. Thereafter, the patient was discussed also by pediatric cardiology and cardiovascular surgery.The heart transplantation was decided to be performed and further examination was initiated to examine the compatibility of the patient.

Conclusion: PAB is a palliative procedure which is used to decrease the pulmonary flow. Our case is one of the unique patients with DILV and transposition of the great arteries,who had undergone PAB without further surgery and survived into late adulthood.

PO-035

Left ventricular outflow tract obstruction after isolated subarterial ventricular septal defect repair

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35 year old male patient attended to cardiology clinic for pre-operative evaluation of his inguinal hernia. He had been operated for subarterial ventricular septal defect (VSD) when he was 9 years old. He had lost follow up but before his military duty, at the age of 20 had a full medical examination. Notes of this examination revealed a systolic murmur of 2nd degree intensity on left sternal border. The transthoracic echocardiography performed back then, showed mild degree aortic stenosis. The patient had stayed asymptomatic. On physical examination, there was a 3rd degree harsh systolic murmur on left sternal border and aortic position. Echocardiography performed. Aortic valve was mildly thickened with tricuspid morphology. The valve opening was slightly limited and aortic velocity was 2.01 m/sec with associated 16 mmHg peak and 10 mmHg mean gradient. There was also mild degree of aortic valve insufficiency.The repair patch of ventricular septal defect visualized as protruding through left ventricular outflow tract (LVOT). The patch caused a turbulant flow in LVOT, associated with 15 mmHg gradient. There also appears to be a suspicious left-to-right shunt from the patch on color doppler examination. The aorta showed aneurysmatic dilatation at sinuses of valsalva, reacing 3.9 cm diameter. There was no obstruction of flow at right ventricular outflow tract or pulmonary valve. Isolated VSD is the most common congenital heart defect. Long term results of VSD surgery has improved over years but late survival of patients are lower than general population. Late arrhthymic deaths, pulmonary hypertension, aortic insufficiency, need for pace-maker implantation are reported in long-term follow up of repaired VSD. Secondary subaortic stenosis (SSS) occurs after repair of some congenital heart defects in which isolated VSD is rare. Left ventricular outflow tract obstruction (LVOTO) and SSS is seen 26% to 57% of patients after repair of atrioventricular septal defect and VSD accompanying interrupted aortic arch with or without initial LVOTO. In one study, incidence of subaortic stenosis (SAS) after VSD closure is reported to be 3.2% and defined as an uncommon complication. Possible mechanisms of SSS development includes, endothelial stimulation in LVOT by turbulent flow, scar formation, left ventricular hypertrophy and remodelling, protrusion of VSD patch to LVOT. SSS patients, like SAS patients are prone to aortic insufficiency, infective endocarditis, decreasing ventricular performance. As most patients with SSS is asymptomatic for years, it is important to promptly recognize this complication. As uncomplicated, isolated VSD patients have been thought to have near normal life expectancy, current practice discharges them from routine cardiac follow up. It would be more convenient to follow up those patients for late development of such complications.

PO-036

Simultaneous hypertrophic cardiomyopathy and muscular ventricular septal defect in an adult patient

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Introduction: Hypertrophic Cardiomyopathy(HCM) is a common genetic cardiovascular disease and Ventricular septal defect(VSD) is a common congenital heart defect. The prevalence of muscular VSD among HCM patients is surprisingly high and is recently reported as high as 50% in familial types. But it is not usually reported.

Case Report: A 52- year-old man presented by himself at our out-patient clinic with dyspnoea and exercise intolerance. However, no syncope or pre-syncope was present. The patient had no history of rheumatic valve disease, congestive heart failure and hypertension. He did not have any family history of sudden cardiac death or congenital heart disease. On physical exam, the patient had a II/VI systolic murmur along the left sternal border. He has a regular pulse rate of 80 beats/min and a blood pressure of 130/85 mmHg. The patient's chest X-ray showed a normal sized heart. The electrocardiogram showed sinus rhythm, left ventricular hypertrophy and ST-T changes in precordial and lateral leads. Two- Dimensional Transthoracic echocardiography (TTE) revealed ventricular hypertrophy predominantly left ventricle anterior and anterolateral wall (1.97cm) No gradient at rest and with valsalva maneuver in left ventricular outflow tract (LVOT) blood flow or sings of mitral systolic anterior motion were detected. Parasternal long- axis and short-axis view, color doppler and continuous wave doppler ecocardiography showed the turbulent of muscular VSD jet through the septum.(Figure)

Discussion: VSD is the most common congenital heart malformation at birth (30–40%). It is mostly diagnosed and when indicated treated before adulthood. Spontaneous closure is frequent. The previous echocardiographic studies have estimated the prevalence of hypertrophic cardiomyopathy (HCM) in the general population to be 0.2% Two-dimensional echocardiogram is universally considered as the "gold standard" test for the initial evaluation of HCM and VSD patients.Generally, patients with these abnormalities can be detected early in their lives. we recommend carefully screening of all patients with HCM for an associated VSD.

PO-037

Atypical shone's complex

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A 21-year-old, female patient did not have any complaints until recent year. Seven months ago she was admitted to an outpatient hospital due to the complaints of severe headache and palpitation for 2 months. On examination the blood pressure was 230/100 mmHg in the right upper limb. The pulses were faint in both lower limbs while the pulse rate was 68/bpm in the upper limb. TTE revealed severe post-ductal coarctation of aorta (peak gradient: 65 mmHg) with a narrow and tortuous long segment, fibrotic mitral and aortic valves, mild aortic, mitral and tricuspid regurgitation, normal left ventricular ejection fraction (65%). After the exact diagnosis of coarctation of aorta, successful endoluminal balloon dilation and stent implantation with a low residual gradient was performed. The patient or her family did not seek any medical advice until the current episode. Subsequently the patient presented at our policlinic for progressively worsening dyspnea and palpitation commenced a few days before. Cardiac auscultation revealed a mild (grade: 1/4) diastolic murmur at the apex. TTE revealed mitral valve stenosis (mean gradient:7 mmHg), bicuspid aorta causing aortic stenosis (mean gradient:11 mmHg), mild to moderate aortic regurgitation, increased echogenicity in descending aorta proving stenting due to coarctation of aorta and poststenting 37 mmHg peak gradient (Figure 1). There was only one papillary muscle connecting all chordea proving parachute mitral valve. The presence of bicuspid aorta, parachute mitral valve (Figure 2) and coarctation of aorta make the diagnosis of atypical Shone's complex.

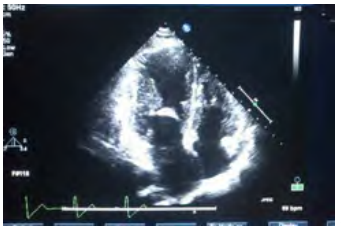

Figure 1. TTE apical 4-C view showing parachute mitral valve.

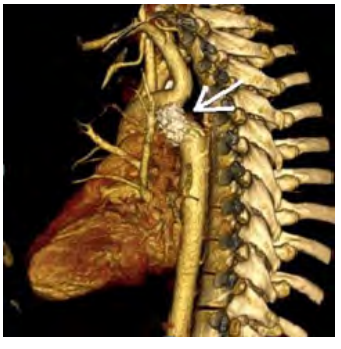

Figure 2. CT 3-D image showing stent in descending aorta.

PO-038

Nadir bir komplikasyon, erken protez mitral kapak endokarditine bağlı gelişen aorta-sol atriyal fistül

Fatma Erdem, Selçuk Öztürk, İbrahim Dönmez, Mehmet Yazıcı

Abant İzzet Baysal Üniversitesi Bolu Tıp Fakültesi Ar. ve Uyg. Hastanesi Bolu

52 yaşında erkek hasta acil servise senkop, konuşma güçlüğü ve sağ hemipleji ile kabul edildi. Hastanın romatizmal kapak hastalığı nedeni ile 60 gün önce aort ve mitral kapak değişimi yapıldığı öğrenildi. Fizik muayenede hemodinamisi stabildi. Elektrokardiyografisi sinüs ritmi ve birinci derece AV blok olarak izlendi. Beyaz küre:17.000, CRP:9.81 saptandı. Hastanın yapılan TEE'de ise mitral kapak sol atriyuma bakan yüzde multiple vejetasyon izlendi (Şekil 1). Aort kapak üzerinde vejetasyon izlenmedi. Hastaya uygun antibiyotik tedavisi başlandı. Genel durumu toparlayan hastaya yapılan kontrol TEE' de protez mitral kapak üzerindeki vejetasyonların tamamen kaybolduğu ancak aorta ile sol atriyum arasında 3 mm çapında 4-5 mm uzunluğunda, içinde sürekli akımın izlendiği, hemodinamik olarak önemsiz fistül izlendi (Şekil 2, Video1-2). Reoperasyonun yüksek mortalitesi ve eşlik eden komorbid durumları nedeni ile hasta operasyonu kabul etmedi. Aorta-sol atriyal fistülün hemodinamik olarak önemsiz olması sebebi ile de konservatif olarak izlenmesine karar verildi. Aorta kaviter fistüller genellikle aort kapak infektif endokarditlerine bağlı gelişir ve protez kapaklarda native kapak endokarditlerine göre daha sıktır. Bizim vakamızda ise mitral kapak endokarditi varlığı ve aorta-sol atriyal fistül izlenmiştir. Bu 2 nedenle açıklanabilir. Birincisi mitral kapak endokarditlerinde enfeksiyonun mitral kapaktan mitral kapak anterior leaflet bölgesi ile aortik annulusun posterior bölgesini birleştiren mitro-aortic intervalvular fibrosa bölgesine yayılması ile fistül gelişebilir.İkinci neden ise aort kapak üzerinde vejetasyon gösteremesekte enfeksiyonun aort kapağa ve çevre dokulara yayılması olabilir. İnfektif endokardit seyrinde fistül geliştiyse mutlaka cerrahi yapılmalıdır. Ancak bizim vakamızda olduğu gibi küçük bir hasta grubunda cerrahi riski yüksek, eşlik eden komorbid faktörler nedeni ile ve fistül hemodinamik açıdan önemsiz ise konsevatif olarak izlenebilir.

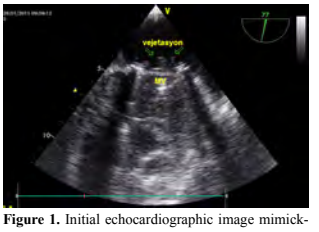

Figure 1. Initial echocardiographic image mimicking fibrin.

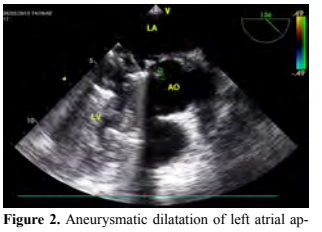

Figure 2. Aneurysmatic dilatation of left atrial appendage.

PO-039

Left atrial appendage aneurysm mimicking fibrin strands in patient with cardiac tamponade

Serdar Fidan[1], Anıl Avcı[1], Elnur Alizade[1], Cüneyt Toprak[1], Ali Metin Esen[2]

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A 70-year-old male with the history of end stage esophageal carcinoma and hypertension presented with worsening dyspnea for 2 weeks. Muffled heart sounds and hypotension (90/40mmHg) with pulsus paradoxus of 15 mm Hg were noted on physical examination. 12 lead electrocardiogram revealed atrial fibrillation with rapid ventricular response around 130beats/min.The initial transthoracic echocardiographic images revealed a large circumferential pericardial effusion and a worm-like structure floating around the left atrium that creates the impression of a thick fibrin strand attached to visceral pericardium (Figure 1). Further echocardiographic images revealed that the echogenic structure was actually aneurysmatic dilatation of left atrial appendage (Figure 2). The diagnosis of cardiac tamponade was made according to the clinical findings with help of echocardiographic findings (the right ventricular diastolic collapse, inferior vena cava plethora and a variation in mitral inflow greater than 25%). Pericardiocentesis was performed to drain 1000 mL of bloody fluid. Because of the short life expectancy due to the end stage esophageal carcinoma, further surgical interventions were not planned. The patient was discharged after successful pericardiocentesis. Aneurysms of the left atrial appendage may be congenital or acquired and they are rare entities in cardiology practice. The majority of patients are asymptomatic, but some patients may present with palpitation, dyspnea on exertion and stroke. In the absence of a knownpredisposing factor the condition has been assumed to be of congenital origin. In our case the probable cause of aneurysmatic dilation seems to be increased atrial pressure secondary to the left ventricular hypertrophy.

Figure 1. Initial echocardiographic image mimicking fibrin.

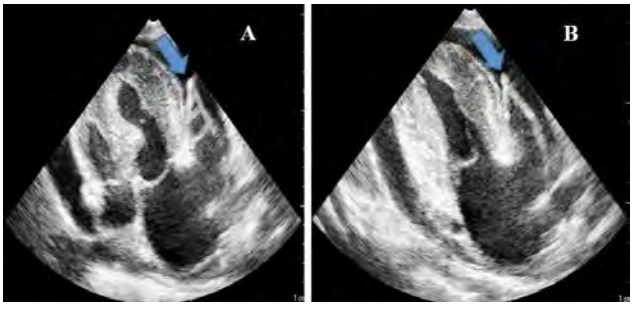

Figure 2. Aneurysmatic dilatation of left atrial appendage.

PO-040

Genç kadın hasta, asendan aorta dilatasyonu ve kronik göğüs ağrısı birlikteliği: Takayaşu arteriti

Reşat Mehmet Baha, İzlem Kıran Çelik

Osmaniye Devlet Hastanesi, Osmaniye

Giriş: Günlük klinik pratiğimizde asendan aorta dilatasyonu(AAD) nispeten seyrek saptadığımız bir patolojidir ve AAD saptandığında etiyolojiye yönelik yapılması gerekenler çoğunlukla eksik kalmaktadır.

Vaka: 32 yaşında, kadın hasta uzun süredir olan,batıcı tarzda göğüs ağrısı nedeniyle kardiyoloji polikliniğimize başvurdu. Öyküsünden ağrısının 3 yıla yakın süredir olduğu,bu şikayetinin hasta gebe iken başladığı öğrenildi. Hasta şikayetlerinin artması sebebiyle doğumun hemen sonrasında hastaneye başvurmuş,orada yapılan tetkiklerde hastada perikart sıvısı saptanmış. Perikardiyosentez yapılmış. Perikart sıvısı etiyolojisine yönelik yapılan tetkiklerde kesin tanıya ulaşılamamış. Geçen süre içerisinde hasta defalarca hastaneye başvurmuş ancak sonuç alamamış. Hastada batıcı tarzda göğüs ağrısı olması ve özgeçmişinde perikardit öyküsü olması sebebiyle hastaya transtorasik ekokardiografi yapıldı. Ekokardiografide hastada perikart efüzyonu yoktu, hafif aort yetersizliği ve en geniş yerinde 4.3 cm ölçülen AAD mevcuttu. Aort kapak normal ve triküspit yapıdaydı. Genç ve kadın hasta olması, şikayetlerinin gebe iken başlayıp giderek artmış olması ve hastanın düşkün görünümlü hali AAD'nin inflamatuvar süreçlerle ilişkili olabileceğini aklımıza getirdi. İlk etapta hastanın iki koldan kan basıncı ölçüldü ve kollar arasında 30 mmHg fark saptandı. Hastanın serum C-reaktif protein düzeyi 127 mg/L saptandı. Takayasu arteriti ön tanısı konuldu. Hastaya kontrastlı toraks bilgisayarlı tomografi (BT) planlandı. BT'de çıkan aorta ve dallarının duvarlarının yoğun inflamasyona işaret edecek şekilde kalınlaştığı görüldü. Tüm bu bulgular ile hastaya takayaşu tanısı konuldu ve hasta ileri merkeze yönlendirildi.

Sonuç: AAD herhangi bir hastada saptandığında etiyolojiye yönelik mutlaka kafa yorulmalı ve en azından aort kapak yapısının incelenmesi (biküspit aort varlığı açısından) önerilmektedir. Zor tanılara ulaşmada, hastalar, detaylı öyküleri, muayene ve tetkik sonuçları ile bir bütün olarak değerlendirilmelidir.

Şekil 1. Asendan ve inen aortada bariz duvar kalınlaşması.

PO-041

Floating left atrial appendage in massive pericardial effusion: an unusual sign of severe paravalvular leakage on a prosthetic mitral valve

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Background: The differential diagnosis for intrapericardial masses includes fibrinous strands, pericardial tumors and cysts which are generally appear in malign and infectious diseases. Pericardial effusion and paravalvular leakage (PVL) are severe complications after prosthetic heart valve replacement.

Purpose: We present an interesting image of a cystic intrapericardial mass which was diagnosed as a floating left atrial appendage (LAA) in a female patient with late postoperative massive pericardial effusion after mechanical mitral valve replacement.

Methods: A 36 years-old female patient referred to the our hospital with transient ischemic attack (TIA) occured three days ago and progressive dyspnea for about one week. She had a history of mitral mechanical valve replacement four weeks ago. Transthoracic echocardiography (TTE) and subsequently transesophageal echocardiography (TEE) were performed at baseline for evaluation of prosthetic mitral valve.

Results: TTE initially revealed a fibrinamatous density image within the massive pericardial effusion (Figure 1a). After a careful examination, multilobular, membrane like, freely moving and ballooned LAA was demonstrated in the massive pericardial effusion as if 'floating' (Figure 1b). TEE revealed a non-obstructive annular prosthetic valve thrombosis and an eccentric severe PVL jet that was directed towards the LAA (Figure 1c and 1d). Since the patient was symptomatic and hypotensive, immediate pericardiosentesis was performed and percutaneous PVL closure was planned.

Conclusion: This case illustrates an unusual presentation of LAA as a floating mass in pericardial effusion after the mitral mechanical valve replacement. This unusual entity was mainly caused by an eccentric severe PVL jet directed towards the LAA and should be remembered as a rare sign of severe mitral PVL.

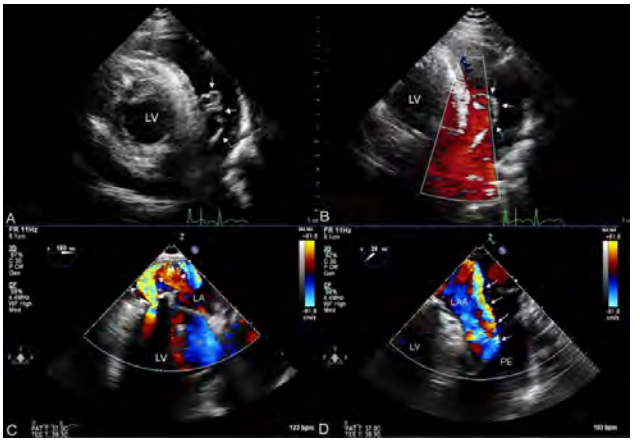

Figure 1. (A-D)

PO-042

Intra-cardiac bullet

Adnan Kaya[1], Emine Çalışkan[1], Reşat Doğan[1], Ahmet Öz[2], Muhammed Keskin[2]

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Penetrating cardiac trauma is rare to be seen but when present there is a short time lag to keep the patients alive. Cardiac firearm injuries are even rare and it is occurred mostly during interpersonal disagreements casualties or a mistakenly fired gun nowadays. A 32-years-old male fighter was brought to our emergency department with several other wounded fighters from the war of Kobani, Syria. Physical examination revealed a superficial wound on the left shoulder and another on the left side of thorax at the 6th intercostals space. There were no exit sides of these wounds which were thought to be bullet wounds. After first evaluation of the patient a thoracoabdominal computed tomography (CT) was ordered. A bullet was seen in his left shoulder at proximal humerus. The patient was interned for extraction of the bullet. There was a moderate dyspnea of the patient which was attributed to anxiety and pain shock. The patient was mobilized during the evening. He had a severe dyspnea which was resolved suddenly at 01.00 am and slept without any symptoms. The radiology doctor and the general surgeon of the hospital noticed an intracardiac bullet in the patient's CT while checking it. The CT shows a hyper-dens material in the heart with up to 2 cm pericardial effusion. Cardiology consultation was made. Electrocardiography showed no abnormality. A bed side transthoracic echocardiography (TTE) showed a hyper-echogenicity embedded to the connection of inter-atrial and inter-ventricular septum in apical 4 chambers view. There were no valvular insufficiency, no inter-ventricular or inter-atrial connections. The pericardial effusion seen in CT was drained to the right pleura. The patient was referred to a tertiary cardiovascular institution for follow up and definite treatment. After a detailed evaluation conservative follow up was decided and the patient was observed one week in the coronary care unit. He was discharged after resection of the bullet in his left shoulder.

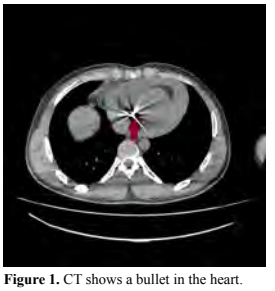

Figure 1. CT shows a bullet in the heart.

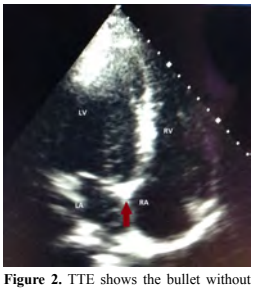

Figure 2. TTE shows the bullet without pericardial effusion.

PO-043

A patient with left ventricular pseudoaneurysm due to mitral valve surgery

Aynur Acıbuca, Demet Menekşe Gerede, Elif Peker, Şengül Aydın, Mustafa Adnan Uysalel, İrem Dinçer

Ankara University Faculty of Medicine, Ankara

Introduction: A left ventricular pseudoaneurysm is defined as a contained rupture or perforation of the myocardium. The rupture of the myocardium can be observed after myocardial infarction or cardiac surgery. Although it is a rare complication, it may lead to mortality. We present a case of left ventricular pseudoaneurysm after mitral valve replacement.

Case: A 72-year-old woman was admitted to our hospital because of a suspected aneurysms bulging from the base of the left ventricle, detected on transthoracic echocardiography. Her history revealed a bioprosthetic mitral valve replacement for mitral regurgitation and tricuspid ring annuloplasty for tricuspid regurgitation four months ago. The patient had no cardiac symptoms on admission. To characterize this aneurismatic structure in details a transesophageal echocardiography was performed and it revealed a pseudoaneurysm, which was located in the sub-mitral valve prosthesis area on the posterolateral wall. (Video 1) The size of aneurysmal neck was 12 mm and the area of aneurysm was 6.1 cm2. A magnetic resonance imaging was planned to make a clear distinction from the abscess formation and it showed a pseudoaneurysm, that was 3.5x3 cm in size. (Figure 1 and 2) Upon this, an interventional treatment was deemed appropriate, but the patient refused surgery and didn't go follow-up visit.

Conclusion: Left ventricular pseudoaneurysm due to late rupture after mitral valve replacement is rare but a life-threatening complication and imaging multimodalities is important for differential diagnosis and choosing the true treatment.

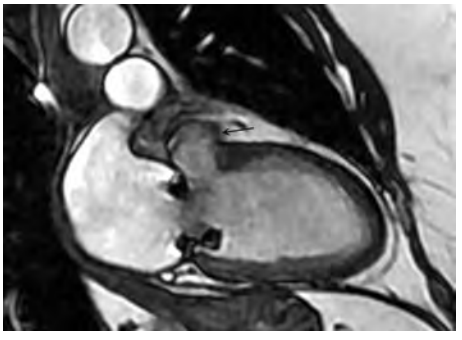

Figure 1. Pseudoaneurysm (arrow) formation on MR imaging.

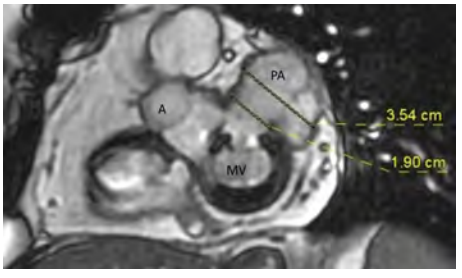

Figure 2. Measurement of the pseudoaneurysm size.

PO-044

EKG'de inferior ST elevasyonu saptanan hipertrofik kardiyomyopati olgusu

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37 yaşında kadın hasta 6 saat önce başlayan tipik istirahat anginası ile acil servise başvuruyor. Hastanın vitalleri nabız: 60/dk ritmik,tansiyon: 90/60 mmHg saptandı. Fizik muayenesinde kalp ritmik, sternum sol yanında 2-3/6 sistolik üfürüm,diğer muayeneleri olağandı. EKG'de sinüs ritmi kalp hızı: 68/Dk DII-DIII-AVF'de 1.5 mm ST elevasyonu ve T negatifliği, DIII-AVF'de patolojik Q,V4-6'da T negatifliği saptandı. Hasta subakut inferior ST elevasyonlu miyokard infarktüsü tanısı ile acilen koroner anjiografi salonuna alındı. Koroner anjiografide normal koroner arterler, ventrikülografide; ventriküler hipertrofi,daralmış ventriküler kavite ve papiller adale distorsiyonuna bağlı sol ventrikül çıkış yolunda (SVYÇ) kuğu boynu deformitesi saptandı. İntrakardiyak basınç ölçümü yapıldı. Sol ventrikül apexinde sistolik basınç 170 mmHg, sol ventrikül bazalinde sistolik basınç 90 mmHg (sol ventrikül içerisinde basınç gradienti 80 mmHg), aortta sistolik basınç 80 mmHg, diyastolik basınç 50 mmHg olarak saptandı. Ekokardiyografide (EKO) septum kalınlığı 2.4 cm, sistolik anterior motion (SAM) pozitif,hafif-orta derecede mitral yetmezliği, sol atriyum 4,0 cm, hafif derecede aort yetmezliği, SVÇY'de maksimum 30 mmHg ve 60 mmHg arasında dinamik gradiyent ve sol ventrikül ejeksiyon fraksiyonu normal olarak saptandı. Hasta ani kardiyak ölüm için risk faktörleri açısından sorgulandı. Ailesinde ani ölüm öyküsü ve hastada açıklanamayan senkop öyküsü yoktu. Hastaya 24 saatlik holter EKG tetkiki yapıldı, sonucunda non sustained VT saptanmadı. ESC 2014 HKMP kılavuzuna göre hastanın ani kardiyak ölüm riski %2.8 olarak hesaplandı. 5 yıllık ani kadiyak ölüm riski <%4 olarak izlendiği için ICD endikasyonu konulmadı. Hastaya metoprolol 50mg tb 1x1 başlandı. 3 ay sonraki kontrolünde benzer anginal yakınmasının tekrarlamadığı öğrenildi. EKG'de V4-6 derivasyonlardaki T negatifliklerinin devam ettiği ancak inferior derivasyonlardaki patolojik bulguların kaybolduğu görüldü. Kontrol EKO'da SVÇY gradienti normal izlendi, SAM'ın kaybolduğu görüldü.

PO-045

Progression of aortic valve endocarditis to a kissing vegetation; Kissing is a cause or a consequence?

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Near East University Faculty of Medicine, Turkish Republicg of Northern Cyprus

Mitral valve kissing vegetation (MVKV) is a clinical entity, which is assumed as a secondary mitral valve endocarditis due to directly contact of aortic valve vegetation. Although this mechanism is generally accepted, there could be another route to contagion aortic valve endocarditis to mitral valve. Herein we present a case report which demonstrate progression of aortic valve vegetation to MVKV via aorta-mitral continuity in an immunosuppressive patient.

Case report: A 69- year- old mail patient was admitted to our clinic because of shortness of breath, palpitation and fever. He was diagnosed with myelofibrosis six months ago. A large mobile mass considering vegetation was seen in transthoracic echocardiography (TTE) (Figure 1A, B) also this finding was confirmed with transeusophagial echocardiography. A couple of days later, his control TTE showed same aortic vegetation and also thickening in mitral valve anterior leaflet. In addition, it was pointed out that thickening was clearly at basal part of the leaflet which was close to aorta-mitral continuity (Figure 1C, 1D) However, disease progressed and vegetation completely covered atrial side of the anterior mital leaflet (Figure 2) and patient died There is a general agreement as mitral valve kissing vegetation is limited with mitral valve anterior leaflet ventricular side and reconstructive surgery could eligible almost all of the patients in that situation. However, in the presented case, unfortunately, we had a chance to follow natural course of aortic valve endocarditis to become a kissing vegetation. As shown in the images, infection affected basal part of the mitral valve anterior leaflet first and only atrial side of the leaflet got involved. This findings suggested that the inoculation was not via aortic vegetation touch, it was related with anatomic contiguity of the aortic and mitral valve

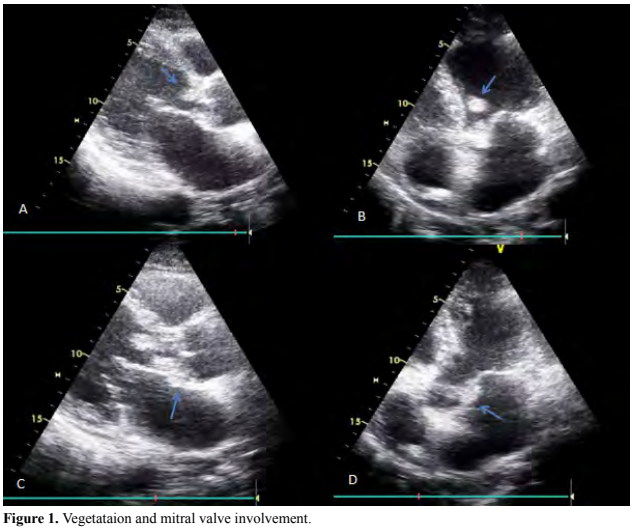

Figure 1. Vegetataion and mitral valve involvement.

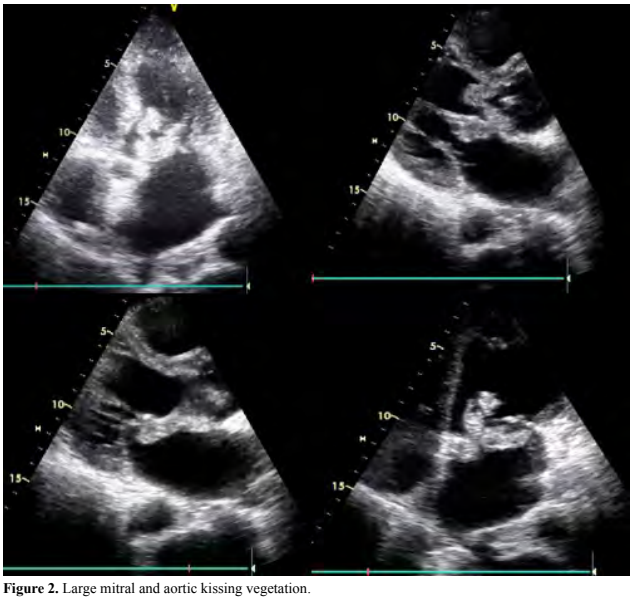

Figure 2. Large mitral and aortic kissing vegetation.

PO-046

Dancing vegetations; a rare endocarditis agent kocuria rosea

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Being a bacterium in the Micrococcaceae family, Kocuria rosea is a normal flora member which is found in skin and mucosa however it causes crucial infections in patients whose immune system has been suppressed. Here we present a rare case of infective endocarditis caused by Kocuria rosea in a patient with congenital heart disease. While 22 years old female patient has been followed up in the hematology department in terms of anemia etiology, it has been consulted to cardiology department due to parasternal pansystolic murmur heard in the physical examination. The patient was on echocardiographic follow-up for congenital membranous ventricular septal defect with 1,2 shunting rate. Transthoracic echocardiography revealed a perimembraneous ventricular septal defect with 0.5 mm diameter and huge mobile vegetations in both ventricular septal defect and right ventricle outflow tract where VSD jet caused erosion. (Figure 1-2) (Video 1)The patient has been referred to cardiology department due to ineffective endocarditis pre-diagnosis. Gram positive bacteria named as kocuria rosea has been isolated in 2 different blood cultures which is a rare pathogen for IE. Contrast enhanced thorax tomography showed extensive pulmonary septic embolies have been observed. Ceftriaxone 2x700 mg and gentamicin 2x70 mg have parenterally been applied for 4 weeks. After the appropriate antibiotic treatment, patient whose vegetations and clinic have been regressed was referred to elective surgery for VSD associated with IE. Although accepted as a member of skin flora, Kocuria rosea rarely may cause IE in immunocompromised patients. In this report we presented an IE case caused by Kocuria rosea which was successfully treated with ceftriaxon and gentamicin.

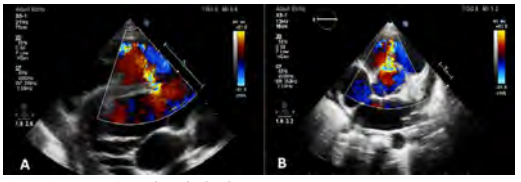

Figure 1. (A, B) VSD and erosioning jet.

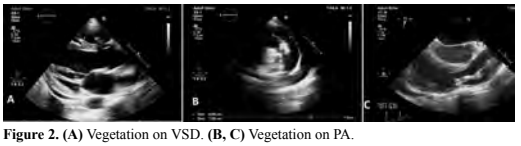

Figure 2. (A) Vegetation on VSD. **(B, C)** Vegetation on PA.

PO-047

Anomalous origin of the right coronary artery from the left sinus of valsalva: acute inferior myocardial infarction

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A 68-year-old-male with hypertension, diabetes and exertional chest pain of three months duration presented with sudden onset of chest pain. ECG showed ST-segment elevation in inferior leads and the patient was taken directly to the catheterization lab. Emergent coronary angiogram performed via femoral approach revealed an anomalous right coronary artery (ARCA) during the inital injection of left coronary system using a JL4.0 diagnostic catheter. ARCA was engaged with JL 3.5 guiding catheter for better guide support during the intervention. During diagnostic angiogram, an ARCA with takeoff adjacent to the left coronary artery was noted with total occlusion in its proximal portion. After inserting a 0.014mm guidewire with a 2.0x12 mm balloon support, multiple dilatations were performed. Finally, two consecutive stents (3.0x22 mm each) were implanted. Echocardiogram performed on the following day demonstrated an EF of 50-55% with a hypokinesia in the basal segment of the inferior wall. There were no post-PCI complications and he has been doing well on subsequent follow-up. ARCA is a rare congenital anomaly with a prevalance of 0.06 to 0.5%. Most ARCA's are asymptomatic but may be responsible from angina pectoris, myocardial infarction, syncope or even sudden cardiac death. Successful percutaneous treatment (PCI) of stenotic lesions in ARCA has been reported infrequently in literature. In order for PCI to be successful in ARCA, relies on optimal guiding catheter seating and catheter back-up should be achieved, both of which may require modification in the kind of guiding catheter used. In addition to anatomic factors, operator preference, familarity and institutional availability all influence catheter selection for PCI in these patients.

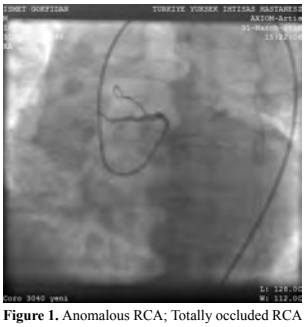

Figure 1. Anomalous RCA; Totally occluded RCA originating from left sinus.

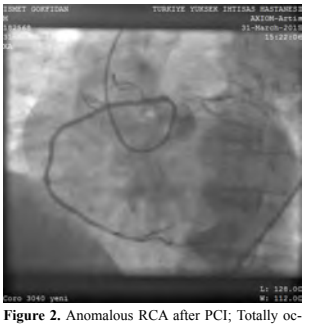

Figure 2. Anomalous RCA after PCI; Totally occluded RCA originating from left sinus.

PO-048

Kemoterapi sonrası oluşan tümör lizis sendromu ve akut böbrek yetersizliğini takiben gelişen Kounis sendromu

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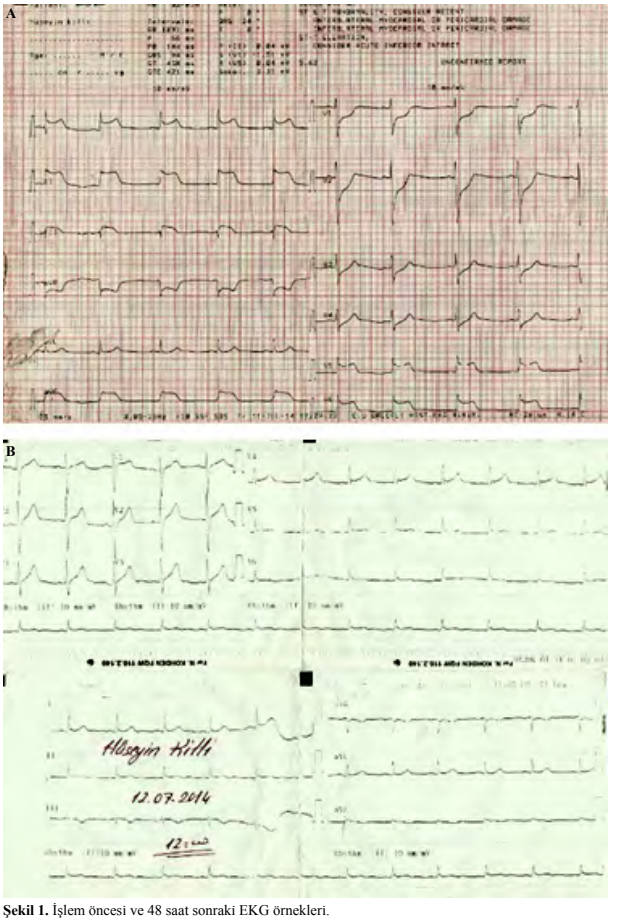
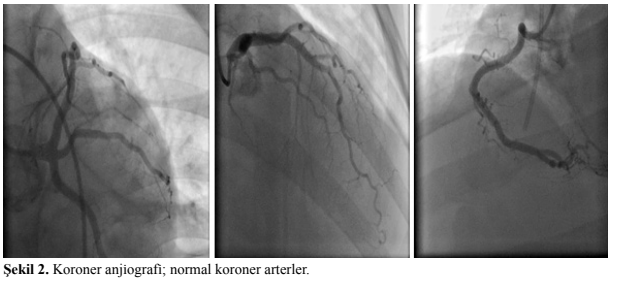
Çukurova Üniversitesi Tıp Fakültesi, Adana

Bir yıldır T hücreli lenfoma tanısıyla takip edilen ve bilinen kardiyovasküler hastalığı olmayan 27 yaşında erkek hastada HYPERCVAD/MTX-ARA-C kemoterapi protokolünü aldıktan sonra tümör lizis sendromu ve akut böbrek yetersizliği gelişmiş. İzlemde monitörde ST segment elevasyonu saptanan hasta bölümümüze danışıldı (Şekil 1A). Hastanın çekilen EKG sinde DI, DII, DIII, a VF, V5, V6 derivasyonlarında ST elevasyonu mevcuttu (Şekil 1B). Hasta acil koroner anjiyografiye alındı. Yapılan koroner anjiyografide normal koroner arterler olarak değerlendirildi (Şekil 2A). İzlemde 48 saat sonra çekilen EKG de ST segmentinin izoelektrik hatta geldiği görüldü.(Şekil 1C) TnT ve CKMB değerlerinde gerileme oldu. Hastada kemoterapiyi takiben Kounis sendromu geliştiği düşünüldü. Kounis sendromu, mast hücrelerinin etkinleşmesi ile seyreden alerji, hipersensitivite, anaflaksi veya anaflaktoid reaksiyonlarla ilişkili olarak akut koroner sendrom (AKS) gelişmesi durumudur. Kounis sendromu ya da alerjik Miyokard Enfarktüsü(MI) olarak adlandırılan bu durumun patofizyolojisine ve koroner arter hastalığı varlığına göre iki tipi vardır. Kounis sendromundaki temel patofizyoloji alerjik uyarıya ikincil olarak mast hücrelerinin aktive olması ve bu hücrelerden histamin gibi biyojenik aminlerin; kimaz, triptaz, katepsin-D gibi nötral proteazların; lökotrienler, tromboksan gibi araşidonik asit derivelerinin ve trombosit etkinleştirici faktörün lokal ve sistemik olarak salınımıdır. Kardiyak mast hücreleri koroner arterlerin intima tabakasında ve aterosklerotik plaklarda yer alırlar. Alerjik reaksiyon esnasında bu hücreler deri ve akciğerdeki mast hücreleri gibi aktive olur ve yukarıda adı geçen mediyatörleri salgılarlar. Bu mediyatörler oldukça önemli kardiyovasküler etkilere sahiptirler. Örneğin histamin koroner vazokonstrüksiyon yapar, doku faktörü sentezini arttırır ve trombositleri aktive eder.



Şekil 1. İşlem öncesi ve 48 saat sonraki EKG örnekleri.



Şekil 2. Koroner anjiografi; normal koroner arterler.

PO-049

Using the same size-balloon technique for reposition and reimplantation of inadequetely expanded and detached coronary stent

Hüseyin Katlandur, Şeref Ulucan, Hüseyin Özdil, Ahmet Keser, Zeynettin Kaya

Mevlana University Faculty of Medicine, Konya

Case Description: A 63-years- old female patient with chest pain and she had a history of type 2 diabetes mellitus. On her ECG showed normal sinus rhythm but blood troponin levels were increased steadily. Coronary angiography revealed 80% narrowing on distal segment of right coronary artery (RCA) with totally occluded posterior descending artery was very thin and small (Figure 1). Percutaneous coronary intervention (PCI) was planned on 80% RCA stenosis. The stenotic segment was predilated with 1,5*20 mm balloon catheter at 16 atmosphere. A 2,5x20 mm stent was introduced up to the stenotic segment while inflating the balloon has been ruptured without proper stent expansion (Video 1). While removing the balloon, stent was withdrawn about 3 centimeter from the its original place (Figure 2). First of all we planned snair the stent. İt was unsuccessful because of tortuosity of the vessels. A 2,5x25 mm Mini Trek balloon is used for the positioning of the stent which is same size with the stent size (Video 2). Then, the balloon was inflated up to the 2 atmospheric pressure in the attached stent. After that, the stent was captured safely and carefully advanced to desired site. Same size balloon inflated up to 16 atm and replantation was completed. The patient has been discharged two days later without any complaint.

Conclusion: Detachment of stent over the delivery system is an important and serious complication of percutaneous coronary revascularization. There are different techniques which can be applied to overcome stent detachment. Reimplantation of a detached stent with the small balloon technique has been previously described. To use same sized balloon procedure with first attempt for the retrieve and reimplantation do not described. We report a case, rupture of balloon during stenting did not deployed properly, so we used same sized balloon for reimplantation at first attempt without any complication.

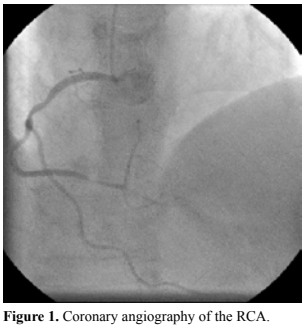


Figure 1. Coronary angiography of the RCA.

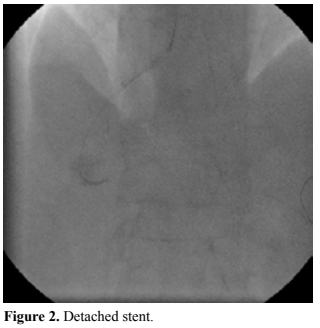


Figure 2. Detached stent.

PO-050

Right arcus aorta and Kommerell diverticulum: a rare challenge encountered as a consequence of increaesed left radial artery interventions

Ekrem Güler, Gamze Babur Güler, Türkay Sarıtaş, Hacı Murat Güneş, Bilal Boztosun

İstanbul Medipol University, İstanbul

A 64 year-old-woman was referred to our clinic for coronary angiography because of abnormal treadmill exercise test result. She had no history of cardiovascular disease. The transthoracic echocardiographic exam was normal except mild mitral regurgitation. A sheath was introduced through left radial artery. After advancing from subclavian artery to aortic arch by hydrophilic guidewire, aortic root could not be visualized although the guidewire was delivered to the location which was supposed to correspond to ascending aortic position at the right side of vertebrae. It was suprising to see the guidewire at the abdominal aortic area repeatedly without any efforts. It seemed like that we passed to inferior vena cava via an arteriovenous fistula, the unknown area was attempted to be image by catheter. It was recognised that the catheter was at the abdominal aortic position. Despite all efforts, the radial approach failed and a right femoral sheath was placed. We detected an unusual aortic anatomy by aortic root angiography performed with pigtail catheter (video 1). An aberrant left subclavian artery, Kommerell diverticulum and right aortic arch were detected. Three dimentional computed tomography images also confirmed these abnormalities (b, c, d, e,f). This rare anatomic variation is a problem for increasing numbers of left radial approach in daily practice. Awareness of this variation may help avoidance of left radial access route and performance of femoral or right radial approach to decrease the procedural duration.

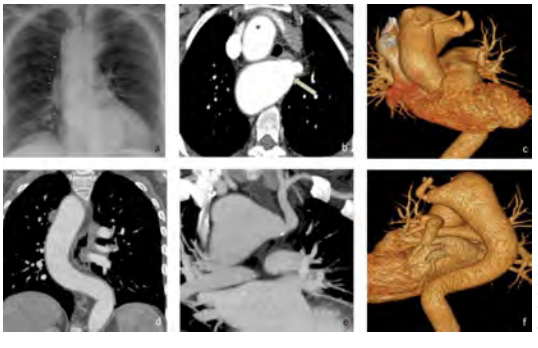


Figure 1. (A-F) Chest X ray, CT images- ascending.

PO-051

LAD'e perkütan koroner girişim sırasında söndürülemeyen non kompliyan balon; çok nadir görülen ilginç bir olgu

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Dicle Üniversitesi Tıp Fakültesi, Diyarbakır

56 yaşında erkek hasta Yatışından 2 gün önce göğüs ağrısı olan ve AKS tanısıyla yatırılan hastaya KAG yapıldı. LMCA plaklı, LAD mid bölgede %70 darlık,D3 sonrası %95 darlık CX ve RCA plaklı izlendi ve LAD PCI kararı verildi. LAD ve D3'e floopy gönderildi. LAD mid kısımdaki %95'lik darlık 2x15 mm balon ile predilate edildi. Ardından 2.5x15 mm NC balon ile 16 atm basınç ile tekrar predilatasyon yapıldı. Ancak balon indeflatör ile söndürülemedi. Defalarca denenmesine rağmen balon söndürülemedi. 50 cc lik enjektör ile balon içindeki opak boşaltılmaya çalışıldı ancak yine başarılı olunamadı. İndeflatör içindeki opak tamamen boşaltıldı ve içine mayii çekilerek balon içindeki opak seyreltilmeye çalışıldı ancak bu manevra ile de balon söndürülemedi. Sonrasında 2.0x8 mm OTW içerisinden Standart ve Miracle tellerin hem proksimal hemde distal uçları ile balon patlatılmaya çalışıldı ancak bununla da başarılı olunamadı. Bu manevradan da sonuç alınamaması üzerine balon 20 atm basınca çıkıldı ve aynı balonun daha proksimalinde aksesuar bir balonun şiştiği izlendi ve sonrasında indeflatör ile her iki balonun söndüğü izlendi. Bu işlem yaklaşık 45 dakika sürdü. Bolunun söndürülemediği süre boyunca hastanın devamlı olarak göğüs ağrısı oldu. LAD mid kısımdaki lezyona 2.5x28 Absorb eriyen stent (BVS) implante edildi. Daha proksimale 3x20 mm DES implante edildi. Absorb stent içine 2.75x15 mm NC balon ile postdilatasyon yapıldı ve işlem sonlandırıldı. İki gün sonra hasta sorunsuz olarak taburcu edildi.

Şekil 1. Balonun dışarıda aksesuar kısmı ile görüntüsü.

PO-052

Congenital ostial atresia of the right coronary artery

Veysel Tosun, Murat Bayrak, Murathan Küçük

Akdeniz University, Faculty of Medicine, Antalya

Coronary artery anomalies are a diverse group of congenital disorders whose manifestations and pathophysiological mechanisms are variable. A comprehensive review identified an incidence of coronary anomalies in 5,6% of consecutive patients undergoing angiographic study. Congenital atresia of right coronary artery is one of the intrinsic coronary artery anomalies. We reported a 78 year old male admitted to cardiology department with exertional chest pain. He was diagnosed with stable angina pectoris taking optimal medical therapy. His history showed that he underwent stent implantation in 1998. On cardiovascular examination there was a systolic ejection murmur on aortic focus. Electrocardiography showed negative T waves in D1-aVL derivations, sinus rhythm 68 beat/min. Laboratuary findings were normal. He underwent coronary angiography and asendan aortography. Aortography demonstrated atresia of Right Coronary Artery ostium (figure-1, video-1). There was 50% stenosis in distal Left Main Coronary artery (LMCA). 80% instent stenosis in proximal Left Anterior Decending (LAD) artery. 70% stenosis in proximal Circumflex (Cx) artery, 50% stenosis in distal Circumflex artery, 80% stenosis in Optus Marginalis (OM) ostium and 70% stenosis in mid Optus Marginalis. Right Coronary Artery ostium was atresic and filling with distal segment of Circumflex artery (figure-2, video-2). He was advised bypass surgery in cardiology council. He underwent to Coronary Artery Bypass Grafting operation (LIMA-LAD, Aort saphenous OM, Aort saphenous Cx grafts). He was discharged six days after surgery with optimal medical therapy.

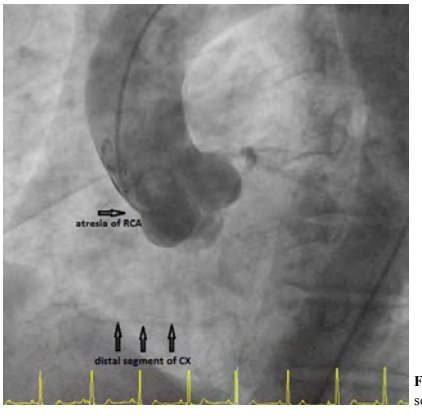

Figure 1. No RCA ostium was observed in the aortography.

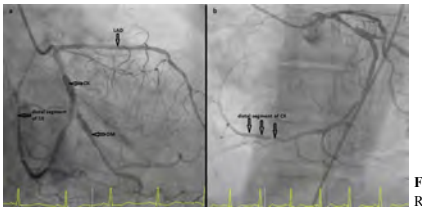

Figure 2. Distal segment of Cx feeds RCA segments.

PO-053

Spontaneous coronary artery dissection; a very rare presentation

Muhammed Keskin, Şahin Avşar, Mert İlker Hayıroğlu, Ahmet Öz, Ahmet Okan Uzun, Berat Arıkan Aydın, Edibe Betül Börklü, Mehmet Eren

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Spontaneous coronary artery dissection (SCAD) is an extremely rare but important cause of acute coronary syndrome, with only about 200 cases reported in the literature. It occurs predominantly in young women. Approximately 15% of dissections involve the left main coronary artery (LMCA). Patients with dissected proximal segments of left anterior descending (LAD), left circumflex (LCX) or right coronary artery (RCA) should be intervened percutaneously if feasible. Emergency coronary artery bypass graft surgery (CABG) should be considered if the dissection involves the left main. A 33-year-old nulliparaous woman without any risk factors for coronary artery disease presented to the hospital with sudden-onset chest pain that lasted for 2 h. There was no history of hypertension, connective tissue disease or family history of cardiac disease. Her electrocardiogram (ECG) on admission revealed ST elevation in leads I, aVL and ST segment depression in leads II,III,aVF and V1-V4. The patient was urgently taken to the cardiac catheterization laboratory. Coronary angiography demonstrated the absence of athero-sclerotic lesions; long spontaneous spiral dissection of LAD and LCX from the LMCA to the mid part of the vessels with a TIMI grade 1-2 distal flow a RCA. Troponin I levels were 1,1 ng/mL at admission and 1,4 ng/mL 2 hours after admission. Antiphospholipid and anti-cardiolipin antibodies were negative. Echocardiography showed hypokinesia of the mid lateral segments. Due to LMCA dissection and acute myocardial infarciton; urgent CABG decision made in a consultation with cardiovascular surgery. After one month from CABG, echocardiography showed normal ventricular motion. We report a very rare case with SCAD which does not have traditional risk factors; fibromuscular dysplasia, history of multiple pregnancy, connective tissue disorder, Marfan's syndrome, Ehler Danlos syndrome, cystic medial necrosis, systemic inflammation and hormonal therapy.

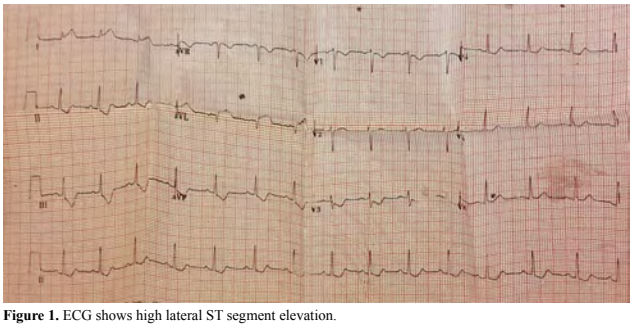

Figure 1. ECG shows high lateral ST segment elevation.

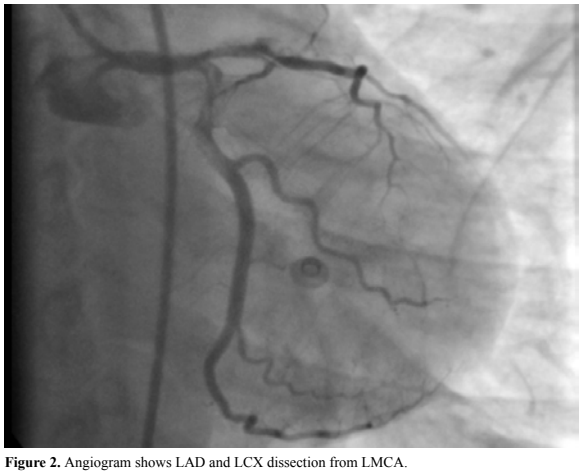

Figure 2. Angiogram shows LAD and LCX dissection from LMCA.

PO-054

The most dangerous complication of percutaneous coronary intervention: stripped stent in the left main coronary artery and its succesful treatment

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Şanlıurfa Training and Research Hospital, Şanlıurfa

We report a case of stent dislodgement in the left main coronary artery (LMCA) which was successfully treated by crushing against vessel wall using another stent. A 47-years-old male was admitted to cardiology department with unstable angina. Coronary angiogram showed a 90% stenosis of the osteal left anterior descending artery (LAD) and a 70% stenosis of the distal LAD (Figure 1, Video 1). Patient was taken for percutaneous coronary intervention (PCI) to LAD. We decided to perform szabo technique to cover LAD ostium. Through right femoral approach, 7F JL 4,0 guiding catheter was used to cannulate left coronary system. LAD and Circumflex (CX) were crossed with 0.014" floppy guidewire. The 3,0x18 mm stent (DES) was prepared outside the patient according

Girişimsel kardiyoloji / Koroner

to the szabo technique. The CX guide-wire was passed through the proximal strut of the stent. And then 3.0x18 mm DES was loaded on LAD guide-wire. The 3.0x18 mm DES was advanced across left main coronary artery (LMCA) into LAD. In spite of repeated attempts, there was resistance to advance the stent to osteal-LAD due to vessel calcification, angulations in proximal-LAD and probably direct stent strategy (without pre-dilation). It was decided to withdraw the stent. When the stent was withdrawn, it slipped from the balloon catheter completely and was floating into LMCA and proximal LAD, and all system (catheter, guide wires) failed. LAD flow was interrupted (Figure 2, Video 2); meanwhile angina and ST elevation occurred within two minutes. LAD and CX were re-wired. We attempted to retrieve stent into guiding catheter using micro snare. However, it was not being possible. We decided to crush stent against vessel wall using another stent. Respectively, LAD ostium-proximal segment was dilated with 2.0x15 mm sprinter balloon, and 3.0x18 mm DES was implanted from osteal LMCA to LAD osteal. And then stent was post-dilated with 3,5x12 mm Non-complain balloon (NC). A second 3.0x28 mm DES was implanted from proximal to mid LAD and another 3,0x15 mm DES implanted in distal LAD. LMCA and osteal-LAD were pos-dilated with 4x20 NC balloon. Final kissing dilatation was performed using the 3.5x21 mm NC balloon for LMCA-LAD and 3.5x12 mm NC balloon for CX. Final result was satisfactory and without complication (Figure 3, Video 3). In this patient, stent dislodgement may be occurred because of inadequately preparation of lesion, repeated to and fro movement of stent to cross the lesion. Several methods of retrieving dislodged stents from the coronary vessels have been described, including the use of two twisted guide wires, loop snares, small-balloon technique, and crushing deployed stent with another stent. Crushing stent technically may be easier than other techniques. Such a long stent may not be retrieved fully by inflating a small balloon. Although attempts to stent retrieve by using micro snare, it was failed. Therefore in our patient, stent dislodgment was successfully managed by using crushing technique.

PO-055

Arı sokması sonrası gelişen akut ST elevasyonlu anteroseptal miyokard infarktüsü olgusu: Kounis sendromu

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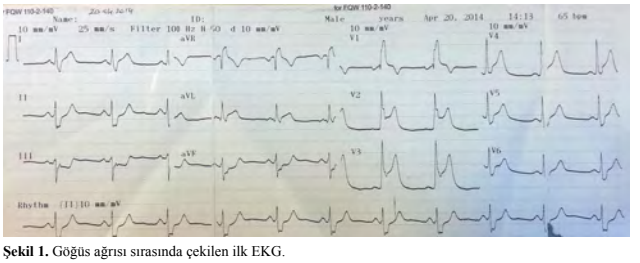
Vaka: Arı yetiştiricisi 48 yaşında erkek, çok sayıda arı sokması üzerine acil servise başvurdu. Bilinen sistemik hastalığı olmayan hastanın 30 paket/yıl sigara kullanım öyküsü mevcut. Başvuru sırasında hastanın kan basıncı 110/80 mmHg, nabız ritmik 76 v/dakika, dakika solunum sayısı 16/dakika olarak ölçüldü. Aşırı terleme, generalize hiperemi, uvulada ödem izlenen hastaya intravenöz kortikosteroid ve antihistaminik tedavi uygulandı. Daha sonra göğüs ağrısı olduğunu ifade eden hastanın çekilen EKG'sinde sağ dal bloğu V2-V3-V4 derivasyonlarında ST segment elevasyonu ve inferiyor derivasyonlarda resiprokal ST segment değişiklikleri izlendi. Hastada muhtemel tanı olarak Kounis Sendromu düşünüldü. Hastaya oral yoldan 300 mg aspirin ve 600 mg klopidogrel yükleme dozu verildi. 100 IU/kg heparin intravenöz olarak verildikten sonra hemodinami laboratuarına alınan hastaya koroner anjiografi yapıldı. LAD'nin %100 tromboze-tıkalı, CX'in normal olduğu ve RCA'da non-kritik plak olduğu görüldü. LAD'ye export xt aspirasyon kateteri ile trombüs aspirasyonu, 2,0x12 mm balon ıle dilatasyon ve 3,0x33 mm xience stent uygulandı. Yapılan ekokardiyografide sol ventrikül ejeksiyon fraksiyonu %48 apikal inferiyor duvar hipokinetik,apikal septum akinetik sol ventrikül konsantrik hipertrofik olarak izlendi. Dört gün süreyle komplikasyonsuz olarak izlenen şikayetleri tekrarlamayan hastaya aspirin 100 mg/gün, klopidogrel 75 mg/gün ve atorvastatin 40 mg/gün reçete edildi. Kardiyoloji poliklinik kontrolü ve alerjik etyoloji araştırılması için alerji bölümüne başvurması önerilerek hasta taburcu edildi.

Tartışma: Alerjik bulgulara eşlik eden göğüs ağrısı şikayetinin olması Kounis Sendromu tanısını da akla getirmelidir.



Şekil 1. Göğüs ağrısı sırasında çekilen ilk EKG.

Şekil 2. LAD %100 tromboze-tıkalı, CX normal, RCA plaklı.

PO-056

Hidden cause of acute myocardial infarction: coronary anomalies

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58 years old male patient with a cardiovascular risk factor of 35 pack-year history of smoking presented to the emergency deparment with 3 hours of chest pain. His vitals were stable including arterial pressure 123/74 and heart rate 86/min in sinus rythm. His ECG revealed ST-segment elevation in leads D1- AVL and V5-V6. After the initial evaluation he was diagnosed as acute lateral myocardial infarction and immediate primary coronary angiography was performed. Coronary angiography revealed non-ciritical stenosis in left anterior descending(LAD) and right coronary artery(RCA). Surprisingly, circumflex(CX) coronary artery could not be viewed in any standart radiological aspects. During cathaterization of right coronary artery a silhouette of an another coronary artery was seen which was totally occluded in the midportion. This one thought to be CX coronary artery which was the culprit lesion for the myocardial infarction. A floppy guidewire was passed through the lesion. Then, a baloon(Blue Medical Invader 1.1x15 mm, 2x15 mm) was advanced and inflated. After the inflation of the ballon, a stent (Xience 2.75x23) was implanted and TIMI 3 flow was established. The patient has an uncomplicated recovery. Maximal values of troponin and CPK-MB were 50 ng/ml ad 249 U/ml respectively. An transthoracic echocardiogram(TTE) was performed before hospital discharge. TTE revealed inferior and lateral hypokinesis and ejection fraction of %45 with mild mitral regurgitation. In conclusion, association of coronary anomalies with sudden cardiac death is well established, sometimes it can cause myocardial infarction with diagnostic difficulties.When a patient presented with acute myocardial infarction and coronary angiography revelaed nothing diagnostic, coronary anomalies should be kept in clinicians minds.

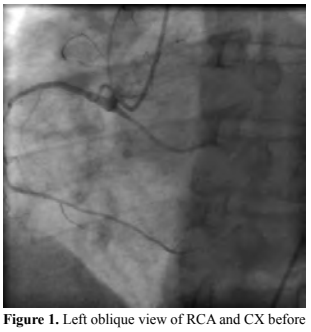

Figure 1. Left oblique view of RCA and CX before stenting.

Figure 2. Left oblique view of RCA and CX after stenting.

PO-057

Primer perkutan girişim sırasında kardiyojenik şokla karışabilen nadir bir hipotansiyon nedeni: Anaflaktik şok

Eser Açıkgöz[1], Salih Topal[1], Ramin Hacıyev[1], Özden Seçkin[1], Ayşenur Güllü[1], Sadık Kadri Açıkgöz[2], Mehmet Rıdvan Yalçın[1]

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Olgu: 52 yaşındaki erkek hasta göğüs ağrısının 1. saatinde acil servise başvurdu. Başvuru sırasında genel durumu iyi, bilinci açık, TA 180/100 mm Hg, nabız 74 atım/dk ve ritmikti. Kardiyovasküler risk faktörü olarak hipertansiyon ve sigara öyküsü mevcuttu. Akut anterolateral miyokart enfarktüsü tanısı konan hasta nazal oksijen, 5000 ünite heparin, 300 mg aspirin ve 600 mg klopidogrel verilip acil olarak kataterizasyon laboratuvarına alındı. İşleme başlandığında hastanın kan basıncı 170/90 mm Hg idi. Yapılan koroner anjiyografide 2. diagonal arter (D2) %100 tıkalı, diğer koroner damarlar açık olarak izlendi ve D2'ye perkutan koroner girişim kararı verildi. İşlem sırasında konfüzyon ve periferik hipoperfüzyon bulguları gelişen hastanın nabzının 110/dk'ya yükseldiği ve kan basıncının 80/40 mm Hg'ya düştüğü görülerek intravenöz sıvı tedavisi başlandı ve D2'ye balon dilatasyonu sonrası başarılı stent implantasyonu yapıldı. Hastada koroner vazospazm bulgusu izlenmedi. Buna rağmen tansiyon, nabız ve bilinç durumunda düzelme olmayan hastaya yapılan ekokardiyografide sol ventrikül ejeksiyon fraksiyonu %50 olarak izlendi ve miyokart infarktüsü komplikasyonu bulgusu izlenmedi. Hemodinamik bulgularının ekokardiyografik bulguları ile uyumsuz olduğu düşünülen hastaya dopamin infüzyonu başlandı. Bu sırada stridor ve solunum sıkıntısı ile birlikte yaygın ürtiker benzeri döküntülerinin ortaya çıktığı ve arteryel oksijen satürasyonunun %85'e indiği görüldü. Anafilaktik şokta olduğu düşünülen hastaya 90 mg feniramin ve 40 mg metilprednizolon intravenöz olarak verilip yoğun bakımda izleme alındı. Gerektiğinde kullanılmak üzere adrenalin hazırda tutuldu ve olası endotrakeal entübasyon ve trakeotomi ihtiyacı nedeniyle anesteziyoloji ve reanimasyon ekibi çağrıldı. Takibinde satürasyonu ve tansiyonu tedricen düzelen hastanın 15 dakika içinde stridoru kayboldu ve tansiyonu 120/80 mmHg oldu. Takibinin 4. saatinde tansiyonu 170/100 mmHg oldu ve 12 saat sonra döküntüleri tamamen düzeldi. Anaflaktik şokun, bulgularının hızlı ortaya çıkışı nedeniyle klopidogrel gibi oral ilaçlara değil, kullanılan radyoopak maddeye bağlı olduğu düşünüldü. İzleminde rutin miyokart infarktüsü tedavisi ile birlikte klopidogrel almaya devam eden hastada herhangi bir alerjik bulgu izlenmedi.

Tartışma: Akut miyokard enfarktüsü sırasında gelişen ani hipotansiyon durumunda etyolojide kardiyojenik şok, enfarktüse bağlı mekanik komplikasyonlar, aritmiler gibi nedenler hemen akla gelmektedir. Özellikle primer PTCA için koroner anjiyografi yapılan akut miyokard enfarktüsü hastalarında ani gelişen hipotansiyon ve taşikardi durumunda radyoopak maddeye bağlı gelişen anaflaktik şok da etyolojik nedenler arasında akılda bulundurulmalı ve anaflaktik şok gelişen hastalarda kalpte mast hücreleri birikimine bağlı oluşabilecek koroner vazospazm nedeniyle hastanın stabil durumunda akut kötüleşme olabileceği hatırlanmalıdır.

PO-058

Nikel allerjisi olan hastada perkütan ASD kapama

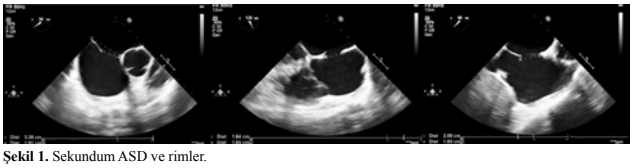
Hasan Arı, Tufan Günay, Ahmet Tütüncü, Selma Arı, Mehmet Demir, Mehmet Melek

Bursa Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Bursa

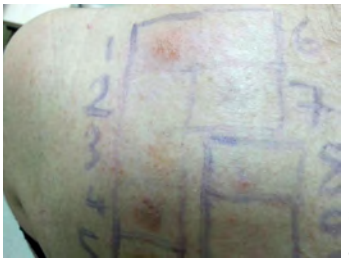
28 yaşında kadın hasta, sekundum ASD tanısı ile kliniğimize başvurdu. Yapılan TEE incelemesinde 1.9 cm sekundum ASD saptandı (Şekil 1). ASD rimlerinin yeterli olması üzerine perkütan ASD kapama işlemi önerildi (Şekil 1). Ancak hastanın metallere karşı allerji hikayesinin olması nedeni ile perkütan kapama öncesi allerji testi yapılmasına karar verildi. Allerji testi Amplatzer ve Lifetech Cera septal kapama cihazları ile yapıldı. Yama testi sonucunda Amplatzer'e karşı allerji tespit edilirken, Lifetech Cera ile allerjik reaksiyon görülmedi (Şekil 2). Hastaya antihistaminik ve steroid proflaksisi altında 30 mm Lifetech Cera septal kapama cihazı ile sekundum ASD perkütan olarak kapatıldı (video 1). Takipte komplikasyon görülmedi.

Tartışma: Kardiyolojik girişimlerde kullandığımız cihazların büyük bir bölümü çeşitli metalleri ve alaşımlarını içermektedir. Özellikle nikelençok kullanılan metallerden biridir. Toplumumuzda metal allerjisi nadir olmayan bir durumdur, bunedenle metalik bir cihaz implante edeceğimiz hastalara mutlaka metal allerjisi olup olmadığı sorulmalıdır. Nikel allerjisi kendini çarpıntı, nefes darlığı, göğüste rahatsızlık hissi ve anjiyoödem şeklinde gösterebilir. Perkütan ASD kapama uygulandıktan sonra allerjik reaksiyon nedeniyle solunum sıkıntısı olan ve medikal tedavi ile rahatlatılamayan hastalar tespit edilmiştir. ASD kapama sonrası allerjik reaksiyon nedeniyle birinci ayda cerrahi olarak cihazın çıkarıldığı vakalar bildirilmiştir.

Sonuç: Perkütan cihaz implantasyonu öncesi hastalar mutlaka metal allerjisi açısından değerlendirilmelidir.



Şekil 1. Sekundum ASD ve rimler.



Şekil 2. Yama testi: 1) Amplatzer ASD cihazı, 2) Lifetech C.

PO-059

The first Turkish patient with severe aortic stenosis and bilateral iliofemoral artery disease treated with lotus valve transcatheter aortic valve implantation

Serdal Baştuğ[1], Cenk Sarı[1], Abdullah Nabi Aslan[1], Hacı Ahmet Kasapkara[2], Hüseyin Ayhan[2], Mehmet Burak Özen[1], Ahmet Göktuğ Ertem[1], Tahir Durmaz[2], Telat Keleş[2], Engin Bozkurt[2]

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Transcatheter aortic valve implantation (TAVI) has been developed as a promising alternative to surgical aortic valve replacement for elderly high risk patients with severe aortic stenosis and severe comorbidities. TAVI is most commonly performed via the femoral artery. About one third of patients for TAVI suffer from severe peripheral artery disease making the Standard retrograde approach through the feomoral artery is not feasible. Currently, the alternatives to the femoral approach are the subclavian, the transapical and the direct aortic access. In the current case, we present the first Turkish patient with severe aortic stenosis and bilateral iliofemoral artery disease who was treated with TAVI using a Lotus Valve and subclavian approach. Case An 74 year old male patient presented with exertional dyspnea (New York Heart Association Class III) for 3 months. He had past medical history of diabetes mellitus (Type II) and hypertension. 10 year ago the patient had been treated with coronary artery bypass surgery and 2 months ago control coronary angiography performed and bypass grafts are patent. Based on the coronary angiography patient refers to our clinic for TAVI. On admission transthoracic and transesophageal echocardiography showed severe aortic stenosis with severe calcification, the aortic valve area was 0.54 cm^2. The diameter of the aortic annulus by transesophageal echocardiography was 24 mm. The mean pressure gradient across the aortic valve was estimated to be 41 mmHg. The calculated STS score was 7.6 and Eurocore was 21.4. Both common iliofemoral arteries also showed significant stenosis and severe calcification (Fig. 1). Computed tomography (CT) angiography revealed that the minimal lumen diameter of the right iliac artery was 5.6 mm and left iliac artery was 5.1 mm (Fig. 2). The diameter of the left subclavian artery was measured to be 8 mm on CT angiography. In this patient heart team decided to perform TAVI by subclavian approach, the procedure was carried out under general anesthesia. The subclavian artery exposed in the deltopectoral groove of the anterior chest wall, surgeon maked a 4 cm incision in the groove and after surgical exposure a 0.035 inch Amplatz Super Stiff wire (260 cm) was inserted into the left ventricle apex through an 20 Fr introducer sheath. 27 mm Lotus valve is constrained within a delivery catheter. After passage of the native aortic valve, the Lotus valve unsheathed to foreshorten and expand radially. The valve performance assessed before final release, the absence of major aortic regurgitation or left ventricular obstruction was controlled during valve implatation (Fig. 3). An aortogram showed good position of the Lotus valve with no aortic regurgitation. Left subclavian artery closed surgically without any complications. The post procedural echocardiography showed well functioning Lotus valve, mean pressure gradient decreased from 41 to 10 mmHg. 3 days after procedure the patient was discharged.

PO-060

Transcatheter aortic valve implantation in a patient with abdominal aortic aneurysm history previously treated by endovascular aneurysm repair (EVAR)

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[2]*Ankara Atatürk Education and Research Hospital, Ankara*

Case report: A 82 year old male patient referred to our hospital with symptoms of dyspnea due to severe aortic valve stenosis diagnosed at a local hospital. The patient had a medical history of coronary arter bypass grafting and abdominal aortic aneurysm treated by EVAR procedure (Figure 1). Other comorbidities included type 2 DM, hypertension, chronic obstructive pulmonary disease. Transthoracic and transesophageal echocardiography showed severe aortic stenosis. Diameters of the common femoral and iliac arteries were suitable for intervention. Coronary angiography revealed patent bypass grafts without any need of coronary intervention. STS score and logistic EuroScore were calculated to be 8.2% and 16.1% respectively. A multidisciplinary heart team evaluated the patient and decided that TAVI procedure is the best option for that case. Standard percutaneous access techniques were used in the left common femoral artery to obtain vascular acces for the catheter. Edwards Sapien XT 29 mm valve was implanted successfully.(Figure 2).

Discussion: Previous cardiac surgery, chronic diseases causing respiratory failure, pulmonary hypertension, porcelain aorta, previous radiation to thorax are also factors that are considered in the decision making. Interaction between the bioprosthesis valve and stent graft in abdominal aorta implanted by EVAR procedure was the main challenging factor in this case. Several reports demonstrated simultaneous implantation of transfemoral aortic valve implantation followed by endovascular repair of thoracic or abdominal aneurysms avoiding the possible interaction between bioprosthesis aortic valve and stent grafts used in endovascular repairs of aorta.

Conclusion: In this case we demonstrated that TAVI using a Edwards sapien XT balloon expandable valve can be safely performed in patients previously undergone Endovascular aneurysm repair procedure for abdominal aortic aneurysm.

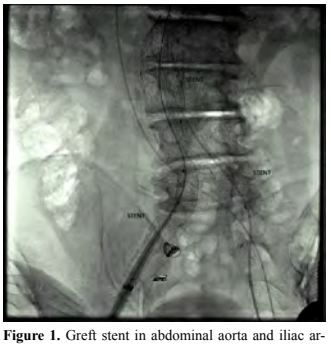

Figure 1. Greft stent in abdominal aorta and iliac arteries.

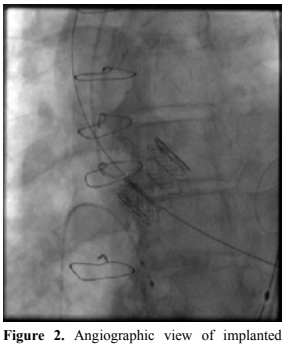

Figure 2. Angiographic view of implanted aortic valve.

PO-061

The closure of long tunnel shaped high muscular ventricular septal defect via 0.018 guiding wire

Cengiz Öztürk[1], Atila İyisoy[1], Turgay Çelik[1], Zekeriya Arslan[2], Sait Demirkol[1], Mustafa Demir[1], Ali Osman Yıldırım[1], Murat Ünlü[1], Hamidullah Haqmal[1]

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Percutaneous transcatheter occlusion techniques have been developed and used safely to close ventricular septal defect (VSD) recently in spite of difficulties. We present a 26 year-old male patient who referred to our clinic for a muscular VSD. Transesophageal echocardiography was used to define the defect size and location. A 3 mm in diameter of orifice of the defect on the left side and 4 mm on the right, which were taken in the diastole those in the largest. The defect located on high muscular septum which was very close to membranous area. Its distance from the aortic root was 10 mm. Following retrograde transaortic access to the left ventricle, VSD size and positioning were confirmed by ventriculography (Figure 1a). VSD was in tunnel type and approximately 20 mm in length. It was not able to be passed with 0,035 gliding wire; hence a 0,014 floppy wire was replaced. This thinner wire was caught with a snare wire sent through the right femoral vein in the RV (Figure 1b); but the wire couldn't be pulled. Avoiding from breaking the wire, the procedure was repeated with a thicker 0,018 wire and it was accomplished (Figune 1c). The 0,018 wire was advanced by making loop with the aid of a diagnostic catheter. Stif wire was then advanced into the RV by being made a loop and passed through VSD. An appropriate muscular VSD closure device was positioned and released through the carrying system; the distal part was opened, and the proximal part was lengthened inside the defect (Figure 1d). The device was tried to be formed by pulling back, but not accomplished. The proximal part of the device stayed and settled in the tunnel. The device was left in this position after that a control ventriculography showed no residual shunt and the echo showed the device fully located inside the defect (Figure 2). That the device was to be stuck in a long tunnel type defect which was passed by only 0,018 wire with looping was intriguing point in this case.

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalığı

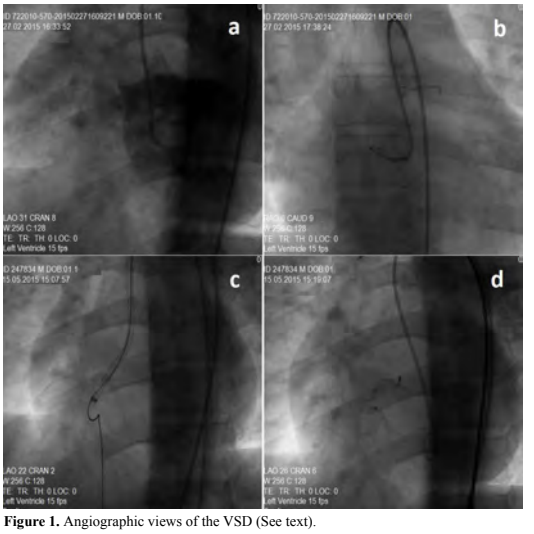

Figure 1. Angiographic views of the VSD (See text).

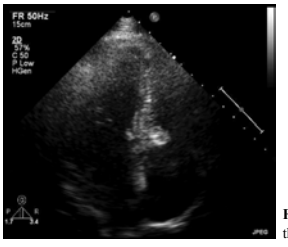

Figure 2. The device fully located inside the defect.

PO-062

Ventricular embolization of two Edwards SAPIEN XT prosthetic valves during trans-subclavian aortic valve implantation

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A 61 year-old women was taken to the catheterization laboratory and TAVI was performed under general anesthesia via left subclavian artery. A 26 mm Edwards Sapien XT valve (Edwards Life-Sciences, Irvine, CA, USA) was carefully introduced and retrogradely deployed with rapid pacing under angiographic and flouroscopic guidance over the extra stiff wire. Immediately after the valve deployment, aortography was performed and demonstrated moderate to severe paravalvular aortic regurgitation and slightly lower position of the prosthesis (Figure 1A). Therefore, postdilatation was performed with the prosthetic balloon that was inflated 1 cc more (Figure 1B). However, there was not observed marked decrease in the degree of paravalvular leak. As the heart beat, the prosthesis gradually displaced downward into the left ventricular outflow tract (LVOT) and ultimately embolized into the left ventricle. After that, the cardiovascular surgeons were informed about the condition to immediately take the patient for open heart surgery. Just after the valve embolization, hemodynamic instability was ensued. In this time, we first tried to take the embolized valve from the left ventricle by inflating the balloon of a second valve through it (Figure 2A). However, we could not succeed. Then, a second 26 mm Edwards SAPIEN XT valve was transmitted to the aorta through subclavian artery and deployed (Figure 2B). However, after implantation second valve was also embolized into the left ventricle rapidly (Figure 2C). Patient was treated by surgical removal of both valves, valves was removed from pigtail by hands and crushed (Figure 3) and a 21 mm mechanical valve (St-Jude Medical, St Paul, MN, USA) was replaced. At the end, the patient was discharged from hospital 9 days after the surgery.

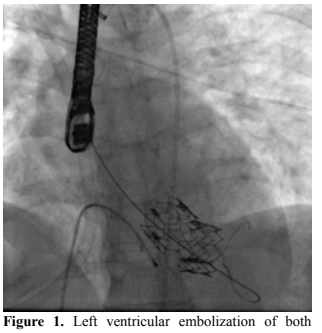

Figure 1. Left ventricular embolization of both valves.

Figure 2. Two Sapien XT valves extracted with surgery.

PO-063

Transcatheter aortic valve-in-valve implantation in lower-lying coronary ostium

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Transcatheter aortic valve implantation (TAVI) has emerged as an alternative to surgical aortic valve replacement for patients with symptomatic severe aortic stenosis considered to be at very high or prohibitive operative risk. Nowadays, despite the TAVI holds great promise for treatment of the severe aortic stenosis, many technological difficulties and limitations still exist. The potential risk for life-threatening complications such as coronary occlusion is still regarded. Many studies reported the impairment of the coronary blood flow was mainly due to the position of the coronary orifice and the close relationship with the aortic leaflets or valve stent during the TAVI. In this case report, 23 mm Edwards Sapien XT prosthesis was planned to implant a 80-year-old woman who has symptomatic severe aortic stenosis with low coronary artery distance. We advanced a bare-metal stent via guidewire into the left anterior descending artery (LAD) before the prosthesis deployment in case of occurrence of coronary obstruction (Figure 1). The position of the prosthesis was assessed by aortography an on the appropriate position and then prosthesis was implanted. Unfortunately, due to migration of first valve into the sinus of valsalva and occurring severe paravalvular leakage after implantation, a second prosthesis which is the same size with the first one was implanted into the first prosthesis on appropriate position, successfully. The coronary flow was seen continued by aortography, finally. The possible reason of continuing coronary flow after the first THV might be coronary ostia lateral to the stent frame or there might be flow laterally from the open sinuses (Figure 1). We have interested in this case that it should be a good pre-procedural evaluation and should be taken an implement additional security measures during the TAVI procedure for avoidance of coronary obstruction when performing TAVI in patients with borderline valve to coronary ostia distance.

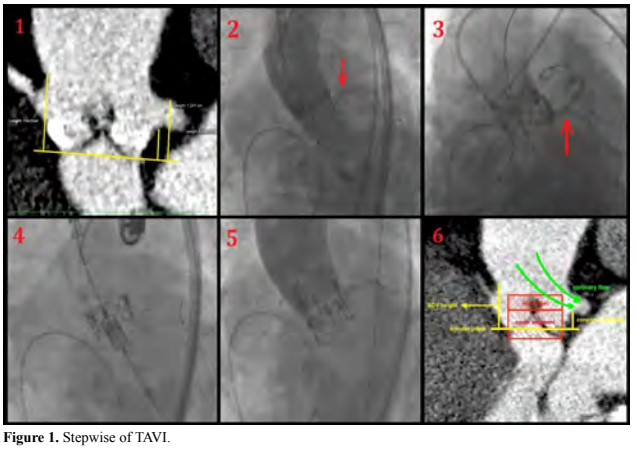

Figure 1. Stepwise of TAVI.

PO-064

Mitral anulüs kalsifikasyonuna bağlı ileri derece mitral yetersizliği olan bir hastada MitraClip ile başarılı perkütan mitral kapak tamiri

Sina Ali[1], Mehmet Bilge[2], Ayşe Saatçi Yaşar[1], Yakup Alsancak[1], Büşra Çolak[1], Mehmet Erdogan[1], Bilge Duman Karaduman[1], Muhsin Beyatlı[1]

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Giriş: Mitral anuler kalsifikasyon (MAK), özellikle yaşlı kadınlarda gözlenen ve mitral kapağın daha çok posteriyor anulüsunu etkileyen kronik dejeneratif bir patolojidir. Bununla beraber, kronik böbrek yetmezliği olan hastalarda daha genç yaşlarda gözlenebilir. Kapağın etkilenme derecesine bağlı olarak yetersizlik (MY) ya da darlık meydana gelebilir. Ciddi MY olan hastalarda tercih edilen tedavi yöntemi açık kalp cerrahisidir. Son yıllarda ciddi dejeneratif veya fonksiyonel MY bulunan ve açık kalp cerrahisi için yüksek riskli olarak değerlendirilen hastalarda MitraClip alternatif bir tedavi biçimi olarak kabul edilmektedir. Bununla birlikte MAK'a bağlı ileri MY olgularında MitraClip uygulanımı henüz bildirilmemiştir. Bu bildiride MAK'a bağlı ileri MY olan bir olguda başarılı Mitraclip uygulanımını sunmaya çalışacağız.

Olgu Sunumu: Dış merkezde hipertansiyon, diyabet, kalp yetersizliği ve kronik böbrek yetmezliği nedeniyle takip edilen 60 yaşında kadın hasta ileri MY nedeniyle kliniğimize yönlendirildi. Hastanın anamnezinden uzun yıllardır sistemik lupus eritematosus nedeniyle takip edildiği ve tekrarlayan kalp yetersizliği atakları nedeniyle defalarca hastanede yatış öyküsü olduğu öğrenildi. NYHA göre fonksiyonel kapasitesi sınıf III olarak değerlendirildi. Yapılan transtorasik ekokardiyografisinde ejeksiyon fraksiyonu %30 idi. Aynı zamanda mitral kapakta ileri derecede MAK ve buna bağlı olarak ileri derecede MY olduğu gözlendi (Video 1 ve 2). MAK'a bağlı olarak hafif derece mitral darlık da izlendi (kapak alanı 3.1 cm^2, ortalama 3 mm Hg gradient). Yapılan transözofajiyal ekokardiyografide A2-P2 skalloplarından ileri derecede MY saptandı. Ayrıca MAK'ın posteriyor yaprakçığa sınırlı olduğu ve posterior yaprakçığın sadece 7 mm'lik uç kısmının kalsifik olmadığı gözlendi (Video 3-4). Hastanın EuroScore %15 olup, cerrahi için yüksek riskli olduğuna karar verildi. Hastanın semptomatik olması nedeniyle MY giderilmesi için MitraClip uygulanması planlandı. Tek bir klip ile hastaya başarılı bir şekilde MitraClip uygulandı (Video 5,6). Tabur culuk öncesinde mitral kapak üzerinde ortalama 4 mmHg gradient izlendi. İşlemden 3 ay sonra yapılan kontrol ekokardiyografisinde ise ortalama 7-8 mmHg gradient izlendi.

Tartışma: MAK'a bağlı ileri MY olan hastalarda standart tedavi yöntemi mitral kapak replasmanıdır. Ancak bazen ileri yaş ve eşlik eden komorbiteler nedeniyle cerrahi imkansız olabilir. İyi seçilmiş olgularda MitraClip ile tedavi iyi bir seçenek olabilir. Ancak, bu olgularda özellikle posteriyor yaprakçığın kısa olması işlemi zorlaştırabilir. Diğer taraftan gelişebilecek mitral darlığı yönünden de dikkatli olunmalıdır. Sunduğumuz bu vaka; MAK'a bağlı ileri MY olgularında, MitraClip tedavisinin uygulanabilirliği göstermesi açısından önemli olabilir.

PO-065

Kalp transplantasyonu planlanan ve ciddi mitral yetersizliği olan bir hastada köprüleme tedavisi olarak MitraClip

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Giriş: Kardiyak transplantasyon kalp yetersizliği (KY) olan semptomatik, seçilmiş hasta grubunda kabul gören tedavi seçeneğidir. Diğer taraftan konjestif KY ile takipli olan hastaların yaklaşık %15-30'unda orta yada ciddi mitral yetersizliği (MY)vardır. Ciddi MY varlığı, KY hastalarında klinik durumu daha kötü hale getirebilmektedir. Son zamanlarda cerrahi operasyon riski yüksek olan MY hastalarında klinik kullanımı giderek yaygınlaşan MitraClip tedavisinin, MY derecesini azaltmada etkin ve güvenilir bir yöntem olduğu gösterilmiştir.

Olgu Sunumu: Dış merkezde uzun süredir noniskemik dilate kardiyomiyopati tanısı ile takip edilen 41 yaşında erkek hasta, ileri MY saptanması üzerine kliniğimize yönlendirildi. Hastanın ara ara KY ataklarından dolayı yatırılarak tedavi gördüğü öğrenildi. Haziran 2012'de yapılan koroner anjiyografisinde önemli darlık olmadığı gözlendi. KY için optimal tedavi almasına rağmen hastanın NYHA'ya göre sınıf 2 olduğu saptandı. Kliniğe kabülünde yapılan ekokardiyografisinde diyastol sonu çapı 6.0 cm, sistol sonu çapı 5.4 cm, sistolik pulmoner arter basıncı 50 mmHg ve ejeksiyon fraksiyonu %27 olarak saptandı (Video 1). Bununla birlikte A2-P2 skallopundan kaynaklanan ileri derecede santral MY saptandı (Video 2,3,4,5). Hastanın semptomatik olması ve tekrarlayan KY atakları sebebiyle, kalp damar cerrahisi bölümü ile birlikte değerlendirilerek hastaya kardiyak transplantasyon planlandı, aynı zamanda hastaya köprüleme tedavisi olarak MitraClip uygulanmasına karar verildi. Haziran 2012'de, A2-P2 skallopuna iki klip yerleştirilip, MY <2 elde edilerek işlemden çıkıldı (Video 6,7). Hastanın NYHA fonksiyonel kapasitesi sınıf 1 düzeyine kadar düzeldi. Altıncı ayda yapılan kontrollerinde tek klibin mitral yaprakçıktan ayrıldığı gözlendi, semptomatik olmayan hastanın MY oranında artış izlendi. 1 yıl boyunca takiplerinde hastanın fonksiyonel kapasitesinde gerileme izlenmedi. Araya giren enfeksiyon sonrası dış merkezde, medikal tedaviye rağmen hastane içerisinde KY semptomları gelişen hasta, tekrar Kalp Damar Cerrahisi Bölümü ile değerlendirilerek acil transplantasyon programına alındı. Hastaya, MitraClip işleminden tam 2 yıl sonra başarılı kalp transplantasyonu gerçekleştirildi. Operasyon sonrasında mitral kapağa implante edilen klipin A2-P2 skallopundaki yerleşimi makroskopik olarak değerlendirildi ve klipin mitral kapak uçlarını sıkı bir şekilde tuttuğu gözlendi (Şekil 1,2,3). Hasta transplantasyon sonrası sağlıklı bir şekilde taburcu edildi.

Sonuç: Özellikle son dönem KY hastalarında eşlik eden ciddi MY, semptomların kontrol altına alınmasını zorlaştırmaktadır. Burada sunulan hasta gibi son dönem KY olup semptomların kontrol altına alınamadığı ve kalp transplantasyonu sırasını bekleyen hastalarda, MY'nin düzeltilmesi için MitraClip yönteminin kullanılması etkin bir yöntem olabilir. Ancak bu konuda daha ileri düzeyde değerlendirmelere ihtiyaç vardır.

PO-066

Sudden cardiac arrest during treadmill test secondary to slow coronary flow

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The coronary slow flow phenomenon is characterized by delayed opasification of vessels in the absence of any evidence of obstructive epicardial coronary disease. The clinical features of the patients with slow coronary flow is extensive. Here we discuss 41 years-old man with sudden cardiac arrest during treadmill test resuscitated for five minutes. The patient with no history of chronic disease was taken to cathetery laboratory after successfull resuscitation. Nevertheless, new left bundle branch block developed after the resuscitation. Despite slow coronary flow in the right coronary artery angiographically normal coronary arteries were seen. No more pathology was detected to explain the sudden cardiac arrest rather than slow coronary flow in the right coronary artery. Sudden cardiac arrest during treadmill test has not been previously reported in slow coronary flow patients out of our case.

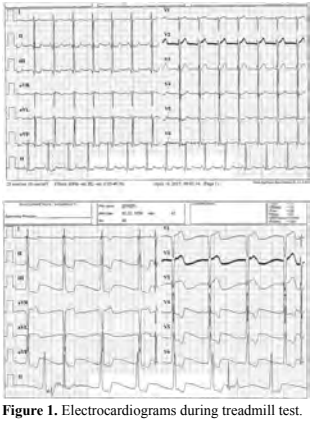

Figure 1. Electrocardiograms during treadmill test.

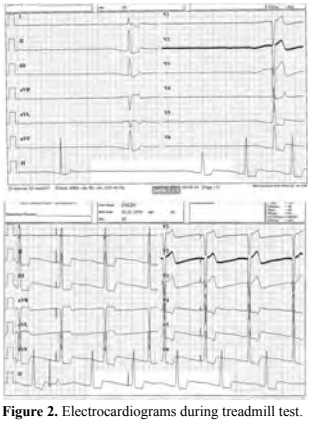

Figure 2. Electrocardiograms during treadmill test.

PO-067

Familial mediterranean fever and non-ST-segment elevation acute coronary syndrome secondary to massive coronary thrombus

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[3]*Mevlana University Faculty of Medicine, Konya*

Case: A 28-year-old man was admitted to the emergency department with complaint of acute onset precordial chest pain for duration of two hours. He was diagnosed with Familial Mediterranean fever (FMF) on the basis of Tel-Hashomer criteria before one year. He was on colchium treatment 1.5 gr. per day orderly. He does not describe any similar chest pain during previous FMF attacks. Cardiovascular risk factors, such as smoking and family history, of coronary artery disease were all negative. He did not use any prothrombotic drug. There was no history of substance or drug abuse. The arrival ECG was sinus rhythm and T waves were negative in precordial leads. His chest pain did not improve with anti-ischemic therapy and coronary angiography (CAG) was decided. CAG showed a normal coronary vasculature (Figure 1). His pain was gradually decreased and ECG changes were improved on the same day after the medical treatment. The patient was evaluated by rheumatology for FMF and his medical therapy was managed. The patient had a severe chest pain on the sixth day of hospitalization. His pain was intense and in precordial crushing sensation and was accompanied with negative T waves in precordial leads. Troponin I was slightly elevated. The patient was taken to invasive laboratory immediately because of ongoing angina and dynamic ECG changes. CAG showed a massive intracoronary thrombus that causes 70% occlusion of LAD after DII (Figure 2). The patient was followed up in intensive care unit with optimal medical therapy. Cardiac enzymes returned to normal after a mild increase. His chest pain and ECG changes improved completely on the second day of angiography.

Conclusion: This case demonstrated that inflammatory diseases, such as FMF, may lead to coronary thrombosis and acute coronary syndrome without evident atherosclerotic lesion. Patients with inflammatory diseases should be evaluated carefully for the decision of CAG.

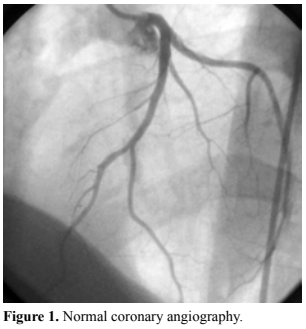

Figure 1. Normal coronary angiography.

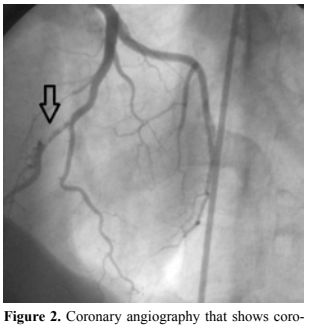

Figure 2. Coronary angiography that shows coronary thrombus.

PO-068

Can extremely overt hypothyroidism cause acute coronary syndrome in a patient with normal coronary arteries?

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A 39-year-old male patient applied to emergency department with squeezing chest pain. Electrocardiography demonstrated slight ST segment depressions on anterior leads that resolved after sublingual nitrate. The patient was hospitalized to coronary care unit and administered ASA, clopidogrel, rosuvastatin, ramipril and enoxaparin. Any beta-blocker was not administered owing to bradycardia. His medical history was unremarkable except history of smoking of 5 years. He quitted smoking two years ago, however, he started to smoke and smoked two packages of cigarette on the day of hospitalisation. The patient denied any illicit drug abuse. Troponin I levels increased up to 28,4 ng/ml and echocardiography revealed normal left ventricular function. Contrary to these findings, TSH was ascertained as 231 µU/ml with low fT3 and fT4 levels. The patient was consulted to endocrinology department and levothyroxine was initiated and titrated up to 150µgr throughout six weeks. After normalisation of TSH levels, coronary angiography demonstrated normal coronary arteries and the patient was prescribed ASA and clopidogrel merely. T3 exerts its cellular actions through binding to thyroid hormone nuclear receptors. It causes decreased systemic vascular resistance via a direct effect on vascular smooth muscles of peripheral arterioles. It is not well-known whether it strongly affects coronary smooth muscles, however, extremely overt hypothyroidism may have an anti-relaxant effect on coronaries and coronary vasospasm might be induced more easily if smoking accompanies extreme hypothyroidism as suppossed to be in our case. We speculate that extremely overt hypothyroidism may enable coronary vasospasm in the presence of other inducers such as smoking, illicit drug abuse, alcohol withdrawal, exposure to cold or severe emotional stress. Our case had a history of intense smoking at the day of ACS. Therefore, smoking and extremely overt hypothyroidism could be the underlying etiology of acute coronary syndrome in our case. In the medical literature, it was demonstrated that hypothyroidism leads to atherosclerosis through diastolic hypertension and increased hypercholesterolemia. However, to the best of our knowledge, myocardial infarction due to extremely overt hypothyroidism in a patient without atherosclerotic coronary arteries has never been published in English medical literature before.

PO-069

İnferior ST elevasyonlu miyokard enfarktüsü ve kardiyojenik şoktaki hastada yaygın koroner spazm

Hasan Çağlayan Kandemir, Fatih Mehmet Uçar, Okan Er, Musa Şanlıalp, Fatma Esin

Denizli Devlet Hastanesi, Denizli

Giriş: Miyokard enfarktüsü oldukça ölümcül seyreden bir klinik durumdur. Beraberinde kardiyojenik şok tablosunda olan hastalarda mortalite oranı çok daha yüksektir. İşlemin daha hızlı yapılması ve koroner arterlerde saptanan darlıkların açılması hasta için hayat kurtarıcıdır.

Vaka: 65 yaşında daha öncesinde koroner arter hastalığı hikayesi olmayan hasta, 3 saat önce başlayan göğüs ağrısı ve nefes darlığı şikayeti ile acil servise 112 ekibi tarafından getirildi. Hastanın acil servise kabulünde TA:70/40 mmhg ve Kalp hızı 50 atım/dk olarak saptandı. Elektrokardiyografisinde İnferior duvar miyokard enfarktüsü tanısı alan hasta katater laboratuarına alındı (Şekil-1). Yapılan sol koroner anjiyografi de LAD proksimal bölgeden mid bölgeye uzanan ciddi darlık ve CX koroner arterde subtotal tıkalı izlendi (Şekil-2). Sağ koroner anjiyografide ise RCA sağ ventrikül dalı hizasında total tıkalı izlendi (Şekil-3). Hasta için öncelikle cerrahi ihtimali düşünüldü ancak acil cerrahi mortalitesi çok yüksek olacağı ve cerrahi hazırlık süreci de dikkate alınarak girişim yapılması kararlaştırıldı. Hastanın şok tablosunda olması ve inferior derivasyonlarda ST yükselmesi olması üzerine öncelikle RCA girişim planlandı. RCA kılavuz tel ile geçilmeye çalışıldı ancak başarılı olunamadı balon desteği ile kılavuz tel ilerletildi ve balon yapıldı ancak çok zayıf bir akım izlendi. Ardından sol koroner anjiografi tekrarlanarak girişime uygun olan bir lezyon alanı saptanmaya çalışıldı. İkinci defa sol koroner pozlar tekrarlandığında ilk görüntülerin aksine LAD ve CX koroner arterlerin çaplarının oldukça arttığı ve sadece CX te orta bölgede bir lezyon olduğu görüldü. Bu sırada hastanın TA sı daha iyi seviyelere ulaştı ve hastanın kliniğinden yaygın koroner vasospazmın sorumlu olduğu anlaşıldı (Şekil-4). CX orta bölge lezyona stent takıldı ancak hem kılavuz tel geçilirken hem de stent implante edilirken hastada koroner spazmlar tekrarladı ve hasta hipotansif olmaya devam etti. Hastanın hipotansif olması nedeniyle hastaya nitrat ya da kalsiyum kanal blokörü verilemedi. İntraaortik balon pompası takılarak hasta yoğun bakım ünitesinde takibe alındı.

Tartışma: Koroner vazospasm epikardiyal koroner arterin anormal kontraksiyonu sonucu oluşmaktadır ve miyokardiyal iskemiye neden olmaktadır. Patogenezinde koroner aterom ve endotelyal disfonksiyon sorumlu tutulmaktadır. Ayırıcı tanıda intravenöz ya da intra arteriyel nitrogliserin uygulanmaktadır. Hastamızda olduğu gibi kardiyojenik şok tablosuna neden olabilecek yaygın vazospasm nadir bir durumdur ve bu hastalarda hipotansif olmalarına rağmen nitrogliserin kullanımı ve İntraaortik balon kullanımı tartışmalıdır.

PO-070

Asymptomatic large left ventricular pseudoaneurysm and inferior notched Q waves on ECG

Yusuf Cemil Gürsoy, Ziyaeddin Aktop, Belma Kalaycı, İbrahim Akpınar, Muhammet Raşit Sayın

Bülent Ecevit University, Faculty of Medicine, Zonguldak

A 71-year old asymptomatic male patient was evaluated preoperative assessment with history of diabetes and smoking habit. There was no significant finding on FM. ECG showed findings of sinus rhythm, notched Q waves observed inferior from previous inferior myocardial infarctus (MI) findings and incomplete left branch block (Figure 1). (Figure 1). The echocardiography revealed that a large pseudoaneurysm (PA) of 52x76 mm containing organized thrombus related to a 2.3 cm defect posterior-inferior and neighboring the lateral wall basal segment (video 1-2-3). Examined cardiac markers were negative. On coronary angiography and left ventriculography, a PA sac showing excess contrast fill was observed (Figures 2-3). The decision to operate was made but the patient refused. Left ventricular PA generally form after MI or cardiac surgery and free wall rupture is limited by the pericardium and thrombus. In patients with left ventricular pseudoaneurysm monitored conservatively with no operation, the mortality rate at the end of the 1st week is 50%. In addition, of 31 patients not operated on in a series of 290 patients monitored for PA; at the end of the first year 12, at the end of 5 years 5 and at the end of the 10th year 2 patients had survived. Additionally it is reported that PA can spontaneously close. The majority of PA's show as non-specific ST/T changes or ST elevation on EKG, but rarely may appear normal on ECG. We did not find any reference to the "inferior notched Q wave" observed in our case in the PA cases in the literature. PA structurally disrupts ventricular communication and this may cause the ECG changes. As we have insufficient data, possible PA diagnosis may be considered in patients with this rare finding. In suspected cases, advanced investigations such as cardiac MR/CT may be planned.

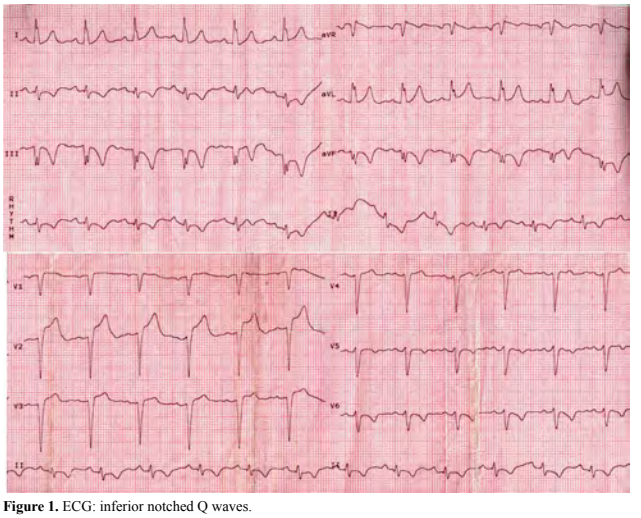

Figure 1. ECG: inferior notched Q waves.

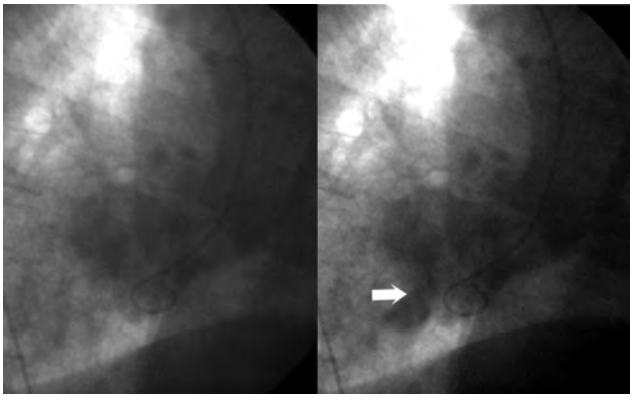

Figure 2. Left ventriculography.

PO-071

Acute aortic dissection case mimicking the acute coronary syndrome

Lütfü Bekar, Volkan Camkıran, Adem İlkay Diken, Adnan Yalçınkaya, Oğuzhan Çelik, Mücahit Yetim, Tolga Doğan, Osman Karaarslan, Yusuf Karavelioğlu

Hitit University Medicine Faculty, Çorum

65 age old female patient was admitted to the emergency service due presyncope and fatigue. The patient had no history of chest pain or shortness of breath in this period. Blood pressure and heart rate of the patient were 80/30 mmHg and 95 beats per minute, respectively. On electrocardiogram, common ST depression and ST elevation on AVR derivation were present (Figure-1). Optimal evaluation could not be performed due to inadequate echogenicity in the transthoracic echocardiographic assessment. Acute pathology was not detected in the cranial computerized tomography. Coronary angiography was performed in the patient and coronary vessels were monitored as normal. Then, aortogram was performed in the patient and intimal flap was monitored in ascending aorta (Figure-2). The patient was operated by the department of cardiovascular surgery with the diagnosis of acute aortic dissection, but the patient died intraoperatively.

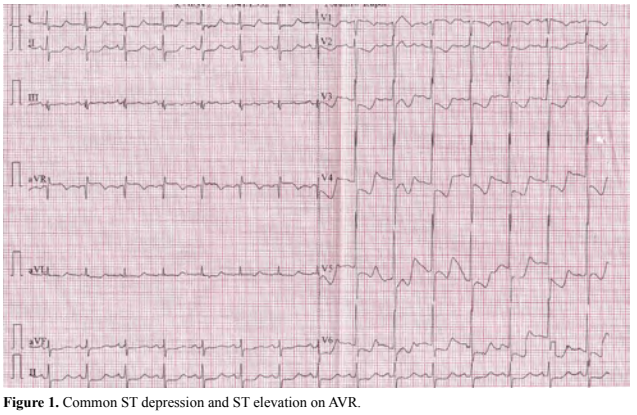

Figure 1. Common ST depression and ST elevation on AVR.

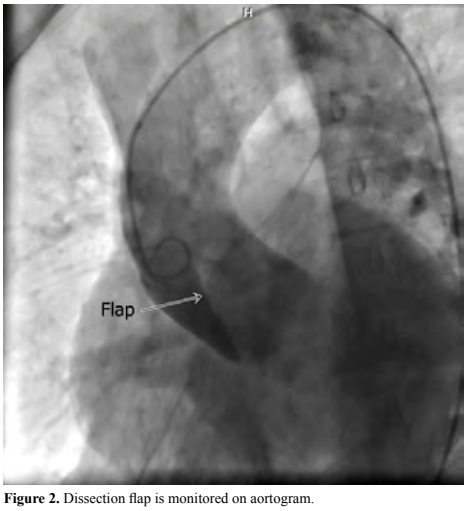

Figure 2. Dissection flap is monitored on aortogram.

Koroner arter hastalığı / Akut koroner sendrom

PO-072

Hyponatremia induced acute coronary syndrome: is it realistic?

Çağın Mustafa Üreyen[1], Görkem Kuş[1], Şakir Arslan[1], Göksel Çağırcı[1], Cem Yunus Baş[1], Sibel Üreyen[2], Murat Esin[1], Emre Asiltürk[1], Erkan Köklü[1], Gülsüm Meral Yılmaz[1]

[1]*Antalya Education and Research Hospital, Antalya*
[2]*Akdeniz University, Faculty of Medicine, Antalya*

A 69-year-old woman admitted to our emergency service with compliants of nause, vomiting, confusion and she had severe diarrhea for one week. Medical history was unremarkable except hypertension that was regulated with amlodipin 10 mg. Her blood pressure and pulse were 110/60 mmHg and 73/min, consecutively.Cardiac examination was normal. Positive laboratory results were as follows: Na level was 101 mmol/l, K level was 3.1 mmol/l, Mg level was 1.6 mmol/l and troponin I was 17.68 ng/mL. Electrocardiography (ECG) denoted ST depressions in leads V1-V6. The patient was hospitalized to coronary care unit and consulted to nephrology department. The patient was administered ASA, clopidogrel, metoprolol succinate, atorvastatin, enoxaparin and %3 NaCl infusion. During NaCl treatment, the serum sodium was not allowed to rise by more than 8 mmol/l over 24h. The blood sodium level was monitored daily. In the fifth day of patient's admission, serum sodium level was 132 mmol/l. Coronary angiography revealed a plaque in proximal LAD; whereas the circumflex artery and RCA were normal. The patient was diagnosed as ACS due to positive troponin I and ST depressions on anterior leads of admission ECG. Amlodipin was the only drug that the patient was taking. She did not have any history of recent upper tract infection or any anginal pain before. Hypokalemia and hypomagnesemia are known to cause coronary vasoconstriction and there are case reports regarding their vasoconstrictor effect. However, our patient had mild hypokalemia and hypomagnesemia, albeit very severe hyponatremia. In English literature, there is not any case report regarding coupling of severe hyponatremia and acute coronary syndrome.Thus, we could not correlate ACS with severe hyponatremia, firstly. However, during literature searching, case reports and articles regarding hyponatremia and cerebral vasospasm cooccurrence were ascertained. Patients with subarachnoid hemorrhage and hyponatremia had more cerebral vasoconstriction. These articles could not explain the mechanism of cerebral vasospasm enhanced by hyponatremia, however, they emphasised this interesting cooccurrence. The patient's ECG denoted ST depressions on anterior leads of admission ECG that demonstrates ischemia of LAD region. Potassium and magnesium levels were mildly low that is not expected to result in severe coronary vasoconstriction in our patient, but still they may ease vasoconstriction in the presence of a more potent vasoconstrictor event. We speculated that very severe hyponatremia might lead to coronary vasoconstriction as it probably caused vasoconstriction in SAH and hyponatremic patients. We can not declare very severe hyponatremia as the exact cause of ACS in our patient, however, we could not explain the pathophysiology of ACS in our patient. Moreover, this article would probably draw attention to the possible cooccurrence of very severe hyponatremia and ACS and would probably lead to studies that investigates the possibility of this cooccurrence.

PO-073

Is anticoagulation required for giant coronary arteries: an irritating dilemma

Çağın Mustafa Üreyen[1], Cem Yunus Baş[1], Şakir Arslan[1], Göksel Çağırcı[1], Sibel Üreyen[2], Görkem Kuş[1], İsa Öner Yüksel[1], Gülsüm Meral Yılmaz[1], Deniz Demirci[1], Duygu Ersan Demirci[1]

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A 54-year-old patient was referred to emergency department with squeezing chest pain that continued for 30 minutes. Electrocardiography denoted sinus rhythm witout any ischemic signs.However, ECG taken in the refrerred center showed mild ST segment depressions at derivations of V1-4. Troponin I, which then increased up to 10.3 ng/dl during hospital stay, was ascertained 0.984 ng/dl and, hence, the patient was hospitalized to coronary care unit. On questioning,he had no atherosclerotic risk factor except a positive family history. He denied any illicit drug abuse or smoking. Echocardiography revealed normal findings other than borderline left ventricular hypertrophy.ASA 100 mg, ticagrelor 90 mg bid, metoprolol succinate 100 mg, atorvastatin 40 mg, ramipril 10 mg and enoxaparin 0.8 cc bid were administered. The patient refused cardiac catheterization initially, however, he gave approval for coronary angiography on the fourth day of hospitalization.Coronary angiography revealed very large coronary aneurysms of LAD and RCA (Figure 1-2) as well as coronary ectasia of circumflex artery. The blood flow through aneurysmatic coronaries was turbulent, albeit any fresh thrombus could not be demonstrated that might be potentially due to antiaggregant and anticoagulant therapy. At that point,what should we do after coronary angiography without any significant coronary lesions other than very large coronary aneurysms? We planned to administer ASA and ticagrelor initially for one year according to guidelines of NSTE-ACS.However, we could not decide what to administer after one year; ASA alone or warfarin alone therapy. Some of our colleagues supported to give ASA alone due to absence of demonstrated coronary thrombus, while others advocated to anticoagulate with warfarin after one year.Moreover,they stated that the most probable mechanism of ACS in our patient was thrombosis and embolisation to distal vessel. As a conclusion, we could not take a joint decision.What do you recommend: ASA or warfarin?

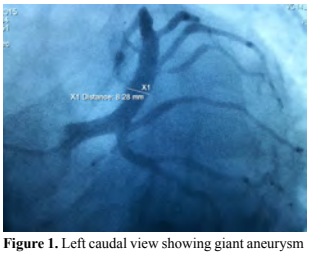

Figure 1. Left caudal view showing giant aneurysm of LAD.

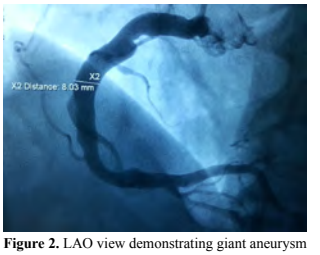

Figure 2. LAO view demonstrating giant aneurysm of RCA.

PO-074

Coronary balloon deflation during percutaneous aortic stenting in a patient with aortic interruption

Atila İyisoy, Turgay Çelik, Cengiz Öztürk, Sait Demirkol, Mustafa Demir, Ali Osman Yıldırım, Mehmet Ali Şahin

Gülhane Military Medical Academy (GMMA) Ankara, Ankara

A 25-year old male symptomatic patient was referred to our clinic with the diagnosis of coarctation of the aorta. There was an marked systolic murmur on interscapular area. The arterial blood pressure of both arms was high (Right arm: 180/100 mmHg and left arm:170/90 mmHg). The pulses on lower extremity were weak. There was90 mmHg systolic gradient was detected between proximal and distal part of the descending aorta. On CT angiography, there was current aortic interruption on proximal part of the descending aorta. The interrupted segment was approximately 1,5 cm distal from left subclavian artery orifice. Approximate length of interruption segment was 1,5 mm. Before and after interruption, the diameters of aorta were between 7 and 15mm on about 1,5 cm area. Both radial and the right femoral artery catheter were inserted. Images were taken from the proximal of interrupted segment and distal (Fig-1). Before the operation, systolic blood pressure on proximal of interrupted segment was 190 mmHg. With the help of right guide catheter sent over left radial artery, 0,014 floppy wire was pushed on from interrupted segment. Firstly, it was dilated with a 2,0X20 mm balloon. Then, it was dilated with a 3,5x20 mm balloon. However, 3,5 mm balloon couldn't be deflated with the help of indeflator inflation device (Fig-1). It was retried with 50cc-injector but, we could not success, the balloon was exploded with wire's sharp part and pushed to distal by forwarding the back side of 0,014 floppy wire through the guide catheter (Picture-3). After seeing that the balloon was completely deflated. After that, 12F sheath was implanted and pre-prepared 30 mm lenght stent was pushed forward. After taking pictures from proximal and distal, the most suitable stent position was enabled and the stent was opened by inflating the balloon (Picture-4). Aortic arc interruption may be easily treated with percutaneous intervention. However, we should be careful for possible complications.

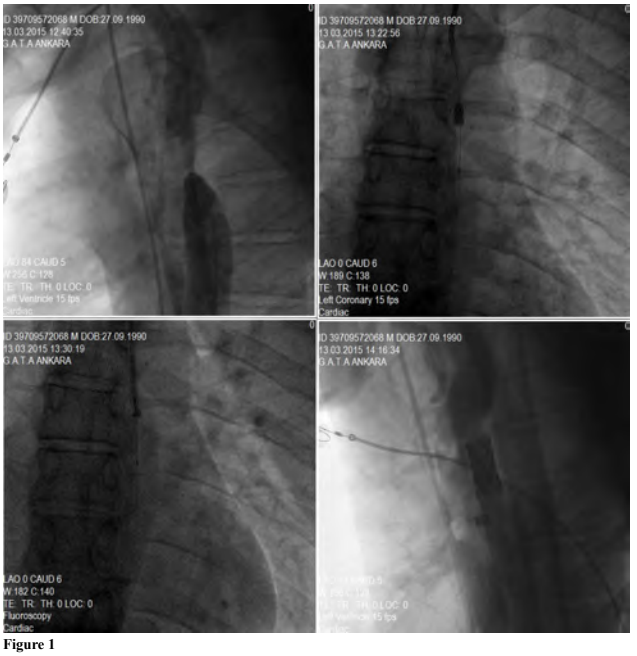

Figure 1

PO-075

Congenital Gerbode type ventricular septal defect accompanying an anomalous origin of right coronary artery

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

Case Report: A 21 year old male patient was admitted to our outpatient clinic with a history of congenital heart disease. In physical examination a 4/6 systolic murmur was heard in all auscultation points pronounced in mesocardiac area. The other physical findings were normal. In transthorasic echocardiography a Gerbode type ventricular septal defect (Figure 1), dilatation of right heart chambers and pulmonary hypertension (sPAB:45 mmHg) were detected. Transesophageal examination was consistent with the previous findings. For further evaluation right heart catheterization, oximetry and coronary angiography were performed. We decided to performed coronary angiography and aortography to exclude any other anomalies. The coronary angiography revealed that right coronary artery was originated from left anterior descending coronary artery (Figure 2). Aortography was normal. In left ventriculography we detected a passage from left ventricle to right atrium. Qp/Qs was 1,4. After these procedures we decided that operation is unnecessarry and medical fallow up was recommended. Ventricular septal defect is the most common congenital anomaly at birth which may close spontaneously. It is important to delineate the defect location and size and also physiologic characteristics of shunt. Besides transthorasic and transesophageal echocardiography, cardiac catheterization has an important role in diagnosis and shaping the management. In this case right coronary artery arises from left anterior descending coronary artery. As we mentioned previously coronary anomalies commonly accompany other congenital heart defects. Selective coronary angiography can be useful diagnostic tools in assesment of coronary artery anomalies patients with congenital heart disease.

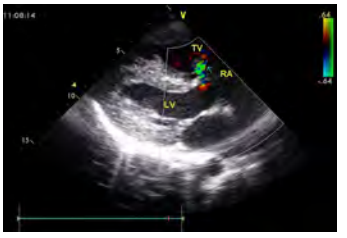

Figure 1. Echocardiographic view of Gerbode type VSD.

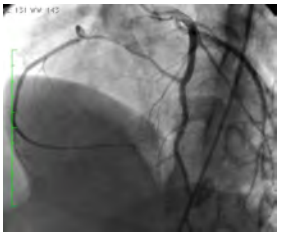

Figure 2. Right coronary artery is originated from LAD.

PO-076

VSD and coloboma in a young adult

Aziz İnan Çelik, Ali Deniz, Muhammet Buğra Karaaslan, Mustafa Gök, Fırat İkikardeş, Mehmet Kanadaşı, Ayhan Usal

Çukurova University, Faculty of Medicine, Adana

A 32 year old man was admitted to cardiology department with effort dyspnea. His NYHA functional class was 2. On his physical examination; blood pressure was 140/85 mmHg, heart rate was 76 bpm, 3/6 pansystolic murmur was heard on meso-caridac area without axillar radiate also bilateral iris coloboma was detected during the examination (Figure 1). Electrocardiogram was normal. Perimembranous ventricular septal defect (VSD) revealed on transthoracic echocardiography (Figure 2) and shunt was detected on coloured doppler echocardiography (Figure 3). Eisenmenger syndrome manifestations were not detected. Proper medical treatment was given and he was included the clinical follow-up. Coloboma and congenital heart defects are associated with various of syndromes. Most of these syndromes detected in childhood and include chromosomal abnormalities. Most of have mental, skeletal disorders, structral heart defects, and neuronal abnormalities. Genetic disorders are responsible for these syndromes. For the current diagnosis and treatment genetic examination is so significant. In our case report we can not detect other physical defect. We examine only coloboma and VSD. Lots of mosaic forms can present in syndromes and this case will be a mosaic form of one of these genetic disorders. We offered the genetic analysis to identfy the genetic disorder however patient was refused. Finally; in clinical practise especially mosaic forms of the genetic diseases can be unobserved in childhood and these patients can maintain their life until adulthood with mild symptoms. Detailed physical examination is important to identfy syndromes in adults.

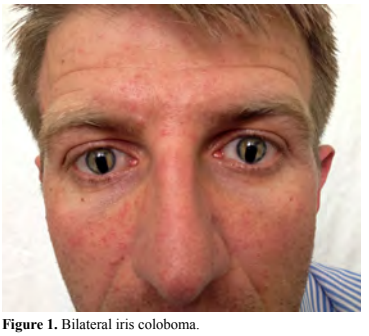

Figure 1. Bilateral iris coloboma.

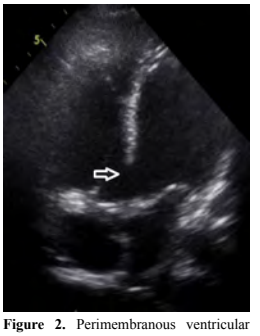

Figure 2. Perimembranous ventricular septal defect (VSD).

PO-077

A rare combination of vascular anomalies: hypoplastic aortic arch, coarctation of aorta and poststenotic aneurysm

Nermin Bayar, Şakir Arslan, Cağın Mustafa Üreyen, Erkan Köklü, Göksel Çağırcı, Selçuk Küçükseymen

Antalya Education and Research Hospital, Antalya

Coarctation of aorta is the fifth most common congenital cardiac anomaly encountered in adults.A 41-year-old male patient admitted to cardiology outpatient clinic with a complaint of exertional dyspnea. The patient was taking metoprolol 50 mg and ramipril 10 mg for 5 years and his blood pressure was regulated. Physical examination revealed II/IV diastolic murmur at mesocardiac area. Lower extremity pulses were weak and there was 20 mmHg difference between upper extremity systolic blood pressures. His electrocardiogram was unremarkable. Left ventricular diffuse hypokinesia with an ejection fraction of 35%, left ventricular hypertrophy and left heart chambers dilatation were ascertained on transthoracic echocardiograhy (TTE). Aortic valve was tricuspid and there was third degree aortic insufficiency with ascending aorta aneurysm of 52 mm. Left heart catheterization was intended, however, guidewire could not advanced through thoracic aorta to arcus aorta via left femoral artery. Stenotic segment between arcus aorta and thoracic aorta was detected by means of contrast media injection from thoracic aorta. Therefore, another catheter was forwarded via right brachial artery to proximal stenotic segment and synchronous contrast agent injection was performed through proximal and distal segments. The digital substraction angiography (DSA) of

aorta revealed coarctation of aorta (Figure 1). Peak to peak pressure gradient of coarctation was 15 mmHg. Any significant lesion was not ascertained on coronary angiography. Aneurysm of ascending aorta, coarctation of aorta following hypoplastic segment of distal arcus aorta and aneurysm with a diameter of 51 mm distal to coarctation were confirmed with 3D-reconstructed CT (Figure 2-3). Percutaneous intervention was not opted due to proximal-distal diameter mismatch and presence of ascending aortic aneurysm. Surgery was recommended, however, the patient refused surgery and he was medically treated and followed up.

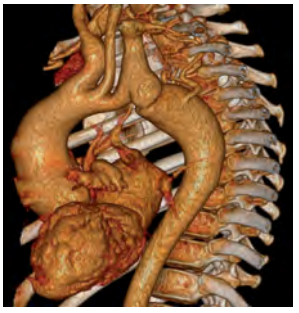

Figure 1. Hypoplastic aortic arch, coarctation of aorta.

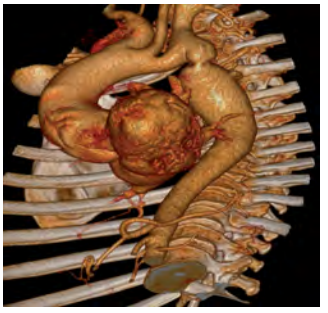

Figure 2. Poststenotic aneurysm depicted by 3D-rec-CT.

PO-078

Dilated coronary sinus without persistent left superior vena cava: atresia of the coronary sinus ostium

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[2]*Gülhane Military Medical Academy (GMMA) Ankara, Ankara*

The coronary sinus (CS) is an important vascular structure that allows for access into the coronary veins in multiple interventional cardiology procedures, including catheter ablation of arrhythmias, pacemaker implantation and retrograde cardioplegia. Coronary sinus anomalies are extremely rare, and they have received relatively little attention, probably due to the lack of both clinical symptoms and significant cardiac functional disturbance. Herein, we present a patient with a relatively rare anomaly of CS. 24-year-old asymptomatic male patient was referred to our cardiology clinic for routine cardiac evaluation. On echocardiography, a dilated coronary sinus was detected. Agitated bubble saline contrast was injected through the left antecubital vein to indicate the presence of a persistent left superior vena cava draining to the CS, however bubble contrast in the dilated coronary sinus could not visualised. Cardiac CT angiography was performed, and CS ostial atresia with a blind-ending sac was observed. CS ostial atresia, may be physiologically benign but may have fatal consequences if unrecognised prior to surgical manipulation. Even asymptomatic, patients with suspected congenital cardiac anomaly should be further evaluated. Cardiac CT and MR imagings are capable of evaluating the precise morphology of the CS, thereby avoiding potential complications during cardiac interventions.

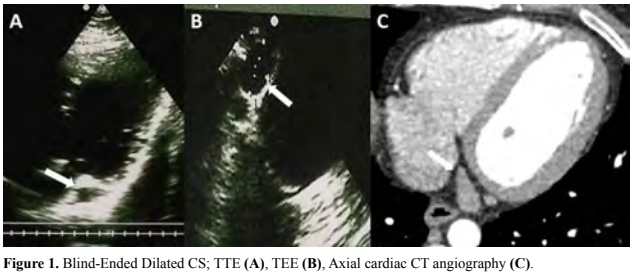

Figure 1. Blind-Ended Dilated CS; TTE **(A)**, TEE **(B)**, Axial cardiac CT angiography **(C)**.

PO-079

Unicommisural unicuspid aortic valve in an asymptomatic young runner

Zekeriya Arslan[1], Murat Ünlü[2], Cengiz Öztürk[2], Sait Demirkol[2], Turgay Çelik[2], Atila İyisoy[2], Şevket Balta[2]

[1]*Ankara Mevki Military Hospital, Ankara*
[2]*Gülhane Military Medical Academy (GMMA) Ankara, Ankara*

Unicuspid aortic valve(UAV) is a rare congenital abnormality with incidence of 2 per 10000 in the echocardiographic evaluation and clinically represents with valvular stenosis or regurgitation. UAV resembles to bicuspid aortic valvular dysfunction and may be overlooked frequently. The treatment changes in according to the level of valvular dysfunction, accompanying stenosis or aortic aneurysm. An asymptomatic 21 year-old sportsman with no cardiac history was admitted to our outpatient clinic for screening examination after success of the sportive pretests. Physical examination revealed a weak diastolic murmur at the left upper sternal border in auscultation, blood pressure of 120/75 mmHg and pulse rate 76 bpm. Transthorasic echocardiography showed a mild aortic regurgitation and CWD revealed 13 mmHg gradient. Transesophageal echocardiography revealed that a unicuspid aortic valve with single commisure located posteriorly (Figure 1a, Video 1). A mild posteriorly eccentric regurgitation in aortic valve was also confirmed with TEE (Figure 1b).

Sinuses of Valsalva and sinotubular junctions were also in normal ranges. The edges of the valve had some degree of fibrotic thickening. Although he had no any symptom, the patient was recommended to avoid from intensive sportive activities. He was referred to follow-up periodically.

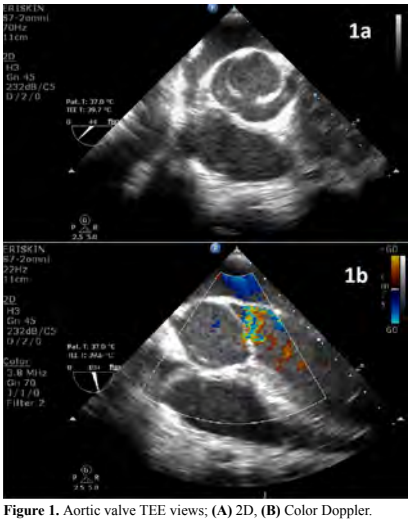

Figure 1. Aortic valve TEE views; **(A)** 2D, **(B)** Color Doppler.

PO-080

An adult case of left ventricular non compaction with severe multiple valve diseases, hypoplasia of thoracic aorta and narrowed main pulmonary artery

Erdem Özel[1], Ali Öztürk[2], Emin Evren Özcan[2], Ömer Şenarslan[2], Samet Uyar[2]

[1]*Tepecik Training and Research Hospital, İzmir*
[2]*Şifa University Hospital, İzmir*

Background: We reported a case with non compaction of left ventricle (NCLV) and severe mitral regurgitation, severe aortic regurgitation, severe pulmonary valve stenosis, hypoplasia of thoracic aorta and diffuse narrowing of main pulmonary artery. According to our knowledge this is the first case in the literature which reports NCLV with severe bivalvular regurgitation and accompanying anomalies of main thoracic vessels.

Case: 26 years old male patient was admitted to our hospital with dyspnea,and generalized oedema.His physical examination; reflects frank congestive heart failure. Electrocardiogram reveals atrial fibrillation and incomplete left bundle branch block. On his transthoracic echocardiography; biatrial dilatation,severe dilatation and global hypokinesis of left ventricle with reduced ejection fraction (20 %), severe aortic and mitral valve regurgitation, moderate tricuspid and pulmonary valve regurgitation and severe pulmonary valve stenosis were detected.There was increased left ventricular trabeculation which reflects NCLV.On his cardiac MR; diagnosis of NCLV was confirmed.On his cardiac CT angiography; complete hypoplasia of the thoracic aorta, diffuse narrowing of the main pulmonary artery and fusiform aneurysmatic dilatation of the left pulmonary artery was detected. He was received therapy for heart failure and discharged with reduced symptoms. 4 months later; mitral valve replacement,aortic root dilatation and aortic valve replacement and pulmonary valvuloplasty were performed. No complication was occured during surgery.40 days after the surgery he had sudden cardiac death at home.

Conclusion: Patients with NCLV must be carefully screened for accompanying cardiovascular disorders and arrythmic conditions. They have to be carefully managed with medical therapy for heart failure and can be directed to surgical treatment options including valve replacement, left ventricular assist device implantation and heart transplantation.

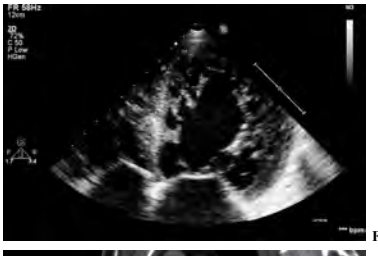

Figure 1. LVNC on echocardiography.

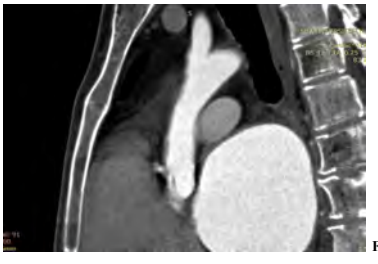

Figure 2. Hypoplastic thoracic aorta on CT.

PO-081

Double-orifice left atrioventricular valve with atrioventricular septal defect

Serkan Seçici, Ahmet Feyzi Abacılar, Savaş Demirpençe, Faik Fevzi Okur, Emin Alp Alayunt

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Double-orifice left atrioventricular valve (DOLAVV) is a rare congenital cardiac abnormalitiy. It can occur as an isolated lesion or with other associated heart diseases, most commonly atrioventricular septal defects (AVSD). The hemodynamic effects of DOLAVV in AVSD range from normally functioning valve to gross regurgitation. We report a case of DOLAVV with AVSD in a three month old infant. Three month old female child, weighing 5.2 kg was admitted to the hospital with shortness of breath and difficulty feeding. On auscultation, grade 3/6 early systolic murmur at left sternal border and rhonchi at lungs were audible. Transthoracic 2 D echocardiography with color Doppler showed situs solitus, levocardia, concordant atrio-ventricular-and ventriculo-arterial connections, ostium primum defect and large inlet VSD with left-to-right atrial shunt, mild pulmonary valvular stenosis (Max PG = 26 mmHg) and mild right atrioventricular valve regurgitation. Superior bridging leaflet extensively attached to the crest of the ventricular septum. However, the accessory orifice was not determined. The patient was diagnosed as AVSD Rastelli type A and congestive heart failure, and hospitalized. After the medication for heart failure she underwent repair of the AVSD. While assessing the left AV valve the second orifice was realized (Figure 1). The accessory orifice was located near the posterior bridging leaflet. The valve was tested with saline. Both orifice appeared competent. The accessory orifice was left untouched. Single-patch technique was performed. However, the patient died on postoperative day 14 because of septic shock. Double orifice of the left atrioventricular valve is a rare congenital abnormality, and is most commonly described in association with AVSD. The accessory orifice is commonly competent and should be left untouched. Although, our patient didn't survive, repair of DOLAVV in AVSD can be performed with a low operative risk.

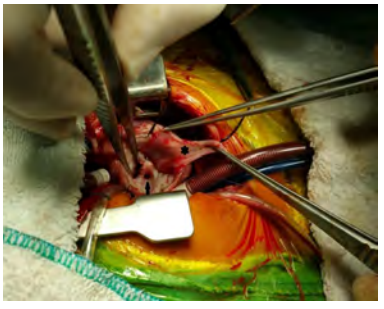

Figure 1. Accessory orifice and pericardial patch.

PO-082

Common atrium; a rare congenital heart anomaly

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We present the case of an asymptomatic 23-year old woman referred because of an abnormal routine electrocardiogram. Her history revealed any medical condition in the past. She had a previous uneventful pregnancy and a healty child. During the auscultation a mild holosystolic murmur was heard in all the auscultation points especially at the apex and a mesocardiac. S1 was normal, S2 was widely split. Her electrocardiogram revealed normal sinus rhythm with a complete right bundle branch block. Oxygen saturation measured with finger pulse oximetry was 89%. Transthoracic echocardiography revealed complete absence of interatrial septum, left ventricular dimensions and functions were normal. Main pulmonary artery and right ventricle was dilated. Mitral and tricuspid valve attachments to the interventricular septum were in the same anatomic plane (Figure). Systolic pulmonary arterial pressure was about 40-45mmHg. A major vessel opening to the left side of the atrium was thought to be a pulmonary vein. The inferior and superior vena cava were opening normally into the right side of the atrium. Patient was informed about disease and the progression then she was referrred to surgery. Common atrium (CA) is a rare congenital anomaly in adults and also known as single atrium and cor triloculare biventriculare. These patients usually admit to hospital with nonspecific sympstoms like dyspne, palpitation, fatigue or syncope at the time of late childhood or young adulthood. Interestingly, although a challenging process like pregnancy, our patient remained asymptomatic as she told in medical history. This is a very interesting case that how and when these rare patient group becomes symptomatic. As our knowledge today we know that the echocardiography is the first line diagnostic method and the surgical treatment is the first choice of therapy. Also the early diagnose is very important that before progression to Eisenmenger its vital for the patient to undergo the corrective surgery.

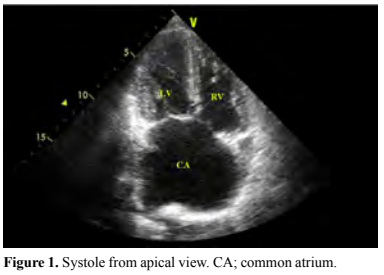

Figure 1. Systole from apical view. CA; common atrium.

PO-083

Achilles' heel of coil occlusion procedure; anticoagulant therapy

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Left internal mammary artery (LIMA) is often used in coronary artery bypass grafting for revascularization of left anterior descending coronary artery (LAD) due to its long-term patency.A 64-year-old man who had undergone coronary artery bypass grafting (LIMA to LAD) and aort valve replacement over ten years ago.Coil occlusion procedure was performed succesfully in 2010 (Figure1A) he was symptom free and only on warfarin therapy due to aortic valve replacement. He was admitted to the coronary intensive care unit with unstable angina.Bypass graft was patent but recanalization was seen on LIMA(Figure1B)Percutaneous coil reocclusion was attempted to LIMA side branch.Two coil was attached to a delivery wire and were released in the appropriate position.During the third coil detachment, attachment apparatus was detached from proximal part in the LIMA.We tried to catch the detach part of the coil via micro-snare but failed. Everolimus eluting stent was considered to implant to the LIMA next to the unligated side brunch to aim fixing the detach part of the coil (Figure2A) LIMA side brunch flow was ceased and detach part of the coil was trapped between stent and vessel wall (Figure2B) Essential component of coil occlusion procedure is thrombus formation. Although acute processual succes is satisfactory, recanalization could occure due to long term using of the anticougulant and antiagregan medication.Our patient's LIMA diameter was relatively small sized for applying vascular plug and graft stent, a coil reocclusion procedure was preferred. We did use larger sized and numerous coils to complete occlusion of the vessel with coil material.Eventually, after coil occlusion procedure the patients taking warfarin with admitted angina should be considered in terms of recanalization.In case of occlusion procedure is preferred, final result of procedure should be satisfactory in terms of totally mechanical occlusion of the vessel with coil rather than only ceased of flow.

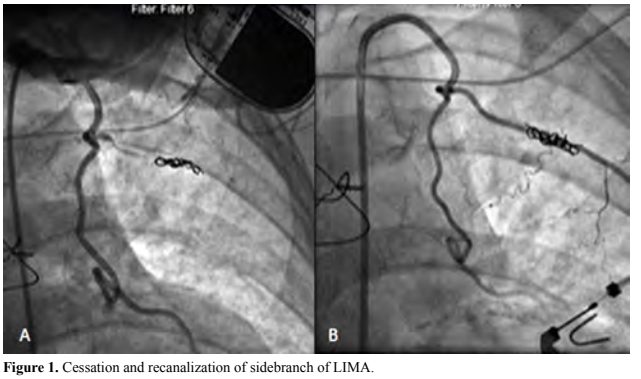

Figure 1. Cessation and recanalization of sidebranch of LIMA.

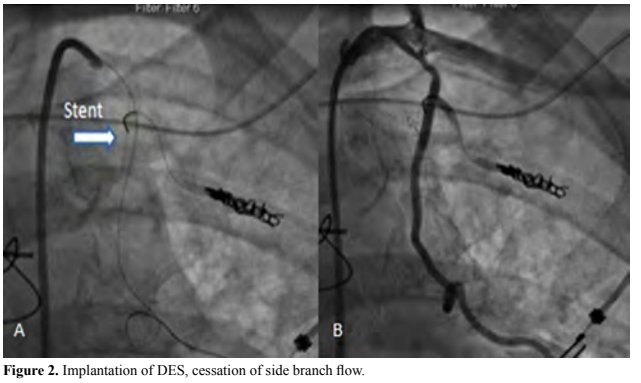

Figure 2. Implantation of DES, cessation of side branch flow.

PO-084

Korunmasız ciddi sol ana koroner lezyonu ve çok damar hastalığı olan bir olguda başarılı perkütan koroner girişim

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Giriş: Koroner anjiyografi yapılan hastalarda %4-6 oranında sol ana koroner lezyonu (LMCA) görülmektedir. LMCA lezyonu saptanan hastaların %70'inde aynı zamanda çok damar hastalığı saptanmaktadır (1,2). Ciddi LMCA lezyonu bulunan hastalarda standart tedavi yöntemi olarak bypass cerrahisi (CABG) kabul görürken son dönemlerde perkütan koroner girişimler (PKG) bu hastalarda güvenli bir biçimde uygulanmaktadır. Yapılan çalışmalar izole LMCA lezyonlarına yapılan PKG'lerin CABG kadar etkili olduğunu ve uzun dönem sonuçlarının benzer olduğunu göstermiştir (%8.7'ye karşın %6.7) (3).

Olgu: Doğuştan işitme kaybı, konuşma bozukluğu ve yürüme güçlüğü (muskuler atrofi) olan 57 yaşındaki erkek hasta, acil servise nefes darlığı ve genel durum bozukluğu ile başvurmasının ardından kalp yetersizliği tanısı konularak koroner yoğun bakıma alındı. Hastaya kalp yetersizliği için medikal tedavisinin verilmesinin ardından yapılan ekokardiyografide segmenter duvar hareket

kusuru (EF:%35) tespit edildi. Progresif troponin artışı izlenen hastaya koroner anjiyografi planlandı. Yapılan anjiyografi sonrasında LMCA distal lezyonu ile birlikte çok damar hastalığı tespit edildi (Video 1,2). CABG cerrahisi planlanan (SYNTAX 44) hasta kalp damar cerrahisi birlikte değerlendirildi, canlılık bakılması ve ardından CABG yapılması planlandı. Yapılan değerlendirilme sonrasında miyokardiyal dokunun viabl izlenmesi üzerine tekrar CABG için cerrahi ile değerlendirildi. Hastanın komorbid durumu nedeniyle CABG düşünülmedi. Hastaya basamaklı PKG yapılması planlandı. Öncelikle RCA osteal lezyonu için PKG yapıldı (3.5x20 mm PTCA,ardından 4.0x28 mm BMS). Sonraki seansta sol sisteme PKG planlandı. Hasta yüksek riskli olarak değerlendirildiğinden işlem İABP altında yapıldı. LMCA ostiumuna 8F guiding katater ile oturuldu. Cx, D2 ve LAD'ye üç ayrı guidewire ilerletildi. LAD distaline 2.5x15 mm ve 3.0x15 mm balonlarla dilate edildi ardından 2.25x18 mm evorilimus kaplı stent implante edildi. Sonrasında D2'ye 2.5x20 mm balon ile predilatasyon yapıldı. Ardından D2'den LAD proksimale uzanacak şekilde 2.5x33 mm DES implante edildi. Sonrasında Cx'de bulunan darlıklara 2.5x20 mm balon ile predilatasyon yapıldı. Sonrasında LMCA'ya uzanacak biçimde 3.0x48 mm DES implante edildi. Sonrasında LAD'ye 2.5x6 mm, Cx'e 4.0x9 mm NC balon ile final kissing balon yapıldı. Son olarak LMCA proksimalde bulunan stente 4.0x9 mm NC balon ile postdilatasyon yapılacak işlem sonlandırıldı. (Video 3,4).

Sonuç: Uzun dönem takiplerde major kardiyovaskuler olaylarda PKG ve CABG arasında anlamlı farklılık izlenmemiştir (%15.8'e karşın %13.7). Ancak tekrarlayan revaskularizasyon ihtiyacı PKG kolunda daha fazla izlenmektedir (%11.8'e karşın %6.5) (4). SYNTAX skoru düşük (33) olan hastalarda CABG daha üstündür. Aynı zamanda sistolik fonksiyonları bozulmuş olan hastalarda CABG cerrahisi öncelikli olarak tercih edilebilir.

PO-085

Guidewire fracture and entrapment during percutaneous coronary revascularization of a chronic total occlusion

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A 56-years old man was admitted to our cardiology outpatient department with Canadian Cardiac Society Class II stable angina pectoris that has been continuing for the last six months. His past medical history revealed hypertension and hyperlipidemia. Myocardial perfusion scintigraphy demonstrated inferior ischemia and diagnostic coronary angiography showed a long CTO of the right coronary artery (RCA) and retrograde distal filling of RCA from septal collateral channels of left anterior descending artery (Figure 1-panel A). Afterwards, percutaneous coronary intervention (PCI) to CTO of RCA was planned. We decided to recanalize the occluded vessel by using antegrade approach. RCA was cannulated with a left amplatz 1 guiding catheter and a stiff guidewire with a tip load of 9 grams (Conquest Pro, Asahi, Intecc) was chosen to penetrate the proximal cap with the support of a microcatheter (Corsair, Asahi, Intecc) due to the absence of microchannels. Conquest Pro succeeded to penetrate the proximal cap of the occlusion, however it went subintimally and caused a dissection. We tried to remove the guidewire but it was trapped and we failed to pull it out. Therefore, we applied a more forceful traction to extract it and succeeded in pulling it out however we saw that the guidewire was broken from its tip. Coronary angiography revealed that the tip of Conquest Pro guidewire was still in the subintimal space (Figure 1, panel B demonstrates the dissection and stucking of the fractured tip of the guidewire in subintimal space). Since the patient did not have any symptoms and there weren't any signs of perforation in the angiography, we decided to leave this guidewire subintimally and went on the procedure with another conquest pro guidewire. We were able to cross the lesion with the second guidewire and recanalized the occluded segment by stent implantation. The fractured and stucked tip of the guidewire was in the subintimal space and fully covered with the stent (Figure 2).

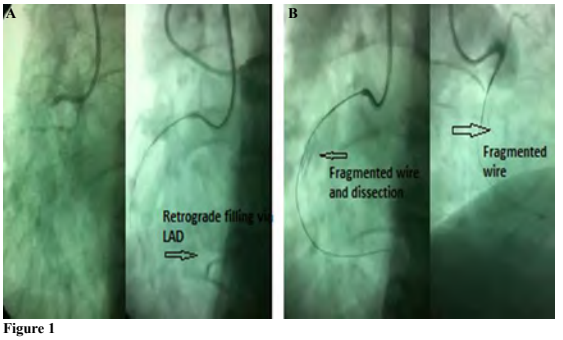

Figure 1

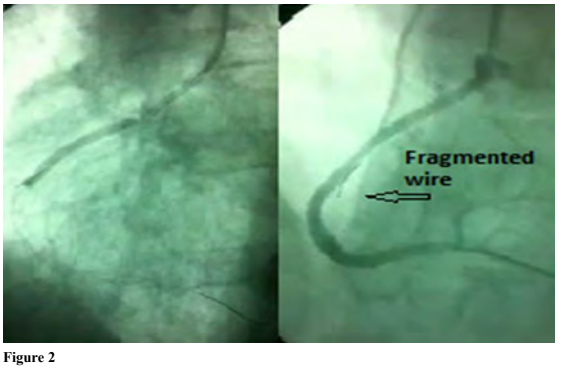

Figure 2

PO-086

Sol ana koroner distal trifurkasyon lezyonuna culotte tekniği ile başarılı perkütan girişim

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Giriş: Sol ana koroner (LM) hastalığında, perkütan koroner girişim (PKG) düşük Syntax skorlu hastalarda (≤22) sınıf I endikasyon olarak önerilmektedir. Bildirimizde, bypass cerrahisi için yüksek riskli olarak kabul edilen, LM trifürkasyon lezyonu olan bir hastada başarılı PKG işlemi sunulmaya çalışıldı.

Olgu: 73 yaşında ileri derecede KOAH tanısı ile takip edilen ve son 1 aydır sınıf 3 efor anjinası tarifleyen hastaya dış merkezde yapılan koroner anjiyografi (KAG) sonrasında hastaya bypass cerrahisi önerilmiş. KOAH nedeniyle işlemden vazgeçilmiş. Hastanemizde kalp cerrahisi ile beraber değerlendirildi ve PKG kararı alındı. KAG'de LM distal gerçek trifürkasyon lezyonu (medina 1,1,1,1) (şekil 1) ve sağ koroner arterde (RCA) ciddi darlık (Syntax skoru: 37) izlendi. Hastanın öncelikle RCA'da bulanan darlığına PKG uygulandı (Şekil 2). Sonraki, seansta LM ostiyumuna 8F sol Judkins guiding kateter yerleştirildikten sonra her üç dal da 0.014 mm floopy guidewire ile tellendi. İşlem öncesi, LAD'den LM artere pull back ile yapılan İVUS incelemesinde LAD ostiyumunu içine alan ve distal LM arteri kapsayan kritik lezyon (yumuşak plak) izlendi (LAD proksimal çapı 3.9 mm, LM çapı 4.5 mm). Ayrıca IVUS ile Cx ostiyumununun iyi olduğu gözlendi. Bunun üzerine provizyonel yan dal stentleme tekniği seçilerek öncelikle distal LM ve LAD ostiyumu dilate edildi. Daha sonra LM ostiyumunu içine alacak şekilde stent, aorta ostiyal LM bölgesinden LAD'ye uzatıldı. İmplantasyon sonrasında 5 mm NC balon ile postdilatasyon uygulandı (POT). Kontrol görüntülemede yan dallara önemli derecede plak kayması izlendi. Göğüs ağrısı da gelişen hastada, yan dallar yeniden tellenerek üçlü kissing balon uygulandı (Şekil 3). Kissing balon sonrası yan dallarda sonucun suboptimal olması ve göğüs ağrısının devam etmesi nedeniyle LM'den Cx'e culotte tekniği ile stent implante edildi (Şekil 4). Daha sonra LAD ve intermediate (yüksek obtus marjinal ?) dalları tekrar tellendi. Cx ve intermediate artere balonlar ilerletildi. Sonrasında, LAD balonu ile LAD, ancak düşük profilli CTO balonu ile predilatasyon sağlandıktan sonra ve Cx'in balon ile anchor edilmesi ile geçilebildi (Şekil 4). Ardından balonlar üçlü kissing için optimal pozisyonda ayarlandı ancak tam bu esnada mekanik arıza nedeniyle cihazın görüntüsü kayboldu. Hastanın göğüs ağrısı olması ve hipotansiyonunun gelişmesi üzerine, balonların uygun pozisyonda olması göz önüne alınarak üçlü kissing yapıldı ve arkasından tüm balonlar ile teller çekildi. 15 dakika sonra cihazın tekrar çalışması üzerine alınan kontrol görüntülerde tam açıklık sağlandığı gözlendi, ardından LM artere 5.0 mm balon ile son POT uygulandı.

Tartışma: Son zamanlarda LM artere uygulanan PKG'lerin en az cerrahi kadar etkin olabileceği gösterilmiştir. Syntax skoru yüksek olan (≥33) hastalarda PKG ile hedef lezyon revaskülarizasyon oranı CABG ile karşılaştırıldığında daha düşüktür. Cerrahi için yüksek riskli olan yüksek syntax skorlu hastalarda PKG seçenek olabilir.

PO-087

Epikardiyal kollateral arter yolu ile retrograd kronik total okluzyon revaskülarizasyonu

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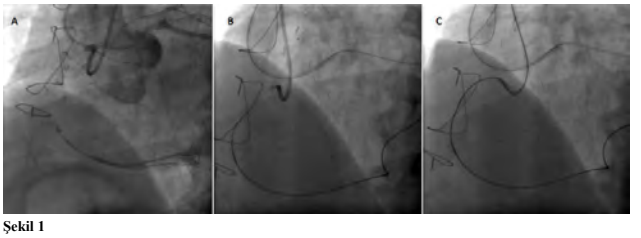
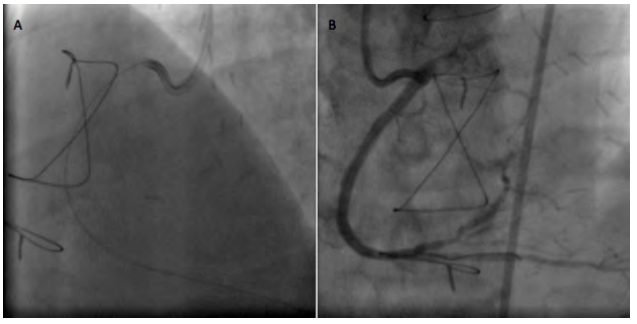
Giriş: Koroner arter hastalığı ön tanısı ile yapılan koroner anjiyografilerin %20 kadarında kronik total oklüzyon izlenmektedir. Kronik total oklüzyon vakalarının %60 kadarı antegrad yol ile revaskularize edilmekte %40 kadarında ise retrograd yaklaşım gerekmektedir, retrograd yaklaşım ile revaskularize edilen vakarda ise %25 kadarında epikardial kollateraller kullanılmaktadır. Olgumuzda 78 yaşında istirahat ağrısı tarif eden bir hastanın sağ koroner arter kronık total okluzyonun epikardial kollateral kullanılarak retrograd revaskülarızasyonu tartışılmıştır.

Olgu: 78 yaşında erkek hasta bilinen koroner arter hastalığı ve hipertansiyon öyküsü olan hasta kliniğimize CCS III stabil angina ile başvurdu. Hastanın yapılan koroner anjiyografisinde sağ koroner arterde kronik total oklüde olduğu ve sirkumfleks arterden epikardial yol ile retrograd doluş olduğu izlendi (Şekil 1A). Yapılan miyokard sintigrafisinde inferior posterior duvar bölgesinde iskemi tesbit edilen hastaya revaskülarizasyon planlandı. Antegrad yol ile corsair desteği kullanılarak fielder, conquest teller ile lezyonun geçilememesi üzerine retrograd yaklaşıma geçildi. Corsair desteği ve sion blue ile sirkumfleksden epikardiyal kollateral yolu ile distal sağ koroner artere düşüldü. Önce GAİA second ardındanconquest 9 ile rca proksimale ilerlendi, true lümenedüşülememesi üzerine antegrad olarak conquest tel ile yol alındı 2.0x15 mm balon ile dilatasyon yapılarak reverse cart tekniği ile lümene düşüldü ve kateterin içine alındı, tel katater içinde trap edilerek corsair retrograd olarak geçildi (Şekil 1B,1C). RG3 teli eksternalize edilip antegrad olarak corsair geçildi floppy tel ile RG3 değiştirildi (Şekil 2A). Balon dilatasyon sonrası stentleme yapıldı ve başarılı revaskülarizasyon sağlandı (Şekil 2B). Kronik total oklüzyon vakalarında antegrad yaklaşımın başarısız olması dusumunda retrograd yol ile epikardiyal koolateral kullanımı uygulanabilinir vakamızda başarılı bir retrograd cto vakası sunduk.



Şekil 1



Şekil 2

PO-088

Percutaneous extraction of the fractured guiding catheter from the tortuous iliac artery by a larger sheath

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Marmara University Faculty of Medicine, İstanbul

85 year-old woman presented to the emergency department with chest pain.Cardiac enzymes were elevated and ECG showed ST elevation on the leads of D1 and aVL. She transferred to cath lab with diagnosis of high lateral MI. Coronary angiography showed total occlusion on Cx artery. and tight stenosis in the midportion of right coronary artery (RCA). LAD was normal. After predilatation of 2.5x15 mm monorail balloon,3x25 mm DES was implanted on Cx artery. 2 days later, to open RCA, 6F femoral sheath was placed on right femoral artery. 6F hockey stick guiding catheter(GC) was engaged on RCA ostium. After predilatation with 2.5X15 mm monorail balloon, 3.5X32 mm DES could not be passed through the RCA lesion. During this manoeuvre, guiding catheter was disengaged from the RCA ostium. While we try to reengage the GC we noticed that GC was twisted and knotted at the level of tortuos right iliac artery. We tried to open the GC by twisting the catheter the oppposite direction and using 0.035 inch stiff wire. When we continue our manoeuvre,we noticed that the GC was fractured on the level of right iliac artery. We changed 6F femoral sheath with 7F one to use the various retrieval devices. At first, we used endomyocardial biopsy forceps But it was failed.Therefore, we used a large loop snare to remove the catheter. We succeeded to catch the distal edge of the catheter with snare and removed through the femoral sheath (Figure 1).2 days later, by entering right radial artery,two extra support 0.014" guidewire were passed through the lesion, 3.5x18 mm bare stent was implanted. In conclusion, all cardiologists must be familiar with at least one snare to remove the foreign objects from the vascular system as required and these devices must always have available at hand in the cath lab for emergent situations.

Figure 1. Removal of fractured catheter by using a snare.

PO-089

Case of type VI dual left anterior descending coronary artery anomaly with acute inferior myocardial infarction

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Bozok University, Faculty of Medicine, Yozgat

Case: A 54-year-old man was admitted to our cardiology clinic with complaint of acute onset precordial chest pain for duration of two hours. ECG revealed ST elevation of 3mm in leads II, III and aVF. Urgent coronary angiography revealed a short LAD originating from the left main, terminating prematurely after branching the diagonal artery. Selective right coronary angiography revealed total occlusion of RCA. A long LAD was originating from proximal RCA, traversing left and downward and entering the mid portion of anterior interventricular sulcus (AIS) (Figure 1). A systolic segmental narrowing was detectable in the mid segment of long LAD indicating the retropulmonic course. Primary percutaneous coronary intervention and stenting was performed for RCA. An elective CT angiography demostrated that long LAD was originating from proximal RCA, following an anomalous course to the left between the RVOT and the aortic root and entering the mid portion of AIS (Figure 2).

Conclusion: Type VI Dual LAD is a quite rare anomaly that was described by Maroney and Klein. Type VI dual LAD anomaly may be clinically very important because the risk of compression of the long LAD between the RVOT and the aortic root which can cause sudden cardiac death. Cardiac catheterization may be insufficient to identify the course of an anomalous coronary artery. In our case, vascular anatomy was clearly defined with CT angiography. A short LAD which does not reach to apex should bring to mind the dual LAD and clinicians should be encouraged to look for an anomalous long LAD. Exact course of anomalous LAD should be determined in paients with compression sign in coronary angiography. In conclusion, type VI dual LAD is a rare coronary anomaly with a potential risk of compression of the anomalous coronary artery between the RVOT and the aortic root. CT angiography can be used to determine the coronary anatomy and the course of the long LAD.

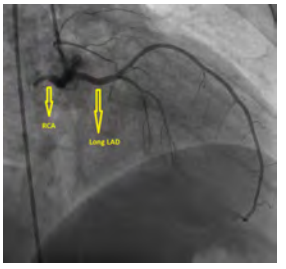


Figure 1. Angiographic view of right coronary and long LAD.

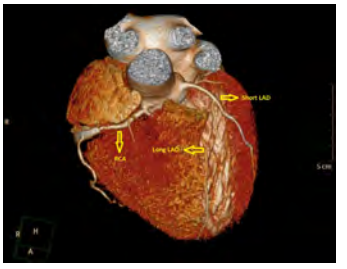


Figure 2. CT angiography.

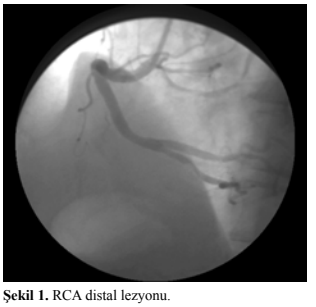
PO-090

Sağ sinüs valsalva çıkışlı LMCA lezyonuna RCA lezyonu ile eş zamanlı perkütan koroner girişim

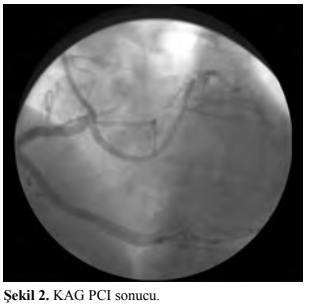
Özge Copkıran, Hakan Tıkız, Eda Özlek, İbrahim Özdemir

Celal Bayar Üniversitesi Tıp Fakültesi, Manisa

66 yaşında erkek hasta polikliniğimize bir hafta önce eforla meydana gelen göğüste baskı tarzında ağrı şikâyeti ile başvurdu. Hastaya dış merkezde yapılmış olan efor testinde EKG'de V 4-6 derivasyonlarda ST depresyonu saptanmış. Koroner arter hastalığı ön tanısı ile hasta koroner arter anjiografisi planlanarak kliniğimize interne edildi. Hastanın yapılan fizik muayenesinde arteryel kanbasıncı 120/80 mm Hg, nabız 98/dk,dinlemekle kalp sesleri ritmik, ek ses ve üfürüm yok, diğer sistemik muayeneleri olağandı. EKG sinüs ritminde idi,DIII-AVF derivasyonlarında rS paterni saptandı. Hastaya yapılan koroner arter anjiografisinde sol 4,0 judkins kateter ile sol valsalvada her hangi bir koroner artere ait ostium kanule edilemedi. Bunun üzerine hastaya yapılan aort kökünde sol sinüs valsalvadan koroner arter çıkışı izlenmedi. Daha sonra sağ 4,0 Judkins kateter ile sağ koroner arter ostiumuna oturulurken yakın çıkışlı ve ostiumu ayrı iki koroner arter izlendi. Önce sağ koroner arter ostiumuna oturularak sağ koroner arter görüntülendi, sağ koroner arter distalinde anlamlı darlık oluşturan lezyon saptandı. Daha sonra kateter biraz geri çekilerek non selektif olarak sol ön yüzü besleyen posterior seyirli (retroaortik) sol ana koroner arter görüntülendi. Görüntülenen sağ sinus valsalva çıkışlı LMCA'da proksimal ve gövde kısmında anlamlı darlık oluşturan iki adet lezyon saptandı. Sağ koroner arter ostiumu 7F sağ 4,0 guiding kateter ile kanulize edildi. Lezyon 0,014" guidewire ile geçildi. Lezyona 3,0x15 mm DES implante edildi. Daha sonra sağ sinüs valsalva çıkışlı LMCA ostiumu 7F Amplatz 1 sağ guidingkateter ile kanulize edildi. 0,014" guide wire ile lezyonlardan geçildi. Daha sonra önce distal sonra proksimal lezyona 3,0x22 mm ve 3,0x15 mm DES implante edildi. İşleme hastanın sağ koroner arterdeki lezyonu ile devam edildi. Hastanın lezyonlarında rezidü izlenmedi. Komplikasyon gelişmemesi üzerine işleme son verildi. Takibinde stabil seyreden hasta medikasyonu düzenlenerek önerilerle externe edildi.



Şekil 1. RCA distal lezyonu.



Şekil 2. KAG PCI sonucu.

PO-091

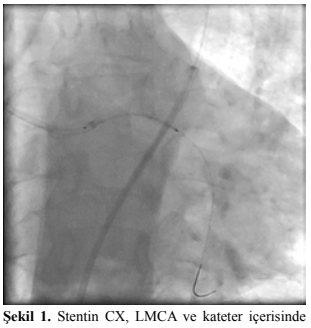
Kopan stentin balon ile katetere sıkıştırılarak çekilmesi

Şaban Keleşoğlu, Bahadır Şarlı, Bekir Çalapkorur

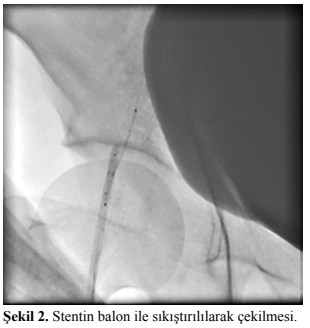
Kayseri Eğitim ve Araştırma Hastanesi, Kayseri

Perkütan koroner girişim esnasında stentin kaybedilmesi yada sıyrılması sık görülmeyen ancak by-pass operasyonu, miyokard infarktüsü (MI) ve ölüm gibi sonuçlara neden olan önemli bir komplikasyondur. Sıyrılan veya kaybolan stentler en çok snail ile yakalanarak veya balon ile arkasına geçilerek çekilmektedir. Bazen stentin koroner arterde şişilmesi veya ezilmesi tedavide tercih edilebilen yöntemlerdir. Bu vaka sunumunda akut MI sırasında şaftından kopan stentin balon ile katetere sıkıştırılarak çekilmesi anlatılacaktır. 47 yaşında hasta 2 saatlik göğüs ağrısı ile acil servise başvurmuştu. EKG'de infero-lateral derivasyonlarda ST segment yükselmesi mevcuttu. Hasta primer PCI amaçlı kateter laboratuarına alındı. Hastanın koroner anjiografisinde (KAG), LMCA normal, LAD D1 sonrası plak, CX OM1 sonrası %95, OM2 sonrası tıkalı (stent içi), RCA sağ ventrikül dalı sonrası plak, sol ventrikül dalı ostealinde %90 lezyon olduğunu göstermektedir (video 1). CX'e PCI uygulanması planlanmıştır. CX OM1 sonrasındaki lezyona 3,5x12 mm'lik çıplak metal stent 18 atm'de direkt implante edilmiştir. Sonrasında tıklalı olan lezyon floppy telle geçildi. 2,0x20 mm'lik balon ile dilatasyon uygulandı. Sonrasında 2,75x32 mm'lik stentin lezyona

implante edilmesi planlandı. Ancak stentin ilerlemesi nedeniyle geri çekilmek istendi. Geri çekilirken stent şaftından koparak stent CX, LMCA ve kateter içerisinde kalacak şekilde ayrıldı (Şekil 1). Stentin bir kısmının kateter içerisinde kalması sebebiyle başka bir balon ile stent içerisinde sıkıştırılarak çekilmesi planlandı. 3,0x20 mm'lik bir balon 18 atm'de şişilerek stent katetere sıkıştırıldı. Sonrasında kateterle birlikte geri çekildi (Şekil 2). İşlem başarılı bir şekilde gerçekleştirildi. Stent koroner arterden komplikasyonsuz şekilde çıkarıldı.



Şekil 1. Stentin CX, LMCA ve kateter içerisinde ayrışması.



Şekil 2. Stentin balon ile sıkıştırılılarak çekilmesi.

PO-092

Sağ koroner arter anjiografisi sırasında opak basıncına bağlı distalde kese görüntüsüne neden olan subadventisiyal diseksiyon vakası

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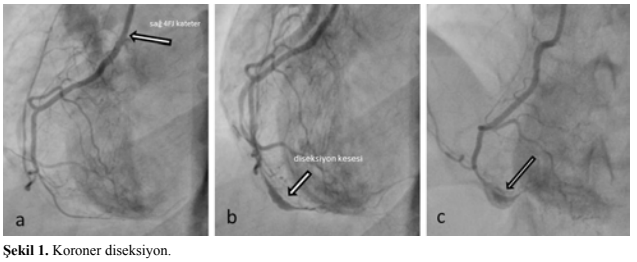
İstanbul Medipol Üniversitesi, İstanbul

Koroner diseksiyon sıklıkla, guiding kateter-wire manipülasyonu, baloon veya stent implantasyonu sebebiyle iatrojenik gelişebilmektedir. Bunların yanında kuvvetli kontrast madde enjeksiyonu sırasında da diseksiyon veya rüptür olabilir.Kırk dört yaşında kadın hasta kliniğimize, pozitif stres elektrokardiyografi sebebiyle koroner anjiografi amaçlı başvurdu.Sol radiyal arterden koroner anjiografi işlemi planlandı. Sol koroner görüntülemede sol anterior desending ve sirkumfleks arterde darlık oluşturmayan plaklar saptandı. Sağ koroner arter (RCA) görüntülemesi sırasında opak enjeksiyonu sonrası distal RCA da diseksiyon ve subadventisiyal opak göllenmesine bağlı bir kese geliştiği izlendi. vakamızda RCA çok ince olmamasına rağmen diagnostik kateterin ostiyuma, geriye opak kaçışına izin vermeyecek şekilde angaje olduğu görülmektedir. İnce damarların görüntülenmesi sırasında ve özellikle kateterin damara angaje olduğu durumlarda güçlü opak verilmesinden kaçınılması veya opak verme süresinin kısa tutulması diseksiyon veya rüptür riskini azaltabilir.



Şekil 1. Koroner diseksiyon.

PO-093

Extensive coronary dissection during diagnostic coronary angiography

Ataç Çelik, İlker Akar, Arif Arısoy, Fatih Altunkaş, Metin Karayakalı, Kayıhan Karaman, Köksal Ceyhan

Gaziosmanpaşa University Faculty of Medicine, Tokat

Iatrogenic coronary artery dissection is a rare but hazardous situation that may occur during coronary interventions. A 71-year-old woman presented with dispnea and angina on exertion. There were no significant findings except an ejection murmur heard during the chest auscultation. After the transthoracic echocardiography she was diagnosed as symptomatic severe aortic stenosis. Coronary angiography and subsequently aortic valve replacement was planned. There was no significant stenosis observed in the left coronary system after the first projection (cranial left anterior oblique). Catheter tip dropped out of coronary ostium by the injection of contrast medium during the second projection (caudal antero-posterior) and the tip was re-cannulated to the coronary ostium. After taking the spider projection extensive coronary dissection was observed (Figure, white arrows) starting from the left main stem and extended to the distal branches of the left descending and also involving the first diagonal coronary arteries (Figure, black arrows). The patient developed substernal chest pain but her blood pressure remained unchanged. After an immediate consultation with cardiovascular surgeons, patient underwent to aortic valve replacement with coronary artery bypass grafting operation. The patient was observed in an intensive care unit for 2 days and no any other complication was occurred. She was discharged 4 days later with medical therapy. This case was an interesting example of how extensive will be the dissection that may occur during diagnostic angiography even in patients with normal coronary arteries. Multislice coronary computer tomography might be the best choice for the evaluation of coronary arteries before valvular surgery.

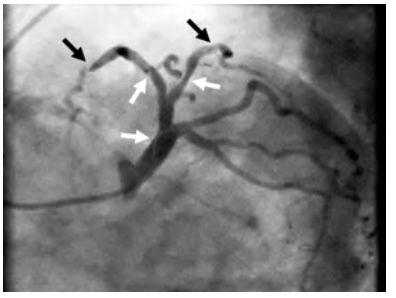


Figure 1. Coronary angiogram showing extensive dissection.

PO-094

Ratchet syndrome -not a twiddler variant; two cases with lead dislodgement

Ersin Çağrı Şimşek[1], Mehmet Erdinç Arıkan[1], Uğur Kocabaş[1], Mustafa Karaca[1], Emre Özdemir[1], Sadık Volkan Emren[2], Abdullah Doğan[1]

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[2]Afyonkarahisar State Hospital, Afyonkarahisar

Ratchet syndrome can be defined as lead retraction and dislodgement due to progressive lead displacement through its fixation parts, facilitated by movements of the ipsilateral arm and due to incomplete lead fixation to the protector sleeve, but without generator rotation over any of its axes. A 78-year-old woman with ventricular tachycardia due to ischemic cardiomyopathy had undergone implantation of single chamber implantable cardioverter defibrillator (VVI-ICD). The lead was inserted through right subclavian vein, because left subclavian vein was totally occluded. A routine pacemaker follow-up 6 months, revealed sense failure of the pacemaker. Chest X ray showed the pacemaker lead coiled around the generator proximally, resulting in lead retraction from the cardiac chamber, without generator rotation (Fig-1). The device pocket was opened, the lead was successfully repositioned. A 65-year-old woman was evaluated for pacemaker interrogation. The dual-chamber ICD had been implanted with active fixation leads for both the atrial and ventircular leads. Upon device testing, the ventricular pacing threshold was unattainable with failure to capture. A chest X-ray revealed that the ventricular lead had become coiled in the device pocket, while the atrial lead remained in place (Fig-2). The device pocket was opened, new ventricular lead was placed, and the pacemaker can was sutured in place. "Ratchet syndrome" may affect only a single lead in a pacing system with 2 or 3 leads. This is a key finding for identifying ratchet syndrome and distinguishing it from the other macro-dislodgement syndromes. Twiddler syndrome is characterized by rotation of pulse generator on its longitudinal axis and subsequent lead dislodgement with lead coiling around the pulse generator. Both of our patients had no symptoms, no mental disorder, and did not give any history of manipulation of the device pocket. These cases highlight the importance of tightly securing the lead to the suture sleeve.

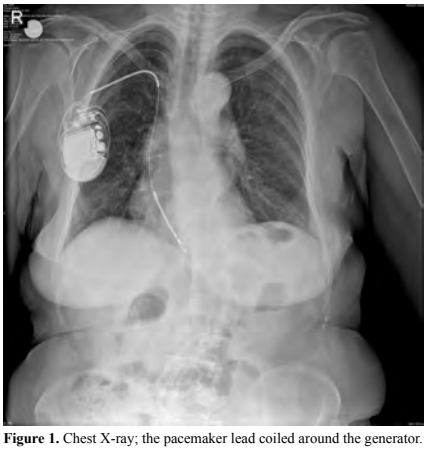

Figure 1. Chest X-ray; the pacemaker lead coiled around the generator.

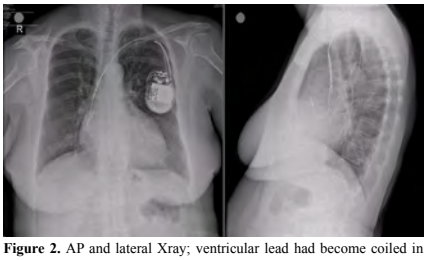

Figure 2. AP and lateral Xray; ventricular lead had become coiled in the device.

PO-095

Sick sinus syndrome and low ejection fraction with emery-dreifuss muscular dystrophy

Fatih Mehmet Uçar[1], Muhammet Gürdoğan[2], Çağdaş Kaynak[3], Mustafa Yılmaztepe[3], Fatih Özçelik[3]

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Introduction: The Emery-Dreifuss muscular dystrophy (EDMD) is a form of muscular dystrophy that frequently presents early contractures and cardiac conduction defects, caused by emerin deficiency in the inner nuclear membrane of the muscular fibers. Almost all people with Emery–Dreifuss muscular dystrophy have heart problems by adulthood. In many cases, these heart problems stem from abnormalities of the electrical signals that control the heartbeat (cardiac conduction defects) and abnormal heart rhythms. We report the case of 32-year-old man with Emery–Dreifuss muscular dystrophy who developed sick sinus syndrome and low ejection fraction.

Case Report: A 32-years-old man presented muscle weakness and hypotrophy in the proximal upper and lower limbs. On physical examination, all findings were normal. Electrocardiography showed sinus rhythm and transthoracic echocardiography revealed decreased ejection fraction, thickened walls and left atrium dilatation. We learned he has sometimes dizziness and planned 24 hr holter monitorization. 24 hr Holter monitor showed lots of pauses. The longest pause was 5.3 second on 02.00 a.m.(Figure-1). Because of the patient has atrioventricular block and low ejection fraction we implant dual chamber Intracardiac defibrillator (ICD).

Discussion: Young males with complete atrioventricular block involve careful review of family history and should always be evaluated for the possibility of EDMD. Pacemaker implantation protect this patients to prevent sudden death from atrioventricular block, sick sinus syndrome and ventricular arrhythmias.

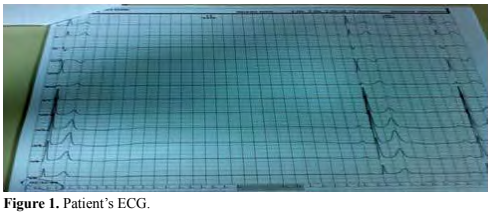


Figure 1. Patient's ECG.

PO-096

Reccurrent cardiac tamponade that developed after atrioventricular nodal reentrant tachycardia ablation

Fatma Yiğit

Başkent Universtiy Hospital, Adana

Radiofrequency ablation (RFA) is frequently used to treat atrioventricular nodal reentrant tachycardia (AVNRT) is a safe and effective curative treatment. One major complication of RFA is pericardial effusion-tamponade. The rate of tamponade was 0.6% and that of insignificant effusions 1.5%. This treatment is also effective and safe in the elderly. Our patient developed recurrent cardiac tamponade after ablation that was treated with pericardiosyntesis. Our patient was 70-year-old woman. In April 2011 was admitted to our clinic with complaints of palpitations, which is 10 years. She has used beta blocker and propafenone for palpitations. In his history was hypertension and diabetes. The patient's physical examination was normal. There was no abnormality on the basal ECG. In the ECG during palpitation supraventricular tachycardia (SVT) was detected (figure 1). Electrophysiology was performed. For this purpose, the EPS catheters were placed into the high right atrium, right ventricle, and His area through right femoral vein and right internal jugular vein. Basal conduction intervals were normal. Ventriculo-atrial conduction was decremented and concentric. Via programmed atrial stimulation, dual AV nodal physiology was shown and typical AVNRT was induced at a cycle length of 300 milliseconds. It was done 3 RF ablations, and eliminated the tachycardia. After the procedure, shortness of breath, chest pain, palpitations and sweating developed. Echocardiography showed moderate pericardial effusion and right atrium and right ventricle collapsed. Emergency pericardiocentesis was done. The patient recovered. Five days later she was discharged. Five weeks later, the patient was admitted to the clinic with complaints of chest pain, back pain, shortness of breath, sore throat, and whooping cough. The patient had hypotension and tachycardia. There was sinus tachycardia on ECG was done. Pericardial effusion and right heart collapsed were detected on echocardiography. We performed pericardiosyntesis, and hemoragic fluid was drained. The chest disease specialist was examined the patient for whooping cough and shortness of breath. She was diagnosed with asthma and pneumonia. Cough and shortness of breath were passed completely with antibiotic and inhaler treatment. The patient was checked daily ECHO. Pericadial effusion and palpitation didn't recur. We thought that recurrent cardiac tamponade was triggered due to whooping cough. She was well after asthma treatment. The patient is followed for 3 years without any problems.

PO-097

Who should follow-up: industry represantatives or cardiologists?

Çağın Mustafa Üreyen, Şakir Arslan, Göksel Çağırcı, Cem Yunus Baş, Murat Esin, Görkem Kuş, Özkan Kayhan, Gamze Korkmaz

Antalya Education and Research Hospital, Antalya

A 26-year-old male patient was referred to our outpatient pacemaker clinic for replacement of the battery due to elective replacement indicator (ERI) alert of his pacemaker. A DDD pacemaker was implanted owing to intermittent complete AV block six years ago. He had been on follow-up by industry represantatives merely for six years. On questioning,the patient was not expressing any significant complaint except that he was no longer able to play effortful football matches, therefore, he had to play as a goalkeeper rather than in forward position. His physical examination was unremarkable and his ECG demonstrated sinus rhythm with a wide QRS which was 192 msec due to ventricular pacing. Interrogation of pacemaker revealed normal pacing thresholds, sensed P/R wave amplitudes and lead empedances. However, battery voltage was low, 2.46 V and alert of ERI was seen. It was also determined that ventricular pacing was 100%. AVI was set to 150 msec. Moreover, assessment of sensing amplitudes revealed that the patient had first degree AV block rather than complete block with an PR interval of 210 msec. Left ventricular (LV) ejection fraction (EF) was determined as 42% on echocardiography with modified Simpson method. Left ventricle was globally hypokinetic and LV end diastolic diameter and LV end systolic diameter were 61mm and 47mm, respectively. Following resetting of AVI to 250 msec, atrial (A) and ventricular (V) sensing were ascertained. The diagram depicting the percentage of ventricular pacing was initialized. The battery was replaced one day after the resetting and the patient's ECG was still A-sense and V-sense on the day of discharge. The patient was called for a control visit after 3 months, however, the patient applied to outpatient pacemaker clinic after 7 months. After interrogation of pacemaker, only 13% of V-pacing was ascertained. Control EF of the patient was 57% with modified Simpson method. The patient shared smilingly that he was still playing as a goalkeeper rather than a scorer. However, it was just only due to the preference of his friends, rather than exertional dyspnea. Optimisation and follow-up may be allowed by inexperienced physicians to be executed only by industry representatives. Therefore, the efficacy of optimisation and follow-up becomes dependent on industry representatives' knowledge and experience unfortunately. We hereby emphasise that an incorporation of education of optimisation and follow-up of cardiac devices into education of cardiac device implantation should be executed in order to enhance physicians' knowledge and experience for better follow-up and prognosis.

PO-098

Case report: atrioventricular block related to glucosamine/ chondroitin in a patient presented with syncope complaint

Fatih Öztürk, Hasan Ali Gümrükçüoğlu, Yunus Emre Okudan, Fatma Nur Gümrükçüoğlu

Yüzüncü Yıl University Faculty of Medicine, Van

Glucosamine (GlcN) is an amino-monosaccharide, and it was believed that it modifies progression of osteoarthritis (OA) as a constituent of glucosaminoglycans in joint cartilage(1). Although it is not a FDA-approved drug, it is on the market in the form of glucosamine/chondroitin sulfate as a nutrition supplement in US and Turkey. Chondroitin sulfate (CS) is the essential constituent of the connective tissue extracellular matrix in hyaline cartilage of which it is responsible for the maintenance of elasticity. Although side-effect profile of GlcN is non-specific, gastrointestinal symptoms, headache and skin-related symptoms have been observed, but no side effect on heart has been reported to date. Atrioventricular (AV) complete block is a bradyarrhythmia, and it may result in mortal consequences if left untreated. AV complete block may arise due to various reasons including acute myocardial infarction (AMI), chronic ischemic heart disease, myocarditis, collagen tissue diseases, trauma, infiltrative diseases, neuromuscular defects, myxedema, medications and electrolyte imbalance. In literature, development of AV block related to use of a number of medications has been reported such as carbamazepine, amphotericin B and somatomedin. However, a case with AV block by reason of glucosamine/chondroitin (GlcN- CS) use has not yet been reported to date. In this case report, we aimed to present a patient with AV complete block developed after GlcN-CS use. CASE A 42-year-old male patient presented to the emergency department with syncope lasting less than a minute followed by dizziness and nausea. He denied any history of a similar syncopal episode. In his medical history, he stated that he had diagnosed with osteoarthritis, and admitted the use of a supplement containing GlcN-CS as the active ingredient beginning 3 days before presentation. He denied taking any other medications or supplements. There was no significant disease other than hypertension in his family history. In his physical examination, he had a blood pressure of 126/86 mmHg, and a lower heart rate of 40 beats/min. A-V complete block was diagnosed on the 12-lead ECG. Blood potassium level was 3.4 mEq/L. In transthoracic echocardiography, there was no pathology other than left ventricular hypertrophy. In applied coronary angiography aiming to exclude AV complete block caused by ischemia, no critical coronary stenosis was detected, and temporary cardiac pacemaker was not inserted since his hemodynamics was stable. The patient was transferred to the coronary intensive care unit (ICU) and began to be monitored. At the 4th hour of his follow up in the ICU, the patient was with a normal sinus rhythm. The patient was monitored for the following 72 hours with no abnormality in holter recordings, and was discharged from the hospital. In the electrophysiological evaluation carried out after discharge, AV nodal func.

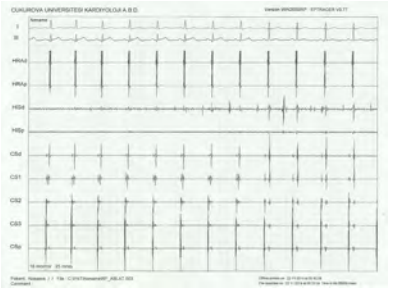
PO-099

İntermitan Wolf Parkinson white sendromu olan hastada atriyal fibrilasyon

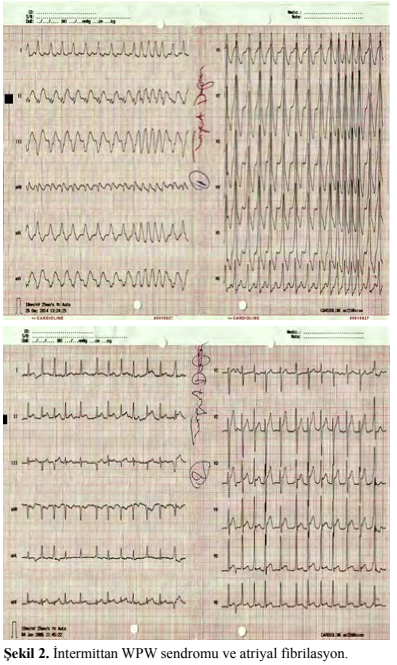
Mustafa Gök, Muhammed Buğra Karaaslan, Mustafa Tangalay, Müslüm Fırat İkikardeş, Ali Deniz, Rabia Akıllı Eker, Onur Sinan Deveci, Çağlar Emre Çağlıyan, Mesut Demir, Mehmet Kanadaşı, Ayhan Usal

Çukurova Üniversitesi Tıp Fakültesi, Adana

31 yaşında bilinen sistemik hastalığı olmayan hasta çarpıntı şikayeti ile başvurdu. Fizik muayenesinde bilinçi açık oryante,koopere TA:90/60 mmhg nabız:200/dk, taşiaritmiktiK. Başka patolojik bulgu saptanmadı. Hastanın çekilen EKG'sinde geniş QRS kompleksli düzensiz taşikardi saptandı, WPW sendromu ve yüksek ventrikül yanıtlı atriyal fibrilasyon tanısıyla kardiyoversiyon yapıldı (Şekil 1A). Kardiyoversiyon sonrasında çekilen EKG'de AF'nin devam ettiği, ancak aksesuar yol iletiminin kaybolduğu ve ritmin dar QRS kompleksli olduğu saptandı.(Şekil 1B) Tekrarlayan kardiyoversiyon ile sinüs ritmi sağlandı ve EKG'de delta dalgalarının varlığı görüldü. Elektrofizyolojik çalışma ve ablasyon planıyla işleme alındı. Sol posterolateral aksesuar yol saptanan hastaya retrograd aortik yol ile ablasyon uygulandı (Şekil 2). İşlem sonrası delta dalgası kayboldu. Komplikasyon gelişmedi.



Şekil 1. Ablasyon sonrası.



Şekil 2. İntermittan WPW sendromu ve atriyal fibrilasyon.

PO-100

Recurrent spontaneous deep vein thrombosis due to PAI-1 4G/5G homozygous mutation

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A 27-year-old female patient applied to outpatient clinic with right leg pain and swelling. On questioning, a history of deep vein thrombosis (DVT) occuring seven months ago was ascertined. She quitted warfarin therapy after three months and was free of symptoms for four months. Thrombosis was demonstrated in right calf veins by doppler ultrasound and the patient was anticoagulated with both enoxaparin and warfarin until INR was 2. The patient denied any history of recent surgical operation, bed rest, long flight, trauma to leg as well as any history of recent pregnancy, oral contraceptive usage and history of inflammatory or autoimmune disease, all of which were also valid for the first DVT attack. Inherited risk factors were evaluated due to absence of any acquired risk factor and presence of recurrent spontaneous DVT. Any mutation of Factor V Leiden, prothrombin G20210A, antithrombin III, JAK2V617F, protein C and S were ascertained except MTHFR heterozygote and PAI 1-4G/5G homozygote mutations. Furthermore, levels of homocysteine, lupus anticoagulant and anticardiolipin antibody were also determined normal. The patient was instructed to take warfarin life long by virtue of recurrent spontaneous DVTs and homozygous PAI-1 4G/5G mutation. 4G/5G polymorphism of PAI-1 gene was supposed to be a risk factor of thrombotic diseases, however, it is not still explicit whether it possesses a significant role in thrombotic disorders. Balta et al demonstrated that 4G/5G polymorphism of PAI-1 gene increased risk factor for development of thrombosis in the vessels of several internal organs. On the other hand, Mansilha et al stated that FV G1691A, FII G20210A, MTHFR C677T or PAI-1 4G/5G polymorphisms were not related to the risk of recurrent deep venous thrombosis in young people. Although the significance of 4G/5G polymorphism of PAI-1 gene is still controversial, we recommended the patient to administer warfarin lifelong by virtue of recurrent spontaneous DVT attacks and homozygous 4G/5G polymorphism of PAI-1. Since, we could not elucidate any acquired or inherited risk factor for thrombosis, we speculated homozygous 4G/5G polymorphism of PAI-1 to be the etiologic factor for recurrent spontaneous DVT attacks. Therefore, our case report supported the data suggesting 4G/5G polymorphism of PAI-1 as a risk factor associated with thrombotic diseases.

PO-101

An interesting solution for coil migration and coronary artery dissection in the same patient

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Introduction: We report the first case with coil migration and coronary dissection to occur simultaneously in the same patient and succesfull treatment of these two complications with stent implantation.

Case Report: A 68-year-old man was referrred to our department with complaints of exertional dyspnea and exertional chest pain. Coronary arteriography did not show significant lesions in the epicardial coronary arteries and a fistula was observed from the proximal portion of RCA draining into the pulmonary artery. The patient continued with symptoms and, therefore, in a second procedure the closure of the fistula from RCA was performed with an AR 2 guiding catheter. A 0.014î and 300 cm hydrophilic guidewire was moved up to the distal segment of the fistula and a microcatheter was then loaded over this and a coil was placed. For total occlusion achievement a second coil was placed. Unfortunately soon after the insertion of the second coil, during contrast injection this coil detached and migrated to proximal portion of RCA. At the same time coronary dissection was observed in the proximal portion of RCA (Figure 1). Therefore, 4.0×9mm bare metal stent was successfully implanted at 14 atm to cover the dissected segment and migrated coil was jailed to side branch to prevent any embolization to the distal portion of RCA or aorta (Figure 2).

Discussion: With catheter closure techniques, complete occlusion of the fistula may be achieved in >95% of the patients. The main complications include either premature deflation of a detachable balloon, inadvertent coil embolisation, transient T-wave changes, transient bundle branch block and myocardial infarction. All the complications are rare, apart from inadvertent coil migration, which may occur as a result of high flow in the large fistulas or with undersized coils.

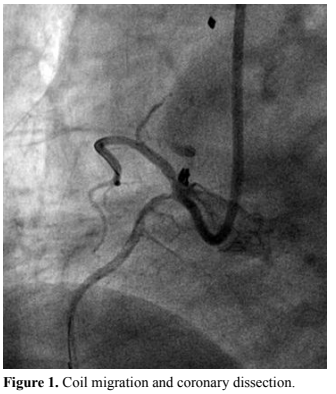

Figure 1. Coil migration and coronary dissection.

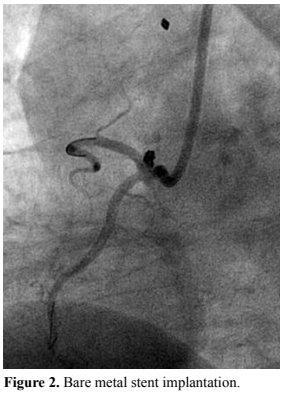

Figure 2. Bare metal stent implantation.

PO-102

An uncommon but dangerous complication after successful pericardiocentesis: pericardial decompression syndrome

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Pericardiocentesis is a life-saving procedure commonly performed in cardiology practice. Injuries of cardiac wall, coronary arteries and lungs are well-known complications; however pericardial decompression syndrome (PDS), a serious but less known complication of pericardiocentesis (1). We aimed to present a case of PDS following pericardiocentesis in a patient with suspected malignancy and cardiac tamponade.A 27 year-old female patient with ovarian mass was evaluated for malignancy and presented with progressive shortness of breathe and palpitation symptoms. Echocardiographic examination revealed pericardial effusion surrounding the entire heart. Signs of cardiac tamponade were detected and pericardiocentesis was scheduled. After obtaining informed consent and local anesthesia, initially 500 ml and then 480 ml of serous fluid was drained via subxyphoid route. Symptoms of the patient improved right after the procedure but two-hours later a new-onset dyspnea, tachycardia and hypotension developed. The patient was intubated for progressive respiratory failure and control echocardiography showed no pericardial effusion. However left ventricular ejection fraction which was normal before the index procedure, was measured 20 % with global hypokinesia. The patient was followed-up with inotropic support but shortly afterwards bradycardia and cardiac arrest occurred and cardiopulmonary resuscitation was undertaken. The patient died despite all supportive treatment.

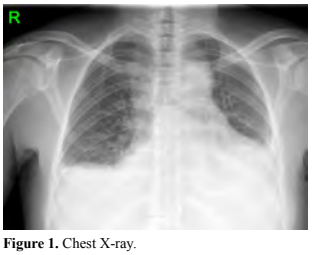

Figure 1. Chest X-ray.

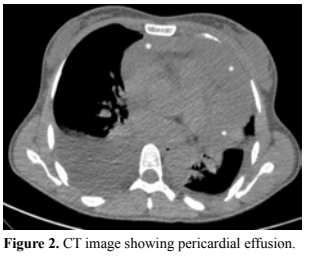

Figure 2. CT image showing pericardial effusion.

PO-103

Rhabdomyolysis secondary to therapeutic hypothermia after cardio-pulmonary resuscitation; a rare complication

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Patient maintenance after successful cardio-pulmonary resuscitation starts with decreasing the neurological damage despite serious difficulties such as hypoxic ischemic infarcts and reperfusion infarcts. Therapeutic hypothermia is the most rejoicing method in use to prevent neurological damage. Here we discuss 35 years-old woman resuscitated for twenty minutes in hospital who was followed because of postpartum cardiomyopathy. Sudden onset of ventricular fibrillation subsequent to ventricular tachycardia was the underlying cause of cardiac arrest. In order to prevent neurological damage therapeutic hypothermia was used, she was cooled for 24 hours. After therapeutic hypothermia her glaskow coma score was fifteen, fortunately no sequela appeared. Although we were successful to prevent neurological damage, rhabdomyolysis arose secondary to therapeutic hypothermia. As a result the intubation process was prolonged, acute tubular necrosis due to myoglobinuria occured. In spite of all complications patient faced, she was discharged on her 31th day; without sequela and with no need for hemodialysis; under medical therapy. In routine clinical practice cardio-pulmonary arrest patients are seen frequent in emergency departments and intensive care units. The approach to utilize most should be rapid and protective. One of the most popular approach, therapeutic hypothermia has been shown to reduce mortality and has improved neurological outcomes after cardiac arrest. Here we discuss an unusual case of a patient resuscitated successfully, cooled for 24 hours therapeutically. Rhabdomyolysis secondary to therapeutical hypothermia occured, her all muscle indicators rised excessively This case is an important example of presentation of rhabdomyolysis can be a complication of therapeutic hypothermia. In order to protect the neurological system, we may cause damage in urinary and skeletal system. Rhabdomyolysis is the rapid breakdown of striated muscle by rupture and necrosis of muscle fibers. Symptoms develop over hours to days after an inciting factor and may be associated with dark pigmentation of the urine. There are so many reasons of rhabdomyolysis, in our therapeutic hypothermia was the underlying cause. The complications of rhabdomyolysis are such as hyperkalemia, hyperphosphatemia, hypocalcemia, disseminated intravascular coagulopathy, acute kidney injury. One of the most important treatment goals is to avoid the kidney injury. Theoretically hypothermia is one of the causes of the rhabdomyolysis. In the literature there are only few cases issued rhabdomyolysis secondary to therapeutic hypothermia. Rhabdomyolysis might be a rare complication of therapeutic hypothermia and rewarming can interrupt its progression.

PO-104

Amiodarone-induced exudative bullous lesion and hepatotoxicity in a patient with ventricular tachycardia

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Introduction: Amiodarone is a class III antiarrythmic drug, which has high lipophilicity properties and high iodine content. Amiodarone therapy has been reported to cause some dermatological lesions like skin discoloration, blue-gray pigmentation and photosensitivity. We report a case of man who developed eryhtematous exudative bullous skin lesions and hepatotoxicity during amiodarone therapy.

Case Presentation: 61-year-old male patient was admitted to the emergency department with complaints of shortness of breath, angina pectoris and palpitation. Electrocardiography showed monomorphic, monofocal ventricular tachycardia (VT). On admission, his hemogram end biyocamical parameters were normal. The patient diagnosed with sustained VT. After loading dose of amiodarone, 150 mg in 100 ml dextrose (D5W) administered over 10 minutes (15 mg/min), the therapy was continued with a slow loading infusion (1 mg/min for 6 hrs and 0.5 mg/min over 18 hrs) containing 900 mg amiodarone injection in 500 ml of D5W. One day later, biochemical and hemogram results were as follows: ALT 99 U/L (normal <41 U/L), AST 89 U/L (normal <40U/L) and basophiles 0.08 c/µL (normal 0.00-0.02 c/µL). The patient had developed multiple bullous skin lesios over the anterior chest wall filled with exudative material. All bullae appeared on slightly erythematous base. Diameter of the largest bulla was 2.3 x 1.5 cm. and the diameter of the smallest bulla was 0.6x0.7 cm. Erythema was more prominent around the hair follicules and occurred both on the normal and involved skin. At the follow-up visit one month later, the patient noted that his symptoms subsided. The patient's biochemical, hemogram and ECG results were normal. Bullae healed leaving scar tissue. In conclusion, Side effects observed with amiodarone are related to dosage and route of administration. Skin lesions do not require discontinuation of amaidarone treatment. Treatment should be stopped if patients develop hepatotoxicity or liver enzyme elevations greater than 3 times the upper limit of normal. It should be kept in mind that association of exudative bullous skin lesions and hepatotosisty may occur during amiodarone therapy.

PO-105

İn transit sağ atrial trombus: İki olgu sunumu

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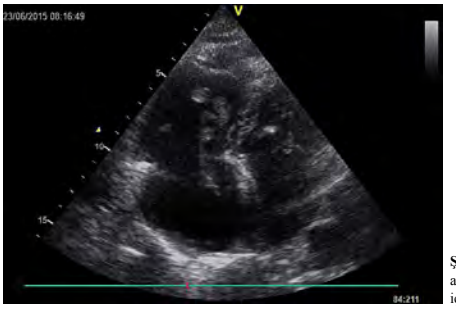
Abant İzzet Baysal Üniversitesi Bolu Tıp Fakültesi Ar. ve Uygulama Hastanesi, Bolu

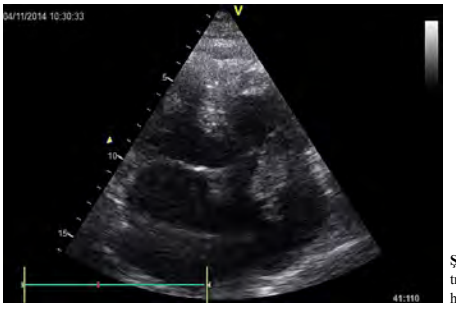
Olgu 1: 66 yaşında kadın hasta 10 gündür mevcut olan dispne ile başvurdu. Hemodinamisi stabildi. EKG sinüs ritmi ve sağ dal bloğu larak izlendi. Transtorasik ekokardiyografide ise hafif mitral darlığı, sağ boşluklar dilate, orta triküspit yetersizliği ve sağ atriyum içinde serbest, 1 cm eninde 7-8 cm uzunluğunda triküspit kapaktan sağ ventriküle girip çıkan trombus izlendi (Resim 1, Video 1). Acil cerrahi eksizyon kararı verildi ancak hastada aniden masif pulmoner tromboemboli ve şok tablosu gelişti. Hastaya trombolitik tedavi verildi. Trombolitik tedavi sonrası klinik durumu toparlamayan hastaya cerrahi olarak pulmoner embolektomi ve De Vaga Triküpit Annuloplasti yapıldı. Hasta şifa ile taburcu edildi.

Olgu 2: 73 yaşında bayan hasta sağ bacakta şişlik şikayeti ile başvurdu. Hastanın beyin tümörü tanısı mevcuttu ve epileptik nöbetler geçirmesi nedeni ile kısmen inmobildi. Hasta derin ven trombozu tanısı ile yatırıldı. Fizik muayenede hemodinamisi stabildi. EKG atrial fibrilasyon olarak izlendi. Transtorasik ekokardiyografide sağ atrium içinde serbest yüzen 1x1.5 cm boyutlarında triküspit kapaktan sağ ventriküle girip çıkan trombus izlendi (Resim 2, Video 2). Sağ atrial trombus cerrahi olarak eksize edildi ve vena cava inferior filtresi takıldı. Hasta şifa ile taburcu edildi.

Tartışma: Sağ atrial trombus oldukça nadir görülmesine rağmen klinikte masif pulmoner tromboemboli ile sonuçlanır mortal seyredebilir. Klinisyelerin bu durumu göz önünde bulundurması gerektiğini düşünmekteyiz.



Şekil 1. Sağ atriyal trombüs; apikal pencerede sağ atriyum içinde trombüs.



Şekil 2. Sağ boşluklar da trombüs; sağ boşluklar içinde hareketli intransit trombüs.

PO-106

Behçet's disease with a large intracardiac thrombus complicated with acute pulmonary embolism: a case report

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Behçet's disease is a chronic, inflammatory and multisystemic condition of unknown aetiology. Although superficial and deep / peripheral vein thrombosis is seen in a quarter of these patients, cardiac involvement is extremely rare. We present a case of Behçet's disease with right ventricular thrombus. A 43-year-old man was admitted to our hospital for the evaluation of acute pulmonary embolism with right ventricular mass diagnosed by echocardiography. On physical examination; blood pressure was 100/60 mmHg and heart rate=110 bpm. The heart sounds were normal without any murmurs. His body temperature was 38. The electrocardiogram revealed a sinus tachycardia. The echocardiography showed 2,9 cmx4,5 cm slightly immobile mass occupying the right ventricular apex. This mass appeared hyperechoic and well circumscribed with heterogeneous density (Fig. 1), systolic pulmonary artery pressure was 40 mmHg. We couldn't detect any thrombus in his venous system and computed tomography of abdomen was normal. There was also weight loss, intermittent fever and blurred vision in his history, lasting for one year. Following the initiation of anticoagulant treatment, we noticed acneiform lesions on the torso and scar lesions of a few millimeters on the scrotum (Fig. 2) Taking these findings together, the patient was diagnosed with Behçet's disease (BD) as he fulfilled The International Study Group of Behçet's Disease criteria. After diagnosis, steroid treatment and cyclophosphamide was started. One months later, the thrombus was decreased substantially in size. The patient was continued to the medical treatment without recurrence. Conclusion: Behçet's disease should be keep in mind, in young patients with intra-cardiac thrombus formation who has no structural cardiac pathologies.

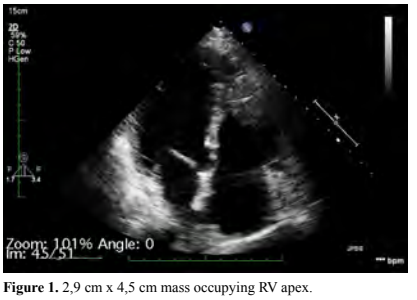

Figure 1. 2,9 cm x 4,5 cm mass occupying RV apex.

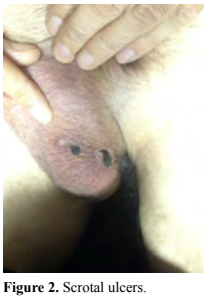

Figure 2. Scrotal ulcers.

PO-107

A very rare reason of stable angina pectoris (Cardiac AL amyloidosis)

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A 37-years-old female, with previously diagnosed AL amyloidosis by bone marrow biopsy was admitted with exercise induced chest pain, weight loss and progressive fatigue for seven months to haematology department. The patient consulted with us because of high troponin level and chest pain. We noted that the pain was in the middle of chest, characterised by a feeling of pressure, was getting higher with minimal exertion, suggested typical stable angina. Blood pressure was 95/65 mmHg, bilateral severe pretibial edema was realised and a 3/6 holo-systolic murmur was heard on left upper sternal border. Troponin T level was high (respectively, 201-139-140-202 pg/ml,) but didn't get higher progressively day by day and at the same time creatinin level was 0.8 mg/dl. Follow of electrocardiogram were also nonspesific in the way of acute coronary syndrome. The transthoracic three dimensional echocardiography demonstrated concentric thickening of the left ventricle with myocardial granular sparkling, normal wall motion and systolic function, thickening of the heart valves and interatrial septum, mild pericardial effusion adjacent to the right atrium, diastolic filling pattern of the left ventricle, which were in consistent with cardiac AL amyloidosis. We decided to display coronary arteries by computed tomography angiography. CTA showed normal coronary arteries and thorax CT excluded pulmonary embolism and aortic dissection which could be the possible aetiology for chest pain. Very rare condition of cardiac amyloidosis, mimicking coronary heart disease, stable angina was diagnosed for this patient. And then we started to follow patient with medical treatment. Subsequently, patient's kidney function started to get worse and the patient died 35 days after admitting. Cardiac amyloidosis can present firstly or after the other main organ presentations as angina pectoris associated with coronary flow reserve abnormalities despite normal coronary angiograms.

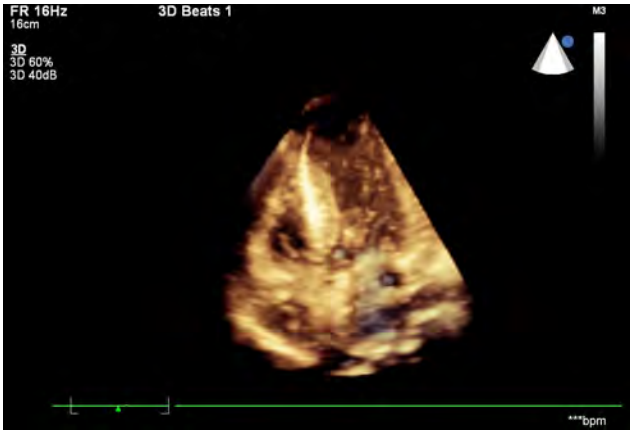

Figure 1. Cardiac wall thickening; three dimension echocardiography.

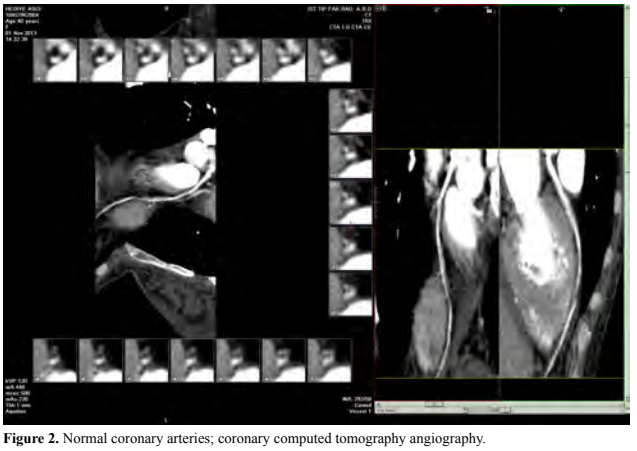

Figure 2. Normal coronary arteries; coronary computed tomography angiography.

PO-108

Unexpected guest of the heart

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Introduction: Cardiac hydatid cysts are rare and represent less than 2% of all hydatid cases. We report two cases, one of them in her late 20s, had presented with haemoptysis and shortness of breath. 84 year-old woman was the other, was admitted to our hospital with complaints of chest pain, shortness of breath, malaise, syncope.

Case 1: 28 year old girl had cats and dogs. Her physician diagnosed as cysts hydatid and referred to us if cardiac involvement present. Echocardiography and CT revealed a cyst at interventricular septum. She also had lung involvement. Albendazol was started to allow preoperative sterilization of the cyst. She operated successfully.

Case 2: 84 year old patient was living at rural area. Her thorax CT revealed cardiac and lung involvement of Echinococcus granulosus. Her relatives refused to be treated.

Discussion: Cyst hydatid is caused by the larva of Echinococcus granulosus. Cardiac echinococcosis accounting for only 0.5%–2% of all hydatid infestations. The left ventricle is involved most frequently (55–60%). Involvement of the interventricular septum is reported in 5–9% of cardiac cases. Because the condition can mimic a number of cardiac diseases, the differential diagnosis includes all other cardiac tumors and cysts, mediastinal tumor, pericardial cyst, and ventricular aneurysm. Diagnosis of cardiac hydatid cyst is simple, reliable and sensitive when transthoracic echocardiography is used. However, computed tomographic scan and magnetic resonance imaging are complementary tools to rule out multivesicular cyst. Medical treatment with albendazole, which allows preoperative sterilization of the cyst and some authors have suggested that postoperative treatment also allows a reduction of recurrences. But the treatment of hydatid cyst disease is surgical and should not be delayed and early surgery should be performed to reduce the risk of life-threatening complications.

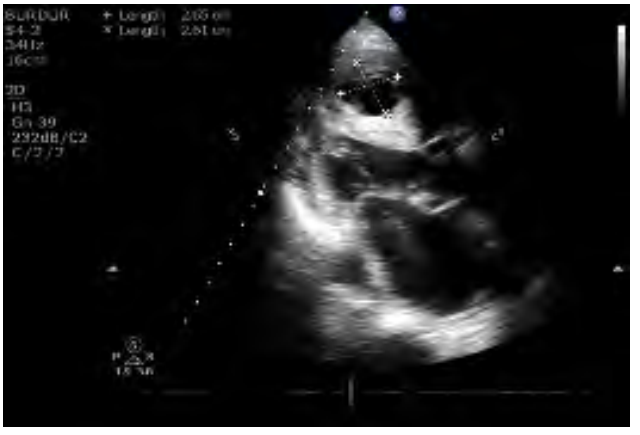

Figure 1. Echocardiography; cyst of the heart.

PO-109

Infective endocarditis case as a multisystem complication of eisenmenger syndrome (ES) developed patent ductus arteriozus (PDA) patient

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42 year old man was admitted emergency department with dyspnea,pleuretic chest pain for last months.His medical history was unremarkable. He wasn't on any medications. Physical examination (PE): tachypnea31/min, right heart overload PE findings, coarse crakles at lower right lung, clubbing on his toes, cyanosis (SPO2: %89), harsh P2,5/6 pansistolic murmur concomitant with trill that can be heard from all auscultation locations, most powerfull from tricuspid valve area. vital signs: bloodpressure: 108/78 mmHg,temperature(T) 36,8C. Electrocardiogram was 110/min sinus tachycardia, right atrial dilatation,axis deviation, ventricular hypertrophy. Laboratory values: hemoglobin16 g/dL; white blood cell(WBC)13300/l L; HTC: %53,2; C-reactive protein 48 mg/L. Chest X-ray: increased cardio-thorasic indeks, ground-glass opacification on mid right lung lobe. Transthorasic echocardiography(E): right ventricular,atrial dilatation, ventricular hyperthrophy (wall thickness:13 mm). Dilated pulmonary artery (PA), mild tricuspid regurgitation, sPAB:70 mmHg, 80 mmHg systolic gradient between PA and descenden aorta. Contrast E:significant contrast passage from PA to descenden aorta. Ventilation-perfusion sintigraphy:extensive segmental-subsegmental perfusion defects. Pulmonary CT-anjiography:emboly releated acute/chronic filling defects on both PA. Right heart catheterization: Mean PA pressure: 90 mmHg. Wedge pressure: 15 mmHg.Qp/Qs:0,81, pulmonary vascular resistance-PVR:44WU, cardiac index: 1,6L/min/m^2. Systemic vascular resistance is less than PVR.10 mm PDA. He diagnosed as PDA and ES. Patient accepted inoperabl, Bosentan 62,5 mg 2x1 as PAH (pulmonary arterial hypertension) spesific agent,Warfarin, 2x500 klaritromisin for pnomonie initiated. In the first 3 months of follow up he applied to hospital with fever,swelling left upper extremity. Functional class WHO3-4, T:38,7C. Laboratory: WBC: 15.300, crp: 35mg/dl, Pro-BNP: 4441pg/ml.Cause of notable right heart overload PE findings, agresive iv furosemid theraphy has initiated. Transthorasic E has demonstrated vegetation of tricuspit and pulmonary valves that confirmed with transoesophagial E. Blood cultures collected. 700 mg daptomisin, 3x80 mg gentamisin, 2x400 mg ciprofloksasin have initiated empirically for IE. Inhale ileoprost 7x1 added to PAH specific theraphy. After 3 days of agresive iv diuresis, he has clinically got comfort,iv furosemide has changed to oral 2x2 form, 25 mg spironolactone added for maintenance theraphy. Blood culture results showed no reproduction for any microorganism. It was associated with the prior antibiotheraphy-AB usage. During his follow-up his functional class regressed to WHO1-2, right heart overload findings disappeared with iv diuresis and spironolacton. Under the AB (1 week triple, 6 weeks daptomisin) the left upper extremity turned normal, inflamatory markers have regressed. Control E vegetations on tricuspid and pulmonary valves have considered as organized. He externalized with medical theraphy as Bosentan 2x125 mg, Ileoprost 7x1, furosemid 2x40 mg, Warfarin.

PO-110

A young patient presented wit acute coronary syndrome and intracardiac thrombus who has been founded heterozygotic for factor V Leiden mutation

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Factor V Leiden(FVL) mutation is the most common hereditary thrombophilia with a prevalence of heterozygous carriers of %3-%5. Association of this mutation with venous thromboembolism is well established. However, there are conflicting data on the association between FVL and arterial thrombosis and acute coronary syndrome. Intracardiac thrombosis (ICT) is a rare clinical condition except those associated with cardiomyopathies, atrial fibrilation and post-infarction period. A rare cause of ICT are hereditary thrombophilias and other hypercoagulable conditions. We present a male patient with a history of peripheral arterial embolism and deep venous thrombosis, presented to the emergency department with worsening chest pain for 3 hours. His troponin level was high and after initial evaluation he was diagnosed as myocardial infarction without ST- segment elevation. Transthoracic echocardiography revealed 4,1x2,1 cm intracardiac mobile thrombosis. Coronary angiography revealed non-critical stenosis. Since the thrombus was relatively large and mobile, surgical thrombectomy was preferred in order to avoid the risk of systemic embolization. The surgery was performed successfully, the patient is discharged and anti-coagulated. Afterwards folllowed in outpatient clinic.

Figure 1. Echo images; intracardiac thrombus shown with TTE.

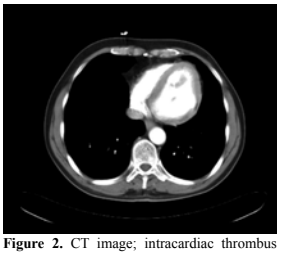

Figure 2. CT image; intracardiac thrombus shown with CT.

PO-111

Nadir bir göğüs ağrısı başvuru nedeni; idiyopatik pulmoner arter anevrizması

Ahmet Gürdal, Mutlu Çağan Sümerkan, Süleyman Sezai Yıldız, Kudret Keskin, Gökhan Aksan, Serhat Sığırcı, Arzu Er

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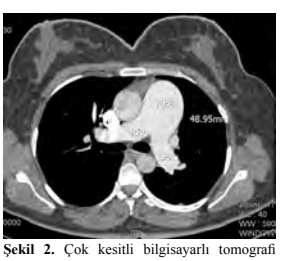
Pulmoner arter anevrizması (PAA) nadir görülmektedir. Burada polikliniğe göğüs ağrısı ile başvuran idiyopatik PAA olgusu sunuldu.

Olgu: 47 yaşında bayan hasta polikliniğe eforla ortaya çıkan göğüs ağrısı şikayeti ile başvurdu. Kardiyovasküler sistem muayenesi normaldi. 5 yıldır hafif bronşiyal astım tanılı hastanın akciğer muayenesinde hafif derecede ronküs duyuluyordu. Transtorasik ekokardiyografide sağ ve sol kalp boyutları ve fonksiyonları normaldi; fakat ana pulmoner ve dalları ileri derecede genişlemişti. (Şekil 1) Patent duktus arteriyozus, atriyal septal defekt ve trombüs ya da kitle saptanmadı. Doppler ekokardiyografide hafif triküspit yetersizlik akımı üzerinden ölçülen sistolik pulmoner arter basıncı 35 mmHg saptandı.Hastanın fizik muayenesinde ve biyokimyasal tetkiklerinde sistemik hastalık veya vaskulit lehine bulgu izlenmedi. Behçet hastalığı açısından yapılan tetkiklerde anlamlı bulguya rastlanmadı. Çok kesitli bilgisayarlı tomografisinde ana pulmoner arter (MPA) çapı 49 mm, sağ ana pulmoner arter (RPA) çapı 30 mm, sol ana pulmoner arter (LPA) çapı 25 mm ölçüldü, pulmoner arter ve dallarında trombüse rastlanmadı, akciğer parankimi normal olarak değerlendirildi. Cerrahi tedavinin riskleri ve uzun dönem sonuçlarının iyi tanımlanmamasından dolayı hastanın takibine karar verildi.

Tartışma: Oldukça nadir görülen PAA etyolojisinden birçok faktör sorumlu tutulmaktadır. Pulmoner hipertansiyona, çeşitli enfeksiyonlar (sifiliz, tüberküloz), bağ dokusu hastalıkları (Marfan sendromu vb..), kistik mediyal dejenerasyon, Behçet hastalığı PAA' ya yol açabilmektedir. Semptomatik hastalar nefes darlığı, atipik göğüs ağrısı, ateş, öküsürük, hemoptizi gibi non-spesifik şikayetlerle başvurabilirler. İdyopatik PAA'nın uzun dönem sonuçları, takip ve tedavi yöntemleri, cerrahi girişim endikasyonları ve zamanlaması konusunda net veri bulunmamaktadır. PAA, göğüs ağrısı ile başvuran hastalarda göz önünde bulundurulması gereken nadir bir durumdur.

Şekil 1. Transtorasik ekokardiyografik görüntü.



Şekil 2. Çok kesitli bilgisayarlı tomografi görüntüsü.

PO-112

Ekokardiyografi ile tanıya varılan akut sağ kalp yetmezliği kliniğinde Hepatosellüler Karsinom olgusu

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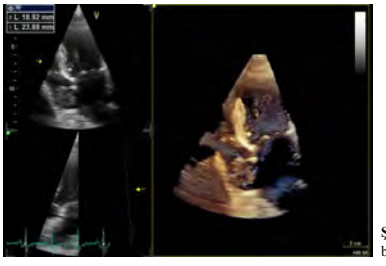
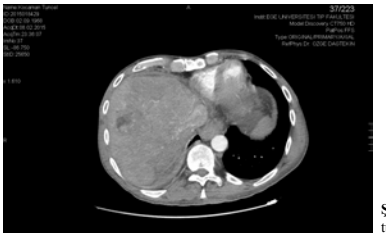
Bilinen kronik hastalık öyküsü olmayan 47 yaşında erkek hasta, 4 gündür olan ve giderek artan bacaklarda ve karında şişlik, karın ağrısı şikayetleri ile acil servise başvurmuş. Hasta akut sağ kalp yetmezliği açısından tarafımıza konsulte edildi. Yapılan ekokardiyografide VCİ'den sağ atriuma uzanım gösteren,sağ atriumda 18,92x23,69 mm boyutlarında tümör trombüsü ile uyumlu kitle izlendi (Şekil 1,video 1,2). Hastaya Hepatosellüler Karsinom (HCC) ? açısından batın BT çekilmesi önerildi. Batın BT'sinde sirotik karaciğer, karaciğer sağ lobta yaygın HCC'yi düşündüren odaklar, sağ hepatik ven yoluyla inferior vena cavaya ulaşmış tümör trombüsünü düşündüren lezyon gözlenen olguda sağda plevral effüzyon ve batın içerisinde yaygın asit sıvısı izlendi (Şekil 2). Hasta gastroenteroloji servisinde takip edildi. Bakılan alfa-fetoprotein (AFP) düzeyi 460 ng/ml (yüksek) saptandı. Hastadan portal ven ve vena kava inferior değerlendirilmesi açısından dopler USG istendi, diffuz HCC'ye bağlı portal venin intrahepatik dallarında, sağ hepatik ven ve VCİ'de tümör trombusleri izlendi. Viral serolojide HBV (+), viral yük açısından bakılan HBV DNA: 5194 IU/ml saptandı. Hasta Gastroenteroloji kliniği Hepatobilier konseyinde tartışıldı, cerrahi açıdan inoperabl kabul edildi, kemoterapi planlanmadı. Medikal tedavisi düzenlenerek taburcu edildi.



Şekil 1. RA'da 18,92x23,69 mm boyutlarında tümör trombüsü.



Şekil 2. VCİ'de ve karaciğerde yaygın tümör odakları.

PO-113

Sinus venosus atrial septal defect with partially anomalous pulmonary venous return and bifid coronary artery in an old patient: a case report

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Congenital atrial septal defects are not so rare and are usually diagnosed at third or fourth decades, but not in patients of advanced ages. We present a 71 year-old female patient with progressive exertional dyspnea. Transthoracic echocardiogram showed right ventricular hypertrophy and dilation and pulmonary hypertension suggesting atrial septal defect and also hypokinesis in interventricular septum. By transesophageal echocardiogram, sinus venosus type atrial septal defect 17 mm in size was detected. For further research, cardiac magnetic resonance was performed and showed sinus venosus type atrial septal defect 18 mm in size with right superior pulmonary vein draining into superior vena cava, so called partially anomalous pulmonary venous return, and subendocardial late gadolinium enhancement in interventricular septum indicating myocardial ischemia. On this basis, coronary angiography was performed and bifid left anterior descending artery was detected with a dominant diagonal artery and a non-dominant left anterior descending artery, that was evaluated as chronic total occlusion and responsible for subendocardial late gadolinium enhancement in interventricular septum. Bifid coronary artery may be a component of this congenital cardiac defect, that is the first report in the literature or may be incidentally diagnosed. The patient was not operated in contrast to the literature because of advanced age, and is followed by medical therapy alone. This is a case report of a rare cardiac pathology and highlights the importance of cardiac MRI in detection of complex congenital cardiac anomalies.

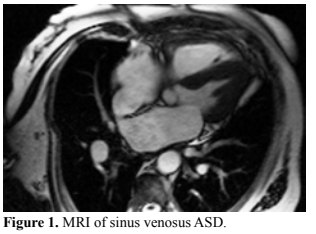

Figure 1. MRI of sinus venosus ASD.

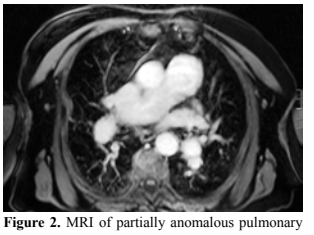

Figure 2. MRI of partially anomalous pulmonary venous return.

PO-114

An unusual cause of syncop; mitral valve myxoma

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A 52 year old woman was admitted to the hospital with complaints of sudden syncope and with a history of palpitations on efforts and progressive dyspnea over the previous three years. On physical examination An intense protodiastolic murmur, where a proto-meso systolic murmur and where a diastolic rumble were also heard in the precordium.. Transthoracic echocardiogram revealed an echogenic mass and the mass could be seen attached to the posterior leaflet of the mitral valve that prolapsed into the left ventricle in diastole (Figure 1). The mitral valve mean gradient was 18 mmHg and functional mitral valve area was 1 cm², TEE was performed and showed a sessile round mass with regular contours and homogeneous appearance, measuring 3,2x2,75 cm, attached to the atrial side of the posterior mitral valve leaflet (Figure 2, video 1). it caused significant obstruction and mild regurgitation of the mitral valve (video 2). Emergent surgical resection was performed,. Pathological analysis showed destruction of the posterior leaflet of the mitral valve by a vegetating mass. Final histological diagnosis was myxoma. The patient was discharged without any complications and remains well after 3 months of follow-up Valvular tumors are very rare. The tricuspid valve is the most frequently involved location, followed by the mitral, pulmonic and aortic valve. About 75% of myxomas originate from the left atrium, 18% in the right atrium, and 4% in the ventricle. The exact incidence of myxomas originating from the mitral valve is not clear. The most frequently seen manifestation in myxomas of the mitral valve was cerebral or peripheral embolism.

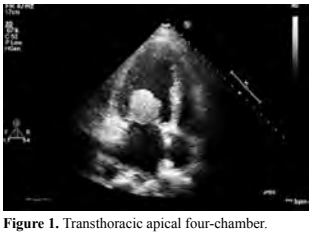

Figure 1. Transthoracic apical four-chamber.

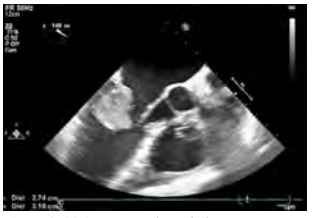

Figure 2. 145º transoesophageal views.

PO-115

Bioprostethic valve thrombosis due to oral contraceptive drug use successfully treated with thrombolytic therapy

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[1]*Kartal Koşuyolu Yüksek İhtisas Training and Research Hospital, İstanbul*
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Background: Prosthetic valve thrombosis is a severe complication, which usually occurs in inadequately anticoagulated patients. Mechanical valve thrombosis is more common than bioprosthetic valve thrombosis (BVT). Oral contraceptive drugs are associated with increased risk of thromboembolism in women. BVT due to oral contraceptive drug use managed with thrombolytic therapy (TT) has never been reported Purpose: We present a case of obstructive BVT occurring after use of an oral contraceptive drug who was successfully managed with TT Methods: A 44-year-old woman who had undergone bioprostethic mitral valve replacement 6 years earlier was admitted to our hospital with NYHA class 4 dyspnea. She had been given up warfarin 6 months after surgery and treated with acetylsalicylic acid (100 mg/day) thereafter. She had started to take an oral contraceptive drug including ethinyl estradiol and desogestrel 1 month before admission. Transthoracic echocardiography (TTE) and subsequently 2-dimensional (2D) and real-time 3-dimensional (RT3D) transesophageal echocardiography (TEE) were performed at baseline for evaluation of prosthetic mitral valve. She was treated with successive low-dose (25 mg) and slow infusion (6 hours) of tissue plasminogen activator (t-PA) without bolus under the guidance of serial TEE Results:TTE revealed increased mean transvalvular gradient of 24 mmHg, decreased valve area of 1.6 cm² and normal left ventricular ejection fraction. Immediate 2D and RT3D TEE showed a large BVT (1,44 cm²) (Fig. 1a and b). After the second TT session normalization of mean gradient (5 mmHg) and valve area (2.3 cm²) with complete lysis of the thrombus was observed without any complication (Fig 1c) Conclusion: Oral contraceptive drug use may increase the risk of thrombosis in women and should be used cautiously in patients with prosthetic heart valves. TT with low-dose (25 mg), slow-infusion (6 hours) of t-PA may be an effective and safe treatment option in patients with BVT.

Figure 1. (A-C) TEE showed large thrombus and complete lysis.

PO-116

Akut pankreatit sonrası beklenmeyen durum; kalp içi trombüs

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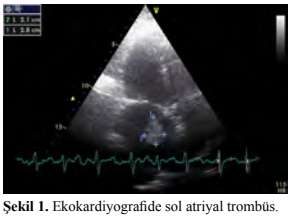
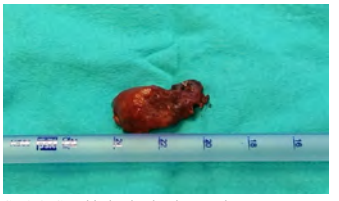
Dr. Siyami Ersek Göğüs, Kalp ve Damar Cerrahisi Eğ. ve Ar. Hastanesi, İstanbul

Akut pankreatit sonrası sol atrial (SİA) trombüs nadir görülen bir durumdur. Akut pankreatitin (AP) sistemik protrombotik süreci tetiklemesi ile altta yatan risk faktörü olan hastalarda intrakardiyak trombüs oluşum süreci hızlanabilir. Elli üç yaşında kadın hasta kliniğimize nefes darlığı şikayeti ile başvurdu. Atrial fibrilasyon (AF) nedeniyle warfarin kullanmakta olan hastanın bir ay önce akut pankreatit tanısı ile dahiliye kliniğinde medikal tedavi gördüğü öğrenildi. Laboratuvar incelemesinde hemogram ve kan biyokimyasında önemli bir özellik saptanmayan hastanın son beş aylık takibinde INR değerlerinin 2-3 aralığında olduğu görüldü. 5 ay önce yapılan transtorasik ekokardiyografisinde (TTE); Ejeksiyon fraksiyonu (EF): %60, romatizmal mitral kapak, orta mitral stenoz (MS), orta mitral yetersizlik (MY), Wilkins skoru: 12 saptanan hastanın nefes darlığı şikayetinin bulunması üzerine TTE'si yenilendi. EF: %60, romatizmal mitral kapak, orta MS, orta ileri MY, SlA lateral duvarına tutunan ve sol atrial apendikse (SIAA) uzanım gösterdiği izlenen 2,8x2,1 cm çaplarında kitle saptandı. Bunun üzerine hastaya transözefageal ekokardiyografi (TEE) yapıldı. TEE'de SlAA lateral duvarından köken alan SlA posterioruna uzanım gösteren 5 cm2 büyüklüğünde kitle görüntülendi. Hasta kalp ve damar cerrahisi ile birlikte değerlendirildi ameliyat kararı verildi. Operasyona alınan hastada sol atriumdaki 2.0x1.5 cm boyutlarındaki trombüs ile uyumlu olan kitle çıkarıldı. Mitral kapak eksize edilerek yerine 29 numara Carbomedics yapay metal mitral kapak yerleştirildi. Kitlenin patolojik incelemesi de trombüs ile uyumlu olarak saptandı. AF ritminde olan hastalarda SIA ve SIAA'da trombüs sık görülür. Bu hastalarda ortaya çıkan akut inflamatuar süreçler trombüs oluşumunu hızlandırabilir. AF ritminde olan hastalar warfarin ile takip ediliyor olsalar dahi AP gibi akut inflamatuar süreçlerde hastaların intrakardiyak trombüs açısından daha sık ekokardiyografik takibi önemlidir.



Şekil 1. Ekokardiyografide sol atriyal trombüs.



Şekil 2. Cerrahi olarak çıkarılan trombüsün görüntüsü.

PO-117

Mobile mass lesion in the aorta after transcatheter aortic valve implantation: thrombus or residue calcification

Sait Demirkol, Cengiz Özturk, Mustafa Demir, Ali Osman Yıldırım, Şevket Balta, Turgay Çelik, Atila İyisoy

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Transcatheter aortic valve implantation (TAVI) is an alternative and effective treatment in patients with severe symptomatic aortic stenosis and high surgical risks. Even though TAVI is a less invasive treatment compared to the surgical aortic valve replacement, serious complications may develop. An 85 year old male patient admitted to our clinic due to complaints of shortness of breath, palpitation and dizziness. There were a 3/6 systolic ejection murmur radiates to the neck and weakiness of the peripheric pulses in lower extremities on physical examination. On transthoracic echocardiography, LVEF 60%, LV diameters were normal, mild to moderate aortic regurgitation and serious aortic stenosis (max/mean PG: 92/56 mmHg) were present. In 3D CT angiography, femoral artery was appropriate for intervention, aortic annulus diameter was 21 mm and aortic valves were calcific. The logistic EuroSCORE II was calculated 18. Because of the high risk for surgery, TAVI was decided. 23 mm Edwards Saphien XT valve was implanted without any complication. One month later, the patient was admitted again with the complaint of shortness of breath. In control two-dimensional transthoracic echocardiography, moving lesion was detected in aortic valve on the side of aorta. On transesophageal echocardiography with the view of upper oesophageal long axis (Figure-1) and short axis (Figure-1),about 10X5 mm semihyperechogenic and semihypoechogenic moving mass lesion was detected. In 3D transesophageal echocardiography, moving lesion was observed on zoom mode (Figure-1,video-1). As an initial diagnosis, trombus or residue calcification related to the natural valve was thought. The patient was anticoagulated for one month but it wasn't regressed. The patient is followed up with current anticoagulant therapy. Because of potential complications of TAVI procedure, the patient should be followed up closely during and after the procedure.

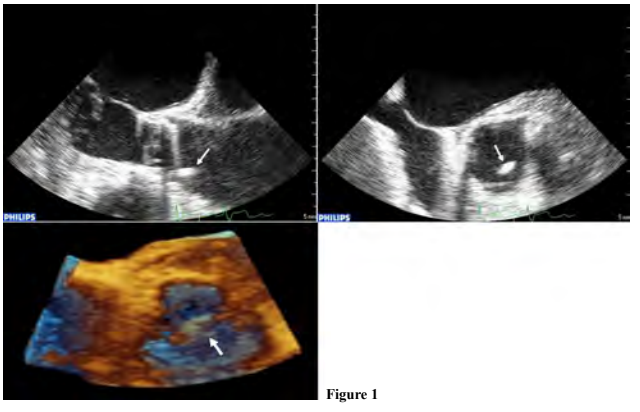


Figure 1

PO-118

Pacemaker dysfunction and syncope: an unusual presentation of pacemaker lead endocarditis

İsa Öner Yüksel, Erkan Köklü, Şakir Arslan, Göksel Çağırcı, Selçuk Küçükseymen, Nermin Bayar, Zehra Erkal, Görkem Kuş, Murat Esin

Antalya Education and Research Hospital, Antalya

An 80-year-old female was admitted for progressively fatigue and syncope of three months. She had undergone DDD pacemaker implantation for complete atrioventricular block one years ago. Upon admission, the patient was afebrile with a temperature of 36.3°C, weakness and had general debility. Laboratory investigations revealed elevated C reactive protein (48.7 mg/dl) and sedimentation velocity (75/1°h), normochromic normocytic anemia (haemoglobin 9.1 g/dl) and creatinine levels estimating creatinine clearance at 73 ml/min. Blood cultures taken at admission (three samples) and repeated later on were consistently negative for microorganisims. On admission, the electrocardiogram showed pacemaker rhythm but there were occasional sensing failure on pace and heart rate was 50/min (Fig. 1). Pacemaker, atrial and ventricular leads were checked under fluoroscopy because of the intermittent pace dysfunction. Pace and sense measurements were made, but did not observe any problems. Transthoracic echocardiography (TTE) revealed the vegetation was observed on ventricular and atrial faces of right ventricular lead (Fig. 2). On the same day, the temporary pacemaker was implanted via jugular vein and previous pacemaker and leads were completely removed. Cultures were taken from vegetation attached to the end of the pacemaker and the other three separate piece of device, also three blood cultures were taken consecutively. Three blood cultures, throat and urine cultures were unremarkable, but Pseudomonas aeruginos were detected from the vegetation attached to the end of pace. The patient was treated with Ciprofloxacin every 6 hours. On the fourteenth day, the new DDD-R pacemaker was placed successfully from the other side and temporary pace was rolled back. Two weeks following new pacemaker implantation, patient remains haemodynamically stable, apyretic, asymptomatic, with no syncopal or presyncopal episodes have been reported. She remains in pacemaker rhythm and no cardiac arrhythmias and pace dysfunction.

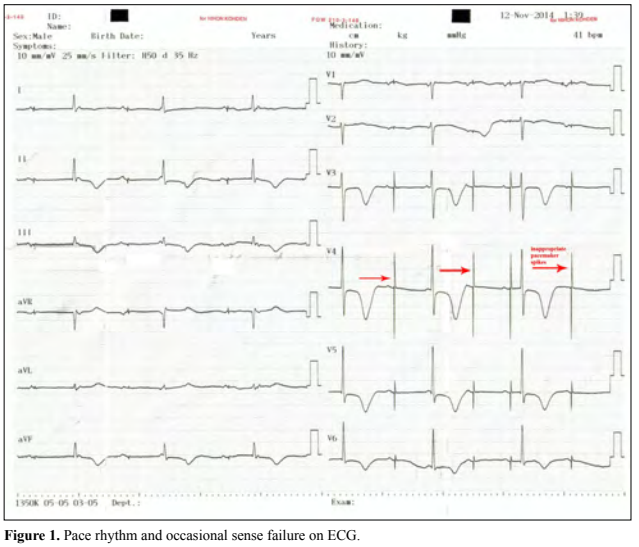


Figure 1. Pace rhythm and occasional sense failure on ECG.

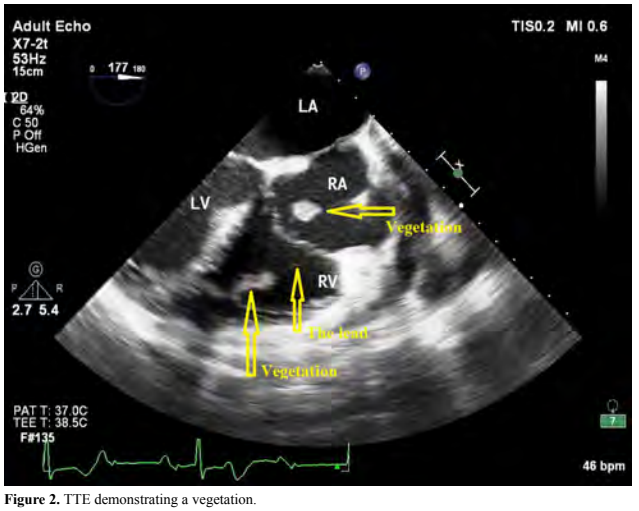


Figure 2. TTE demonstrating a vegetation.

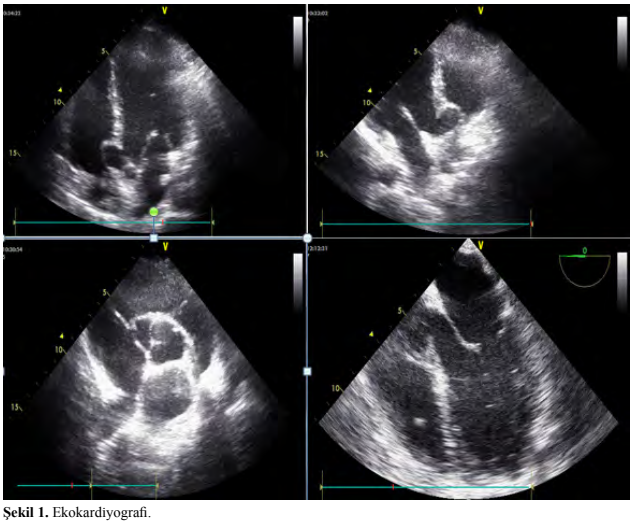
PO-119

Ventriküler septal anevrizma ile kapanmış ventriküler septal defekt

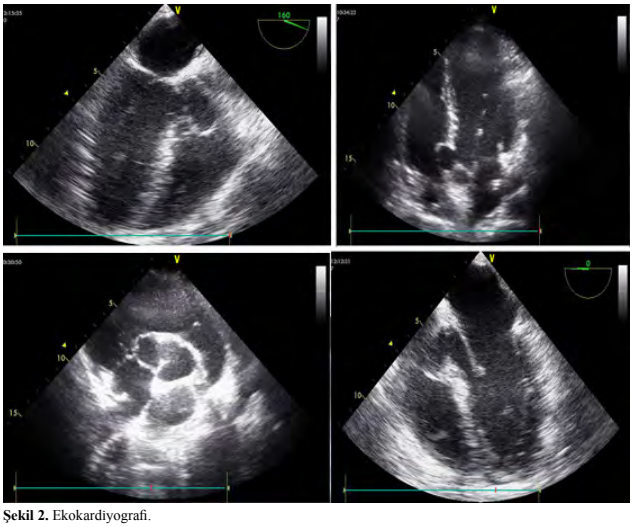
Mustafa Gök, Aziz İnan Çelik, Aslıgül Cüreoğlu, Ali Deniz, Rabia Akıllı Eker, Onur Sinan Deveci, Çağlar Emre Çağlıyan, Mesut Demir, Ayhan Usal, Mehmet Kanadaşı

Çukurova Üniversitesi Tıp Fakültesi, Adana

30 yaşında belirgin sistemik hastalığı olmayan hasta son zamanlarda olan atipik göğüs ağrısı ve çarpıntı ile başvurdu. Kardiyovasküler sistem muayenesinde aort odağında erken diyastolik üfürüm mevcuttu. Ekokardiyografi de: İnterventriküler septumda perimembranöz outlet bölgede ventriküler septal anevrizma ile kapanmış ventriküler septal defekt görünümü mevcuttu. Aortik kapak üç küspitli, eser aort yetersizliği ve koroner arter çıkışları normal yerinde olarak değerlendirildi.



Şekil 1. Ekokardiyografi.



Şekil 2. Ekokardiyografi.

PO-120

Positional atrial sense failure triggeringmultiple inappropriate rate-drop responses

Recep Hacı, Nizamettin Selçuk Yelgeç, Ahmet Taha Alper, Ahmet İlker Tekkeşin, Ceyhan Türkkan

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Selected patients with Cardio-inhibitory reflex syncope may benefit from cardiac pacemakers with rate drop algorithms. But sometimes inappropriate rate-drop responses may present challenging diagnostic problems. Thirty years old woman presented with frequent syncope episodes without apparent reason. A tilt test then performed. During the tilt test she had a pause of 25 seconds and she had a syncope episode simultaneously with the pause very similar to previous episodes. We implanted a permanent pacemaker with a rate-drop algorithm. During the first week of implantation she complained tachycardia attacks of short duration particularly when she was asleep. Interrogation of the device with telemetry did not diagnose any problem with atrial and ventricular sense, capture and impedance parameters, but telemetry has recorded five rate-drop episodes in

one week. We reprogrammed the rate-drop algorithm. But she continued to have recurrent short tachycardia attacks when she was asleep though. She said she increased pillow number to prevent tachycardia attacks. Then pacemaker controls repeated when she was laying supine and sitting position. In supine position atrial sense values were low and were about 0, 7 mV, in sitting position it was approximately 4 mV. There was positional atrial undersensing because of lead dislodgement when she was laying supine that caused the pacemaker interpret it as atrial asystole and triggering a rate-drop response. We corrected atrial lead by second intervention and her tachycardia attacks disappeared afterwards. Since rate-drop algorithm works generally in AAI mode, atrial sense failures may be interpreted by pacemaker as sinus pause triggering rate-drop response. So if a patient with rate-drop response pacemaker describes frequent nocturnal tachycardia attacks developed after pacemaker implantation we must think the possibility of intermittent atrial sense failure, and we must check atrial sense values for both supine and erect position.

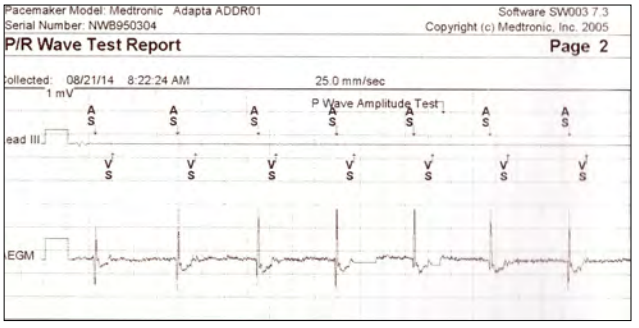

Figure 1. Atrial EGM; atrial sense in sitting position it was about 4 mV.

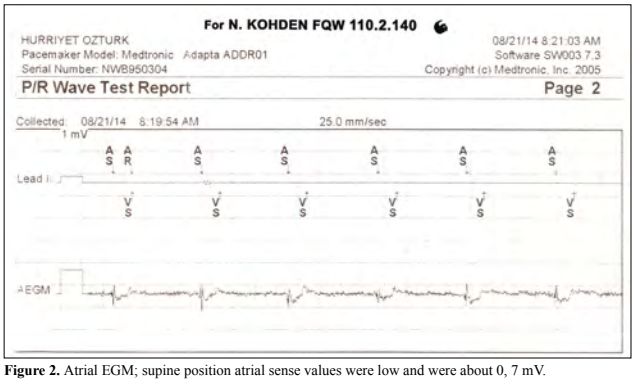

Figure 2. Atrial EGM; supine position atrial sense values were low and were about 0, 7 mV.

PO-121

The impact of convenient resetting on life quality

Çağın Mustafa Üreyen, Şakir Arslan, Göksel Çağırcı, Cem Yunus Baş, İsa Öner Yüksel, Zehra Erkal, Görkem Kuş, Emre Asiltürk

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A 24-year-old female patient visited our outpatient pacemaker clinic for her routine pacemaker follow-up. DDD pacemaker was implanted due to atrioventricular (AV) block following mitral valve replacement three years ago. She has been on follow-up by industry represantatives for three years. On questioning, she denied any significant complaint and was not taking any medication except warfarin. Her physical examination was quite normal, other than, metallic prosthesis sound. Her electrocardiography (ECG) denoted sinus rhythm. During interrogation of the pacemaker, all parameters including pacing thresholds, sensed P/R wave amplitudes, lead empedances and battery voltage were all at normal limits, however, it was noticed that postventricular atrial refractory period (PVARP) and atrioventricular interval (AVI) were set to 400 milliseconds (msec) and 200 msec, respectively. Hence, total atrial refractory period (TARP) was 600 msec. Moreover, upper tracking rate (UTR) was set to 130/min which was low for a young patient. Owing to the duration of TARP, the pacemaker was allowing 2:1 AV block when heart rate exceeds 100/min even before Wenkebach upper rate response. Records of pacemaker were inspected and there were three consecutive alertsof pacemaker mediated tachycardia (PMT), albeit none of alerts of PMT was true. The device was adjusted by industry representatives and the PVARP was increased in the prior follow-ups based on PMT alert without controlling whether the alert was true or not. These pacemaker settings rendered a further questioning for patient's effort capacity.She admitted her unability to climb more than two flights of stairs due to exertional dyspnea, however, she was not complaining of her low effort capacity by virtue of the fact that she have adapted a more sedantary life style, perceiving this as her normal life standart. That's why she denied any complaint on the first questioning. Ventriculoatrial conduction time was measured as 174 msec with manufacturer's special programmer, thence, the PVARP was set to 200 msec. Furthermore, AVI and UTR were set to 150 msec and 160/min, respectively. Two months later, she was joyful about the results and the effort capacity was incremented to climbing five flights of stairs without any complaint. Moreover, she admitted starting jogging and socializing more in her daily life, quoting her happiness for feeling "normal" again. This case emphasises the importance of follow-up and optimisation of pacemakers that should be executed by cardiologists. As in the above-mentioned case report, it was demonstrated that how simple, albeit effective and proper resettings can alter patients' life quality dramatically and resocialize them and make them individuals of our society again.

PO-122

Heart needs two weeks to forget; a case report about cardiac memory

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At her last control of a 32 years old lady who had a dual chamber pacemaker (PM) implanted 10 years ago due to sick sinus syndrome, generator depletion and "elective replacement time" warning was detected. She was PM dependent and the device was found switched to VVI mode (figure 1) while it was on AAI mode throughout the follow-up until the previous visit of three months ago. After replacement, the new PM generator's "managed ventricular pacing, MVP" function allowed to atrial pacing, ventricular sensing and a narrow QRS rhythm. But this time on ECG recordings there were deep T wave inversions on D2-3-aVF and precordial leads (Figure 2). For ruling out of the casues other than electrical remodeling such as myocardial ischemia, pericarditis etc, echocardiography, serum cardiac marker measuring and exercise test were performed. All the results were normal. Therefore repolarisation abnormalities on ECG considered as electrical remodeling which also called as 'cardiac memory' due to right ventricular pacing lasted for a time in between one day to three months. Patient's ECGs recorded following two weeks with 3 days intervals until all T wave invertions gradually disappeared (Figure 3,4,5). Cardiac memory refers to particular type of electrical remodeling is expressed as an altered T wave on ECG that occurs after a period of altered activation induced by ventriculer pacing or arrhythmia.These changes were associated with alterations of potassium channels specially 'Ito' channels implying that electrical remodeling is heterogeneously expressed in the different cell types across the transmural wall. Finally, remodeling of gap junctions may also play a prominent role in action potential changes during remodeling. Clinicians need to be aware of cardiac memory since it can imitate myocardial ischemia and suggesting that remodeling of ventricular myocardium can enhance arrhythmia susceptibility.

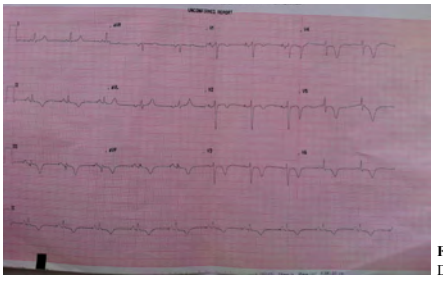

Figure 1. T wave inversions on D2-3-aVF and precordial lead.

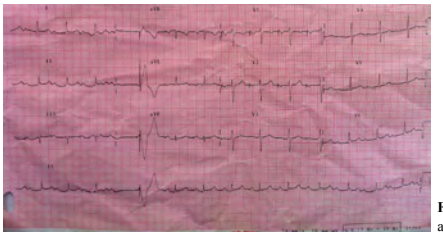

Figure 2. T wave invertions disappeared on last ECG.

PO-123

Recurrent Torsades de pointes due to amiodarone toxicity treated succesfully with lidocaine

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Amiodarone is a potent antiarrhythmic agent that is used to treat ventricular arrhythmias and atrial fibrillation. Proarrythmias due to amiodarone therapy is rare. We discuss a case who had amiodarone induced incessant Torsades de Pointes (TdP), that could only be suppressed by lidocaine. The patient was a 56 years -old woman who had benthall operation for aortic aneurysm. She was prescribed amiodarone for rhythm control because of paroxysmal atrial fibrillation. After many years without any problem one day she was brought to a private hospital's emergency department with syncope. ECG revealed polymorphic tachycardia and long QT. Physicians did not recognize that these arrhythmias were due to amiodarone induced proarrhythmia but paradoxically increased amiodarone dose to control these arrhythmias. Then she was referred to our hospital. Here she continued to experience frequent new episodes of TdP and multifocal ventricular extrasystoles. These arrhythmias were responsive to DC cardioversion and transvenous overdrive pacing but although potassium and magnesium supplements were given the arrhythmia was not controlled and she continued to have recurrent episodes of TdP. Her ECG showed very long cQT interval about 600 msn, very frequent long QT-related multifocal ventricular ectopic beats and recurrent attacks of TdP. We decided to try lidocaine to control this VT storm since lidocaine has no proarrhythmic potential and shortens QT interval in contrast to other class I and class III antiarrhythmic drugs. Fortunately three hours after we started the standard dose of lidocaine her QT shortened dramatically, and her polymorphic ventricular tachycardia were controlled and suppressed successfully. Her rhythm was stabilized First-line treatment for acquired long QT syndrome is intravenous magnesium sulfate that is highly effective for both the treatment and prevention of recurrence of long QT-related ventricular ectopic beats or TdP. Temporary transvenous overdrive pacing or isoproterenol generally is reserved for patients with long QT-related TdP who do not respond to intravenous magnesium. We do not administer the usual antiarrhythmic agents for ventricular tachycardia to a patient whose rhythm has deteriorated into TdP, because other drugs used for conventional VT treatment will prolong the QT interval further. Exception might be class IB drugs. Class IB antiarrhythmic drugs, such as lidocaine, phenytoin and mexiletine may be helpful for suppressing torsades, they shorten action potential duration and, based upon small case series, may be effective in the acute management of TdP and ventricular fibrillation. But class 1B antiarrhythmic drugs appear to be less predictably effective than pacing or isoproterenol. This case demonstrates that Class 1B antiarrhythmic agents have an important place for the treatment of acquired long QT induced resistant arrhythmias and should be considered as a valuable option when standard therapy fails to control long QT induced recurrent TdP.

PO-124

Follow-up and optimisation of cardiac resynchronization therapy by industry represantatives: is it reliable?

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A 64-year-old male patient was hospitalized to cardiology ward due to decompensated heart failure. On physical examination, bilateral basilar crackles were oscultated and +2 pretibial edema was seen. On echocardiography, EF was 25% with a dilated left ventricle and mitral regurgitation of moderate to severe. His ECG demonstrated sinus rhythm, ventricular pacing spikes and left bundle branch block (LBBB) with a QRS duration of 202 msec. His prior ECG before implantation denoted LBBB with a QRS duration of 153 msec. He had been already taking perindopril/indapamid 10/2.5 mg, carvedilol 25 mg b.i.d, spironolactone 50 mg, furosemide 40 mg, acetylsalicylic acid 100 mg, rosuvastatin 20 mg and digoxin 0.25 mg. The patient remarked that a cardiac resynchronization therapy with defibrillator (CRT-D) was implanted 5 months ago. On questioning, he had hospitalizations approximately every two months before CRT-D implantation. However, he emphasised that the frequency of hospitalizations increased after CRT-D. During interrogation of CRT-D, pacing thresholds, sensed P/R wave amplitudes and lead empedances of atrium and right ventricle were all normal as well as battery voltage. Contrary to these findings, coronary sinus (CS) lead's pacing threshold was so high that even 7.5 volt was not sufficient to pace. Therefore, only RV pacing was present with a 98% pacing ratio due to short AVI setting (120 msec). Pacing mode was shut down and only defibrillator mode was kept on. On questioning, the patient told that his prior physician recommended surgical implantation of one of the leads one day after implantation due to malfunctioning and unsuitable vascular anatomy, albeit he refused. We again recommeded surgical implantation of CS lead, however, he again refused. Thus, only ICD function was kept on. RV pacing even in patients with normal EF increases risk of heart failure and atrial fibrillation on long term by virtue of nonphysiological pacing. Most probably, our patient did not have an appropriate coronary sinus anatomy, thus, LV pacing could not be achieved. However, unnecessary RV pacing should not have been allowed after patient's refusal of surgical CS lead implantation. RV pacing further deteroriated left ventricular function and made the patient more symptomatic. CRT of this case was completely optimised and followed-up by industry represantatives. Therefore, we hereby stress and demonstrate how convenient follow-up and optimisation can be so substantial and wrong settings can deteriorate patients' life quality and prognosis. Consequently, cardiologists dealing with device implantation should also be trained to have satisfactory knowledge regarding optimisation and follow-up of implantable cardiac devices and should execute optimisation and follow-up by themselves.

PO-125

The significance of proper setting of implantable cardiac devices

Çağın Mustafa Üreyen, Şakir Erslan, Göksel Çağırcı, Cem Yunus Baş, Selçuk Küçükseymen, Görkem Kuş, Emre Asiltürk, Murat Esin, Gamze Korkmaz, Özkan Kayhan

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A 67-year-old male patient with a history of coronary artery disease applied to our outpatient pacemaker clinic for follow-up. It was his first time being controlled in our clinic due to immigration. An ICD was implanted two years ago for primary prevention of sudden cardiac death in an other hospital. He was taking 100 mg of acetylsalicylic acid, 12.5 mg of carvedilol b.i.d, 32 mg of candesartan, 80 mg of atorvastatin, 25 mg of spironolactone, 40 mg of furosemide t.i.d, 0.25 mg of digoxin and 200 mg of amiodarone. His physical examination was unremarkable except pretibial edema of +1. His ECG revealed sinus rhythm with ventricular pacing at a rate of 60/min. Interrogation of ICD displayed that pacing threshold, sensed R wave amplitude, lead empedance and battery voltage were normal.Three convenient shocks were delivered 19-19.5 and 20 months ago. Contrary to these findings, ventricular pacing (Vp) ratio was determined 51%. Lower rate was determined to be set to 60/min which was its factory default setting and it had been most probably never changed after its implantation. On questioning, it was ascertained that the patient was not using digoxin and he was using single dose of furosemide 40 mg daily before implantation. Moreover, hospitalisation intervals had become more frequent after implantation. We could not compare left ventricular EF and diastolic and systolic diameters of pre- and post-implantation by virtue of absence of previous echocardiography report. Nonetheless, it was certain that there was deterioration of patient's status either due to disease's natural period or this unsuitable setting. Recording of Vp ratio was initialized and lower rate was set to 40/min due to absence of any history of necessity of pacing. After this resetting, patient's intrinsic ventricular pacing was obtained with a rate of 55/min and with a QRS duration of 92 msec. The patient was invited for a visit after six months. In the second follow-up after six months, the patient's heart rate was 53/min and Vp ratio was ascertained lesser than 1%. The patient answered that he was taking one or two pills of furosemide daily and he remarked that he was able to go to praying more frequently and comfortably than before. Implantation of a device is only the beginning of a tough way. To make a good start is very important, however, to keep on going succesfully on this tough way is at least or more vitally substantial than implantation of a device. A partial or zero benefit from a successfully implanted device would give nothing to patients concerning life quality and prognosis. Hence, cardiologists dealing with device implantation should also be trained to have satisfactory knowledge regarding optimisation and follow-up of implantable cardiac devices. Because inadequate experience would potentially result in inconvenient optimisation and follow-up that would lead to deterioration of patients' life quality and prognosis.

Aritmi / Elektrofizyoloji / Pacemaker /CRT-ICD

PO-126

Development of ST elevation myocardial infarction and atrial fibrillation after an electrical injury

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Introduction: Cardiac effects of electric shock include accelerated hypertension, arrhythmia, acute myocardial infarction (MI), and cardiac rupture. Ventricular premature beats, ventricular tachycardia, ventricular fibrillation, atrial tachycardia, atrial fibrillation (AF), bundle branch blocks, and AV block can be seen as the arrhythmic complications of electric shock. MI is one of the rarely seen complications which may lead to fatal outcomes. In our case report, we presented a patient who developed ST elevation MI with atrial fibrillation after an electric shock.

Case: 50-year-old male patient admitted to the emergency because of an electric shock which occurred 15 minutes before (alternative current, 50 Hz, 220 V). The patient indicated that as soon as he touched the wall plug to switch on light, he was shocked from his right hand and found himself lying on the floor of his home. He stated that he had lost his consciousness with the shock and after recovery he had found out he had a tightening chest pain radiating to his left arm. His pain had continued increasingly. Sinus tachycardia, increase in the amplitude of the T-wave at inferior derivations, and ventricular premature beats were monitored in the performed ECG. Nearly after ten minutes, atrial fibrillation developed with a sharpened chest pain. In the performed ECG, ST segment elevation in inferior derivations and ST segment depression in V1–V3 derivations were observed (Figure 1). In order to maintain the ventricular rate under control, intravenous beta-blocker was applied to the patient whose blood pressure was stable. Before coronary angiography in the hospital where he transferred, the patient's chest pain disappeared, ST segment descended to isoelectric line, and the patient returned to sinus rhythm approximately 100 minutes after the detection of symptom. In the performed coronary angiography, normal coronary arteries were determined and dominancy of right coronary artery was observed. The patient was thought to have right coronary arterial vasospasm (Type 2 MI) which was probably triggered by electric shock.

Conclusion: our case, association of electric shock induced ST elevation at inferior derivations and AF was observed. The AF developed during monitorization can be due to either direct arrhythmic effect of the electric shock or ischemic myocardium. AF disappeared soon after normalization of ST segment. This makes us consider the ischemic etiology foreground. ince the primary cause is vasospasm, fibrinolytic therapy should not be considered as the first option for electric shock induced ST elevation MI treatment.

PO-127

Type 1 Kounis Syndrome following scorpion sting

Muhammet Buğra Karaaslan, Aziz İnan Çelik, Aslıgül Cüreoğlu, Müslüm Fırat İkikardeş, Mustafa Gök, Mustafa Tangalay, Vildan Yüksekdağ, Ali Deniz, Mustafa Demirtaş

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A 31year-old-man was admitted to emergency department with dyspnea, vomiting and presyncope. He had been bitten by a scorpion (androctonus crassicauda) (Figure 1) fourty minutes ago. His medical history was unremarkable. On admission his blood pressure was 200/100 mmHg, heart rate was 198 bpm. On physical examination; he was tachycardic, pupils were midriatic and loss of consciousness. On electrocardiogram (ECG) ventricular tachycardia (VT) (Figure 2A) was detected. 300 mg amidarone, 100 mcg/min nitroglycerine was given intravenously and scorpion antivenom was given subcutaneously. After given pharmacological therapy, ventricular tachycardia are turned sinus rhythm (Figure 2B). The analyses of blood samples revealed that elevated cardiac biomarkers (CKMB and Troponin I), basophilia and eosinopenia. There was no wall motion abnormality on Transthoracic Echocardiography. With these findings Type 1 Kounis Syndrome was thougt in this patient. Kounis syndrome due to scorpion sting was a rare clinical condition.

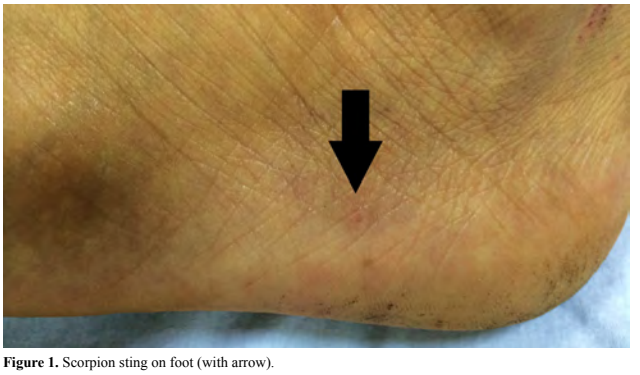

Figure 1. Scorpion sting on foot (with arrow).

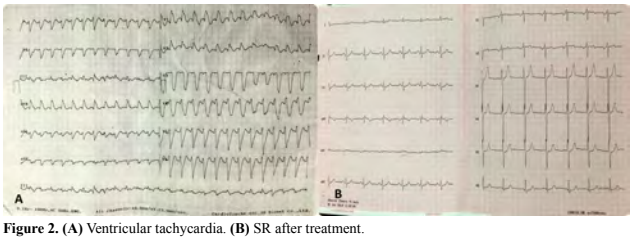

Figure 2. (A) Ventricular tachycardia. **(B)** SR after treatment.

PO-128

An unusual cause for the sudden death: short QT syndrome

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[2]*Pamukkale University Faculty of Medicine, Denizli*

A 20-year-old man presented to our cardiology clinic due to several episodes of transient loss of conciousness during the last 6 months. The patient was afebrile with blood pressure of 110/70 mmHg, heart rate of 60 bpm. Cardiac examination showed a regular heart rhythm with no murmurs. Electrocardiography (ECG) showed a sinus rhythm, 55 bpm; QTc: 313 ms (Bazzet formula) (Figure 1). Biochemical tests were within normal limits. The father reported one episode that took place approximately 1 year ago in which the patient was taken to the nearest hospital, a 3-5 minute drive. Cardiopulmonary resuscitation (CPR) had been performed with total CPR time of 30 minutes. He had been monitored and treated in the intensive care unit with undetermined etiology and discharged in 25 days without any neurological deficit. At that time the patient underwent a coronary angiography, which was found to be normal. He had no other known disease or history of sudden death in the family. He denied smoking, the use of illicit drugs or medications. Twenty four-hour Holter monitoring was performed in our clinic and showed infrequent premature ventricular contractions with no repetitive patterns. In the absence of other precipitating factors with ECGs depicting a shortened QT interval without structural heart disease, a likely diagnosis of short QT syndrome (SQTS) is made. Based on the diagnostic criteria proposed by Gollob, the patient is categorized as high-probability SQTS with a score of 6. Malignant arrhythmias due to SQTS have been considered as a primary cause of the syncopal attacks and sudden cardiac death. An implantable cardioverter-defibrillator was recommended but the patient has refused. SQTS is a recently defined entity included in the group of inherited primary electric disorders. It has been shown to be associated with atrial fibrillation and sudden cardiac death. In a young person who has no alternative diagnosis for the life-threatening events, SQTS should be kept in mind.

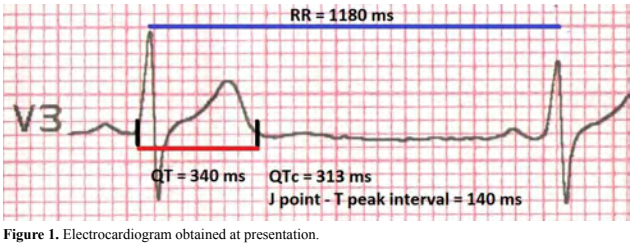

Figure 1. Electrocardiogram obtained at presentation.

PO-129

Dual-chamber pacemaker implantation through persistent left superior vena cava

Özcan Özdemir, Muhammed Ziaeitorbati

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A 65-year-old female patient was referred to our clinics for pacemaker implantation due to syncope and second-degree atrioventricular block. During pacemaker implantation, puncture of the left subclavian vein revealed PLSVC. Venography showed that this vein drains into the right atrium through a dilated coronary sinus (Figure 1). The typical passive ventricular lead was used and introduced into the right atrium, then a U-shape stylet was formed and through a wide loop within the right atrium. We were able to redirect the RV pacing lead into the RV by reflecting its tip off the RA free wall thus looping the lead around into the RA and back into the RV. Once across the tricuspid valve, the straight stylet can be advanced further if necessary to guide the pacing lead to the RV apex. An active atrial lead is screwed in the high lateral wall of the right atrium (Figure 2). The total procedure time was similar to that of the classical ICD implantation – about 30 min. Fluoroscopy time was also brief - 15 min. No complications were encountered during the procedure. Electrical parameters were controlled 24 hours and 7 days after implantation and were comparable to those during the operation. A chest X-ray also confirmed the stable location of leads. In conclusion, in the era of increasing procedures with device implantation operators should be aware of the possibility of PLSVC. After the left subclavian vein is punctured the guide wire takes an unusual left-sided downward course, crossing to the coronary sinus or directly to the right atrium. PLSVC in general does not prevent successful placement of a pacemaker or ICD lead and left-side device implantation. If PLSVC is recognized intra-operatively, we suggest continuing the transvenous left-sided approach and considering a right venous access in case of failure to reach a convenient site.

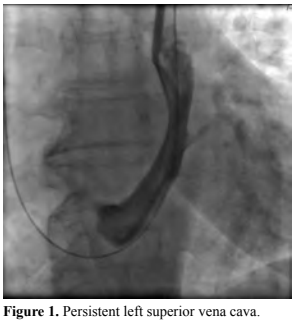

Figure 1. Persistent left superior vena cava.

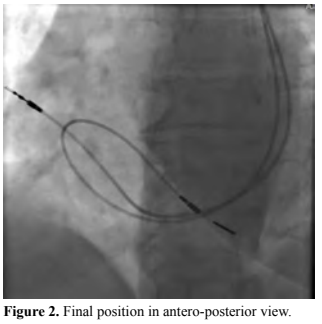

Figure 2. Final position in antero-posterior view.

PO-130

Which treatment should be chosen for the catheter-related thrombus in the right atrium; three case reports

Mustafa Candemir, Burcu Bilaloğlu, Atiye Çengel

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Introduction: One of the main complications of tunneled catheters is formation of thrombus at the catheter tip in the right atrium. This can be explained by motionless catheter tip causing damage to the right atrium wall. The optimal treatment of the catheter-related thrombus in the right atrium (CRTRA) is unclear. We report three cases with CRTRA in the patients who received thrombolytic therapy, and we review literature about CRTRA.

Case 1: A 22-year-old woman was on hemodialysis via internal jugular catheter. In the routine echocardiographic follow-up a thrombus measured as 7x11 mm was observed at the catheter tip located in the right atrium. The appearance of the thrombus was confirmed by the transesophageal echocardiography (TEE), and the catheter was removed. She was hospitalized because thrombus view persisted after catheter removal 50 cc alteplase (tPA) was given within six hours. After the thrombolytic therapy, dimensions of thrombus didn't change. Re- thrombolytic therapy wasn't considered due to chronic kidney disease. Warfarin therapy was started and she was discharged when INR level was effective. The thrombus was seen dissolved on the echocardiographic control after three weeks.

Case 2: A 42-year-old female patient was diagnosed with the breast cancer. She had a tunneled catheter for chemotherapy. On the routine echocardiographic images, a thrombus measured as 20x12 mm was observed in the catheter tip located in the right atrium. The thrombus was confirmed by TEE. She was hospitalized, and 50 cc alteplase (tPA) was given within 6 hours. After the thrombolytic therapy, no change in thrombus size was observed and the catheter was withdrawn. She was discharged on oral anticoagulation with warfarin. The control echocardiograms showed a progressive decrease in the size of the thrombus.

Case 3: A 21-year-old male patient, who had a diagnosis of Hodgkins lymphoma, had a tunneled catheter for chemotherapy. During his routine echocardiographic follow-up a thrombus was detected in the right atrium associated with catheter tip which was measured as 8x3 mm. After confirming the thrombus with the TEE, the catheter was removed.He was hospitalized, and 25 mg alteplaz (tPA) was given within six hours. This treatment was repeated four times. There was no change in the thrombus size after the thrombolytic therapy. He was discharged with warfarin therapy. Six weeks later, the thrombus view disappeared on the echocardiography.

Discussion: The mechanical irritation of catheter tip can cause end-damage and create a tendency to formation of thrombus. This situation is usually diagnosed incidentally in asymptomatic patients, and transthoracic echocardiography is usually sufficient for the diagnosis. After diagnosis, the treatment of the thrombus is unclear. In our cases, the alteplase therapy was'nt successful to dissolve the thrombus before or after catheter removal. On the other hand, thrombus was dissolved or reduced in size with oral anticoagulant.

PO-131

Bradicardia and first degree AV block associated with concominant use of metoprolol and sertraline

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Case Presentation: 55 year-old female patient was admitted to our hospital complaining of dizziness though five days. She had a 3-year history of metoprolol 50 mg daily treatment for hypertension. She was started sertraline treatment twenty days ago due to depression. On physical examination, her vital singns: Blood pressure was 140/85 mm Hg, pulse rate was 37 beat /min, chest sounds were clear, rhytmic and no murmur. Baseline electrocardiography showed sinus bradicardia of 39 beat/min and first-degree AV block with a PR interval of 0.24 seconds. Laboratory studies were unremarkable. Her initial Holter monitoring showed sinus bradicardia with first-degree AV block and sinus pauses (maximum pause 2.4 sn.). All medications were discontinued and her condition was carefully observed. The bradycardia resolved in the 48-hour and and Holter monitoring had normal findings the first 7 days. After eight days, firstly metoprolol 25 mg /day and then metoprolol 50 mg/day was given carefully. After sixteen days, she was discharged from hospital with stabile cardiac condition and heart rate. There were no any abnormal findings her clinical situation and Holter monitoring within the 60 days.

Discussion: Sertraline appears to have less cardiac effect. Although tachycardia is frequently seen, there are limited number studies in literature to show that sertraline increases risk of bradicardia. Our previous case report presented a patient who had sinus arrest after her medical treatment switching from the citalopram to the sertraline. It was discussed that sinus arrest might have been releated with the sertaline treatment started before the elimination of citalopram. Metoprolol is the most commonly used beta-receptor antagonist and it is eliminated mainly via the enzyme cytochrome P450 CYP2D6. This enzyme is inhibited to a varying degree by antidepressants. Some antidepressants are known potent CYP2D6 inhibitors like fluoxetine and paroxetine, and these agents have been shown to cause 4-to 6-fold increase biologically dose of metoprolol. Bradicardia and releated presencop or sencop may be an important adverse events, when these antidepressant agents add to metoprolol treatment. However, it is known that Sertraline effects CYP2D6 very little and so it is tought that it is safer antidepressant than the others in patients who are treated with metoprolol.The answer of why the some patients devoleped bradicardia may be releated to the polymorphism of the CYP2D6 gene, because CYP2D6 activity varies markedly between individuals. Our patient may be poor metabolizer and sertraline may inrease of metoprolol plasma concentration. Metoprolol plasma concentrations were found to be several fold higher in poor metabolizers than in extensive metabolizers in literature. In conclusion; sertraline should be considered possible cause of severe conduction defects and sinus bradicardia in the patients who are treated with metoprolol.

PO-132

Effectivity of hemodialysis in dabigatran overdose: report of a case with acute gastrointestinal bleeding

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Introduction: Dabigatran is the first novel oral anticoagulant agent approved to prevent thromboembolic events in nonvalvular atrial fibrillation (AF). The major disadvantage of dabigatran is the lack of a reversal agent in clinical use. Hemodialysis is currently the first line of treatment in patients with life-threatening bleeding associated with dabigatran. We presented a case with acute gastrointestinal (GI) bleeding associated with dabigatran overdose and treated by hemodialysis successfully.

Case Report: A 75-year-old woman with a medical history of hypertension, ischemic cerebrovascular disease and non-valvular AF ongoing 10 years presented with complaints of confusion, weakness, hematemesis and melena. Previously warfarin was initiated by a cardiologist because of nonvalvular AF but as a result of GI bleeding due to the warfarin overdose, the patient was prescribed dabigatran 150 mg twice daily. The recommended dose of dabigatran is 110 mg twice a day in patients with high GI bleeding risk according to the product Information. At that time, creatinine clearance calculated by Cockcroft-Gault formula was 24.1 ml/minute. Laboratory test results of patient shown in Table 1. The patient was admitted to the intensive care unit with a diagnosis of upper GI bleeding, acute renal failure, dabigatran overdose, hypernatremia and urinary tract infection. Patient was treated with proton pump inhibitor, intravenous fluid replacement, antibiotic treatment and fresh frozen plasma. The patient underwent hemodialysis twice within 24 hours. aPTT, PT and INR values decreased and active GI bleeding stopped after hemodialysis. Gastric endoscopy was normal. The patient was discharged on 110 mg dabigatran treatment twice daily.

Discussion: Hemodialysis may be preferred as a treatment option also in patients with hemodynamically stable significant GI bleeding associated to dabigatran.

Table 1. Laboratory test results of patient

	Admission	After first hemodialysis	After second hemodialysis	Before discharge
Urea (mg/dL)	293	80	74	32
Creatinine (mg/dL)	2.07	1.04	0.85	0.51
Hemoglobin (g/dL)	12.4	11.2	10.1	10.6
Hematocrit (%)	37.9	33.9	30.8	32.7
aPTT (sec)	167.0	41.3	29.2	34.7
PT (sec)	16.7	14.0	17.4	13.0
INR	1.46	1.23	1.53	1.14

PO-133

A misleading diagnosis in acute coronary syndromes: tirofiban induced alveolar hemorrhage

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In this report we present a patient with alveolar hemorrhage occured after Tirofiban administration after primary PCI. A 42 year old male patient was admitted to coronary care unit with acute anterior STEMI. After administration of 5000 units UFH, 300 mg ASA and 600 mg clopidogrel, the patient underwent primary PCI. A BMS was implanted to occluded proxiaml LAD. Tirofiban infusion was initiated during the procedure for coronary slow-flow which occured after PCI. 1,5 hours after administration of Tirofiban the patient became severely dyspnoeic with low PaO2 levels and haemoptysis. Dyspne continued with increasing severity despite 100% O2 support. TTE revealed a left ventricular ejection fraction of 40%, no valvular dysfunction and no pericardial effusion. Chest X-ray revealed diffuse alveolar shadowing (Figure 1). He was treated with iv. furosemid 80 mg presuming left ventricular failure, but he became progressively hypoxic and hypotensive. Soon after, the patient required endotracheal intubation and mechanical ventilation support. During intubation, large amount of bright red blood were seen in his airway. Hemoglobin level declined from 14.4 g/dL to 10.9 g/dL. We stopped Tirofiban infusion and administered iv. protamin but continued to clopidogrel and ASA. 24 hours after Tirofiban was stopped, bleeding from endotracheal tube has gradually terminated. He was weaned from mechanical ventilator at 3rd day. Diffuse alveolar shadowing was diminished at chest X-ray performed before extubation (Figure 2). Alveolar hemorrhage is an extremely rare complication of Tirafiban use. Increased alveolar pressure reflecting the increased LV end diastolic pressure (LVEDP) probably plays role in development of alveolar hemorrhage caused by Tirofiban. Acute respiratory distress, worsening alveolar infiltrates, and hemoptysis should alert clinician to the possibility of diffuse alveolar hemorrhage in patients receiving Tirofiban.

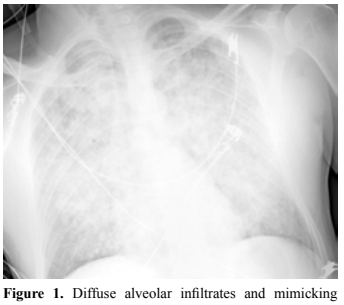

Figure 1. Diffuse alveolar infiltrates and mimicking P.edema.

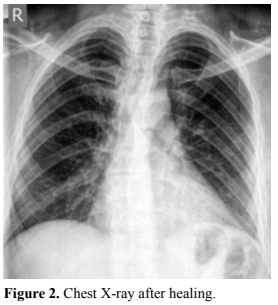

Figure 2. Chest X-ray after healing.

PO-134

A left atrial mass: not as easy as it looks!

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A 70 years old man who suffered a transient ischemic attack, was referred to the cardiology clinic for the evaluation of a cardioembolic source. The ECG showed sinüs rhythm. The echocardiographic examination on the other hand, revealed a 4x4.5 cm left atrial mass attached to the interatrial septum which was initially interpreted as a myxoma. The patient had history soft tissue sarcoma that had cured without reccurence for 10 years. A chest CT was carried out before surgery, that suprisingly revealed a larger mass posterior to the heart with extension into the left atrium through pulmonary veins. The thansbronchial biopsy from that mass was diagnosed as a chondrosarcoma. The patient was considered inoperable due to invasion of all of the pulmonary veins and chemotherapy was started. Intracavitary growth of metastatic heart tumors is unusual. Cardiac metastases are usually small and multiple affecting right side of the heart more commonly than the left side. Metastatic pathway is usually hematogenous or lymphatic but transvenous extension through vena cava to the right atrium is also reported. With our patient who presented with neurologic symptoms, on the other hand, a metastatic chondrosarcoma was shown to extend into left atrium through pulmonary veins imitating a left atrial myxoma. This is a very rare case with only a few reports published to date.

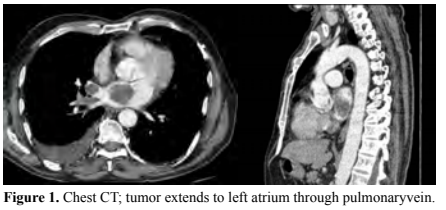

Figure 1. Chest CT; tumor extends to left atrium through pulmonaryvein.

PO-135

Persistent ST segment elevation due to myocardial metastasis in a patient with squamous cell lung carcinoma

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Adnan Menderes University, Faculty of Medicine, Aydın

ST segment elevation is often attributed to myocardial infarction however it may also occur due to some other rare pathologies. CASE A 63-year-old male patient admitted to our emergency department with palpitation, fatigue, burning type chest pain and syncope. He had been monitored for squamous cell lung cancer. Anterior ST elevation (Figure 1) was detected in the ECG. İmmediate coronary angiography revealed no significant and/or thrombotic lesion (Figure 1). The patient, on whom sustained ventricular tachycardia progressed on the first day of monitoring, was applied defibrillation, and sinus rhythm was provided. He received ıv amiodarone infusion. During follow up no other ventricular tachycardia was detected with the patient in later observations. On left ventricular septum anterior and apex was identified hypokinetic on the echocardiography while it was observed to be compatible with the mass in interventricular septum, mid and apical anterior and left and right ventricular apex (Figure 2). The patient went through cardiac MRI (Figure 2) and PET scan for lung carcinoma staging. PET scan showed metastatic lesions of the primary squamous cell lung carcinoma in the bilateral renal, bone, paracardiac and apex. Persistent ST segment elevation and VT developing after that were diagnosed to occur due to myocardial metastasis. ICD implantation was not performed since the life expectancy was less than one year. He was discharged with 50 mg metoprolol, 2.5 mg ramipril and 200 mg/day amiodarone once a day oral treatment. During the first month of his follow up no other arrhythmia episode occurred. DİSCUSSİON The tumors with cardiac involvement can be presented to emergency room with persitent ST segment elevation or malign arrhythmia. Clinicians should recall the phenomenon of persistent ST segment elevation due to the myocardial metastasis in atypical cases where clinical judgement is difficult.

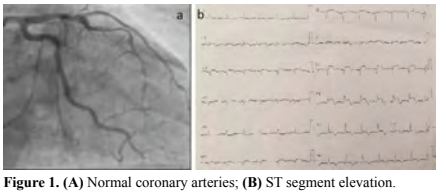

Figure 1. (A) Normal coronary arteries; **(B)** ST segment elevation.

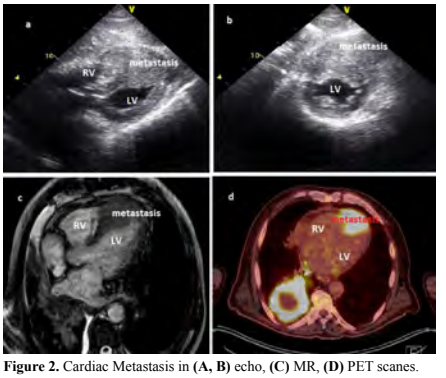

Figure 2. Cardiac Metastasis in **(A, B)** echo, **(C)** MR, **(D)** PET scanes.

Diğer

PO-136

Bitkisel ürün kullanımına bağlı gelişen digoksin intoksikasyonu

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Olgu: Bir yıldan beri hipertansiyon tedavisi alan 50 yaşında bayan hasta acile servise son 48 saattir olan olan bulantı, kusma ve fenalık hissi şikayetleri ile başvurdu. Hasta perindopril 1x5 mg ve indapamid 1x1,25 mg kullanmaktaydı. Fizik muayenede kan basıncı 130/80 mm Hg ve nabzı 40 atım/dk olarak saptandı. Çekilen EKG sinde sinüs aresti izlenen hasta yoğun bakıma yatırılarak takibe alındı. Hemodinamisi stabil olan hastanın yapılan biyokimyasal değerlendirmesinde digoksin düzeyinin 2,19 ng/ml olduğu saptandı. Hasta tekrar sorgulandığında; hastanın hemoraidal şikayetleri olması nedeniyle çevresinden almış olduğu bilgilere dayanarak halk arasında mayasıl otu olarak bilinen bitkiyi kaynatıp suyunu iki gün boyunca içtiği öğrenildi. Hastanın kullanmış olduğu bitki tarafımıza getirildiğinde digitalis lamarckii olduğu belirlendi (Şekil 1). Takiplerinde semptomları ve digoksin düzeyleri gerileyen hasta taburcu edildi.

Tartışma: Halk arasında mayasıl otu diye bilinen tek isim altında birden çok çeşit bitki vardır. Bunlar arasında digitalis türleri de mevcuttur. Semptomatik bradikardi, nodal ritim, AV blok ile başvuran hastalarda altta yatan bir patoloji saptanamadığında ilaç suistimali ve madde kullanımı sorgulanmalıdır. Günümüzde çok moda olan bitkisel tedavilerinde vakamızda olduğu gibi digoksin intoksikasyonu bulgularına neden olabileceği mutlaka akılda tutulmalıdır.

Şekil 1. Kullanılan bitki ve digitalis lamarckii sürgünü.

Kalp damar cerrahisi

PO-137

İntramural hematomu taklit eden bir lenfoplazmasitik aortit vakası

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İstanbul Medipol Üniversitesi, İstanbul

İlerleyen görüntüleme tekniklerine rağmen Akut ortik sendromlar (AAS) tanınması ve tedavisi hala zordur ve yaşamı tehdit etmektedirler. Yaygın görülen akut aort diseksiyonu (AD) dışında, aort duvarında meydana gelen kanama ile prezente olan intramural hematom (IMH) daha az sıklıkta görülmesine rağmen mortalite oranları AD ile benzerdir. Bununla birlikte klinik ve görüntü benzerlikleri sebebiyle IMH taklid eden aortitler yanlış tanı ve tedaviye neden olabilmektedir. Biz bu vakada; akut Tip A IMH vakasını taklit eden bir lenfoplazmasitik aortit vakasını sunduk. Kırk dokuz yaşında erkek hasta bölümümüze retrosternal yeni başlayan baskı tarzında göğüs ağrısı ile başvurdu. Elektrokardiyografide iskemik bulgu izlenmedi. Ekokardiyografide sol ventrikül sistolik fonksiyonları normaldi. Ancak daha önceki takiplerinde izlenmeyen ileri aort yetersizliği saptandı. Asendan aortada dilatasyon görüldü ve en geniş yerinde 4,7 cm ölçüldü. Parasternal uzun aks ve kısa aks görüntülerde aort kapak seviyesinin üstünde trombüs imajını düşündürten kile izlendi. Aort diseksiyonu ön tanısı ile CT yapılması planlandı. CT de aort kökünden arkus aortaya kadar uzanan ve aorttan çıkan büyük dalların orifisini içine alan, kalınlığı 12 mm e ulaşan sirküler IMH izlendi, flap veya giriş yeri izlenmedi. Göğüs ağrısı devam eden hastanın heart team tarafından operasyonu uygun görüldü. Asendan aorta ve arkus aortadan çıkan damarlar, bu bölgenin ileri derecede yapışık ve sert doku karakterinde olması sebebiyle, aort dalları seçilemedi. Aort dış yüzeyindeki sert doku koter ile kesilerek lümene ulaşılabildi ve aort duvar kalınlığının 11mm olduğu saptandı. Aort ön yüzü arkus aortaya kadar 30mm Gelwave greft ile aortoplasti yapıldı. Asendan aort biyopsi değerlendirmesinde medial, adventisiyal ve periadventisiyal lenfosit ve plazma hücrelerinden oluşan kronik inflamasyon saptandı.

Şekil 1. Asendan aortada spiral İMH taklit eden aortit.

Lipit / Koruyucu kardiyoloji

PO-138

Premature reflection of Type IIa hyperlipoproteinemia in the peripheral arteries

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Type IIa hyperlipoproteinemia is characterized by the increased levels of serum low density lipoprotein (LDL) cholesterol levels. Absence of chylomicrons in the plasma is prominent. Serum triglyceride levels are also normal. It leads to premature coronary artery disease as well as atherosclerosis of entire arterial network. The subjects are generally exposed to atherosclerotic burden in the early fourth decade. Herein we report a 20 years old male presenting with atherosclerotic disease of both carotid and iliac arteries. The patient was referred to cardiology polyclinic from primary care physician with swelling on the eyelids and extensor face of elbow which were likely to be xanthelesma. The patient underwent echocardiographic evaluation following a questionnaire about the history of the past years, physical examination of the cardiovascular system, sampling of blood and recording of a resting 12-lead electrocardiogram. All the findings were normal except serum lipid profile revealing a LDL cholesterol level of 447 mg/dl, high density lipoprotein (HDL) cholesterol level of 36 mg/dl and total cholesterol level of 499 mg/dl. Serum triglyceride level was normal as 81 mg/dl. The lipid profile was consistent with familial type IIa hyperlipoproteinemia. Doppler ultrasound evaluation revealed 50% and 40% stenosis in both carotid and iliac arteries. Lying behind the atherosclerotic process is a well known feature of familial hyperlipoproteinemia. However development of atherosclerotic lesions in the peripheral arteries at so early age is worthy of reporting in terms of emphasizing the crucial role of hyperlipidemia in atherosclerosis which is subject to speculations even in the scientific area.

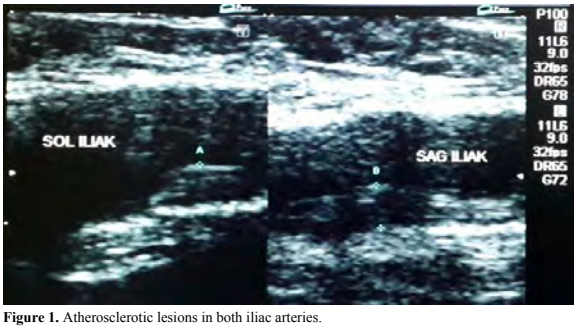

Figure 1. Atherosclerotic lesions in both iliac arteries.

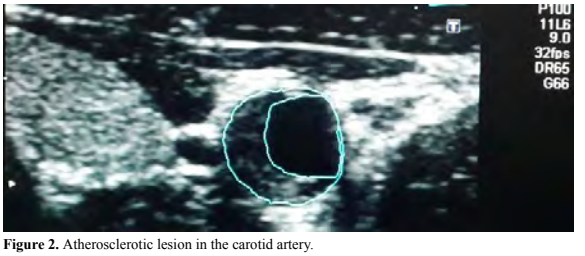

Figure 2. Atherosclerotic lesion in the carotid artery.

Koroner arter hastalığı / Akut koroner sendrom

PO-139

Acute necrotizing pancreatitis complicated with ST elevation myocardial infarction

Muhammet Naci Aydın[1], Güney Sarıoğlu[1], Sevcan Dinçer Aydın[2], Şıho Hidayet[1]

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Acute pancreatitis has been reported to be associated with electrocardiographic changes imitating acute myocardial infarction. Nevertheless, cases of acute pancreatitis complicated with true acute myocardial infarction are very rare. This case report describes that a 82-year-old man was admitted to the emergency department with acute left chest and left upper quadrant abdominal pain. Patient's medical history revealed that a myocardial infarction had occurred one year ago and was implanted drug eluting stent to right coronary artery (RCA). Electrocardiogram (ECG) showed that 1-mm ST-segment elevation in leads II, III, AVF, with reciprocal changes in leads I, AVL. Serum levels of troponin I was high (25 ng/ml, normal: 0-0,05 ng/ml).A diagnosis of acute inferior ST-segment elevation myocardial infarction was made.The anjiography showed a 100% instent restenozis in the RCA but no stenozis at the other coronary arteries.This time balloon angioplasty was performed after that drug eluting stent was implanted(Figüre 1). However, the abdominal pain persisted and extended to the epigastric region over the subsequent few hours. Additional laboratory investigations revealed an elevated while cell count (25,2 x10^3/ML), raised serum lipase level (2068 U/L), amylase level (1381 U/L), AST level (311 U/L), ALT level (60 U/L) and creatinin level (2,24 mg7dl).The diagnosis was hence adjusted to acute pancreatitis; and a referral to the gastroenterologist was made. Abdominal computed tomography (CT) revealed acute necrotizing pancreatitis(Figüre 2).The patient soon developed severe septicaemia with altered mental state,

and the condition was complicated by disseminated intravascular coagulopathy as well as acute renal failure.The patient was treated with intensive antibiotic therapy including imipenem 4x500 mg, intravenous fluids and nutrition.The patient died due to multiple organ dysfunction caused by acute pancreatitis a day later.

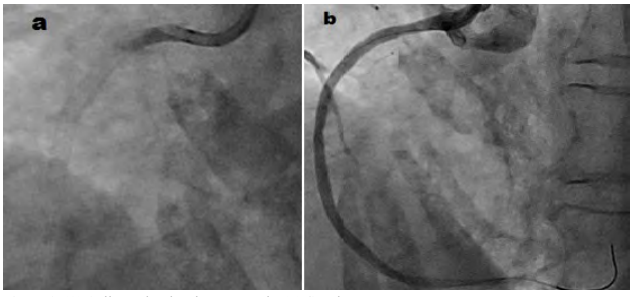

Figure 1. (A) Anjiography showing restenozis. **(B)** Stenting.

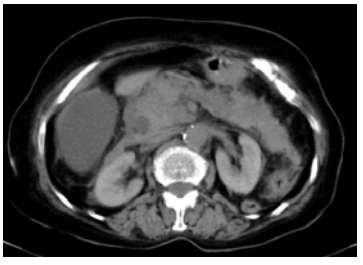

Figure 2. AbdominaL CT showing that necrotizing pancreatitis.

PO-140

Acute myocardial infarction and ischemic stroke togetherness due to marijuana abuse in an adolescent

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Marijuana is a rare trigger of acute myocardial infarction (AMI). A 15 years old man was admitted to our emergency department with complaining of chest pain. He has smoked marijuana cigar for 2,5 years. Electrocardiogram (ECG) showed inferolateral ST segment elevation. Transthoracic echocardiogram (TTE) showed: left ventricular ejection fraction (LVEF) was 40%, inferior hypokinesis, severe mitral regurgitation (MR) and mild depression in right ventrciular (RV) functions (tricuspid annular systolic velocity (TASV): 11 cm/s, tricuspid annular plane systolic excursion (TAPSE):1.84 cm. Coronary angiography revealed normal coronary arteries. Cardiac enzymes were also elevated (creatinine kinase isoenzyme MB: 32 IU/L, troponin I: 6.4 ng/mL). Considering of coronary vasospasm we administered diltiazem treatment. Following 2 hours of observation the patient complained of right sided blurred vision. Cranial computed tomography (CT) scan was normal. Twelve hours later, second CT showed an acute occipital lobe infarction and mild cerebral shift. Because of probability of occlusive cerebrovascular disease; the patient was given warfarine treatment. After seven days of medical treatment the patient's hamiparesis spontaneously resolved. Blurred vision has been alleviated but did not completely resolve. The patient did not complain of chest pain afterward. The patient was discharged and called for a control after a month. His visual impairment was resolved. ECG showed sinus rhythm, inverted T waves in lead DI,II,aVL,V4-6. TTE showed LVEF was 55%, mild MR and mild depression in RV functions TASV: 10.8 cm/s TAPSE:2.1 cm. Wall motion abnormality was not seen. Cardiac magnetic resonance imaging (CMRI) was performed for confirmation and etiology of stroke. CMRI showed LVEF was 55%, late myocardial enhancement in the posterolateral mid-basal portion of the heart and minimal right ventricular hypokinesis (EF:30.5%). CMRI did not show and intra-cardiac mass or thrombus.

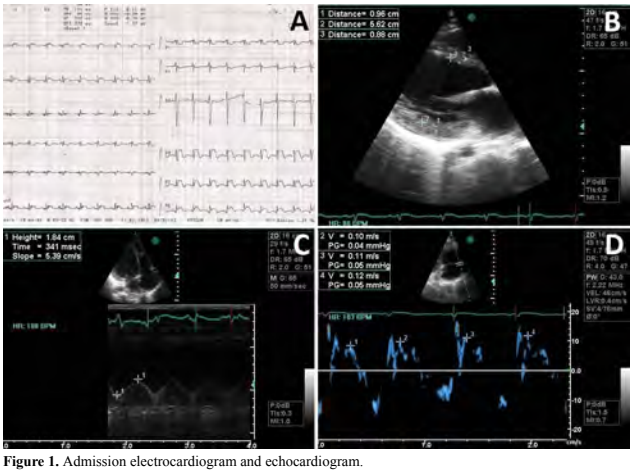

Figure 1. Admission electrocardiogram and echocardiogram.

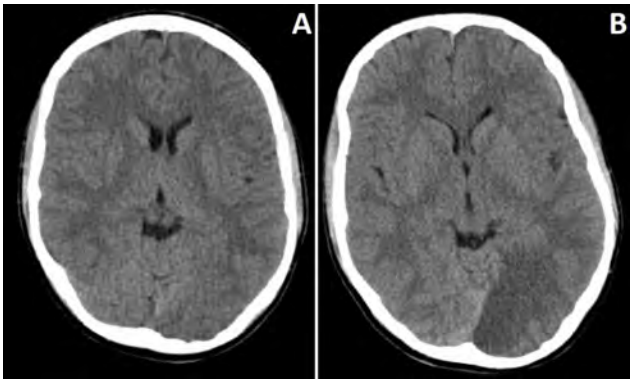

Figure 2. CT shows left occipitale infarction zone.

PO-141

The healing of spontaneous coronary artery dissection with conservative treatment: when to stop

Cengiz Öztürk, Turgay Çelik, Sait Demirkol, Mustafa Demir, Şevket Balta, Murat Ünlü, Atila İyisoy

Gülhane Military Medical Academy (GMMA) Ankara, Ankara

A 58 year old man who presented with acute chest pain for 4 h was admitted to our coronary care unit. He had no previous trauma or emotional stress. There was mild–moderate elevation of the troponin T level. There was a slight change on electrocardiogram (T inversions on precordial derivations). The patient was diagnosed to have a non-ST-elevation myocardial infarction (NSTEMI). Dual anti-platelet therapy was initiated. Invasive coronary angiography 2 days after presentation showed a 2 cm long irregular stenosis with extending to the distal, which angiographically was significant in the left anterior descending artery (LAD) but distal flow was enough. In addition, the distal vessel lumen diameter was less than 2 mm. There were also no atherosclerotic lesions in the remainder of the LAD system, left circumflex and the right coronary arteries. The dissected segment of the vessel was not changed after intracoronary nitroglycerin injection. We can do testing of the lesion with fractional flow reserve and intravascular ultrasound (IVUS) examinations but we did not do it because of the small diameter and the patient was hemodynamically stable, no chest pain and the flow was good. We decided to treat the patient conservatively with heparin and nitroglycerin for 24 h. During the three day period, the patient was asymptomatic and we performed coronary angiography again. The dissection of the LAD was healed completely (Figs. 1). The electrocardiography and echocardiography were also normal. The patient had no atherosclerotic coronary artery disease lesions with only smoking as a risk factor. Spontaneous healing was done with only conservative approach in our case. In our case, we did not need to attempt wiring the lesion in order to prevent the propagation of dissection and blocking theflow to the distal part of the LAD. It should be kept in mind that the conservative treatment may be an alternative choice in stable lesions. It is important to decide when to stop.

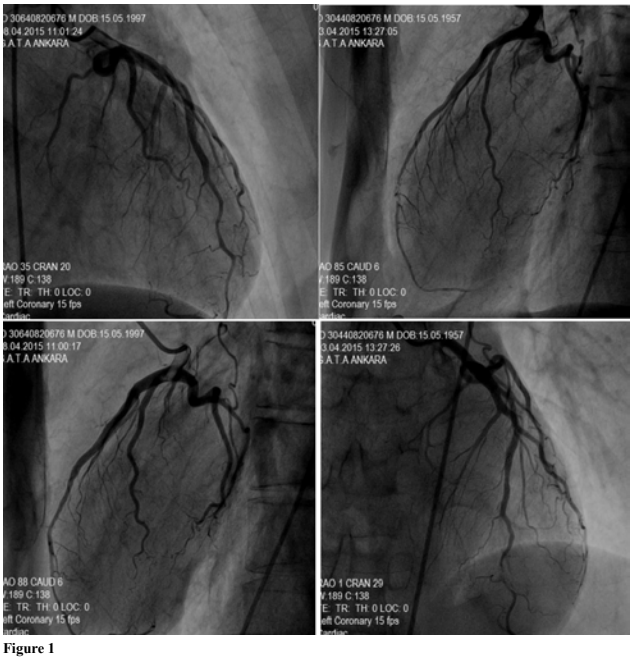

Figure 1

PO-142

A case of coronary vasospasm due to 5 Fluorouracil treatment

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A 50 year old woman was referred to our intensive care unit with a diagnosis of myocardial infarction. The patient was diagnosed colonic adenocarcinoma and started 5- FU, oxaliplatin, follic acid and altuzan (Folfox- 6- Altuzan) protocol. During 28th day of the treatment the patient developed typical angina pectoris at rest. The therapy was discontinued and the patient transferred to our intensive care unit because the electrocardiogram (ECG) showed ischemic changes. The first ECG showed ST elevation and T wave peaking in DI-III, aVL, aVF and V2-6 derivations as well as reciprocal changes in aVR and VI. In detailed history; two weeks ago, a coronary angiography was performed to her in another center because of the same clinical presentation and revealed normal findings. The pain resolved after treatment with nitroglycerin infusion (5-100 mg/min) and diltiazem. The ECG findings improved rapidly. The transthoracic echocardiography and consequitive control of CK- MB (14-16 U/ L) and Troponine T (0.002- 0.24 ng/ml) measurements revealed normal findings. Also other possible causes of chest pain like pericarditis, hyperventilation and alkolosis were ruled out. After these evaluations the case was diagnosed as a coronary vasospasm due to 5- FU treatment. She was discharged on the other day with a decision of considering a modification in the current chemotherapy protocol. At the follow up, the patient was free of chest pain with the modified chemotherapy regimen. This case was presented to underline the cardiotoxic side effects of 5- FU and the importance of getting a detailed patient history before primary percutaneuos angioplasty which is sometimes a race against time in clinical practice. In conclusion, 5- FU is the key chemotherapeutic in colonic adenocarcinomas and its cardiovascular side effects are so serious that can not be ignored. The incidence of these side effects increase with higher doses and continues infusions. In the case of a cardiovascular event, the chemotherapeutic should be modified and 5- FU should not be used again. It must be kept in mind; 5- FU administration can make coronary vasospasm mimicking acute myocardial infarction and this situation can be treated successfully by nitrates and calcium channel blockers.

PO-143

Recurrent acute posterior myocardial infarction in a patient due to cisplatin-based combinatin chemotherapy

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Fifty-six year old male was presented to our emergency department with persistent retrosternal chest pain succesting acute coronary syndrome on the second day of the second chemotherapy course with conventional doses of cisplatin and gemcitabine. Electrocardiography showed 1 mm ST-segment depression in leads V1-3 and 1 mm of ST elevation in leads V7-8. The patient underwent urgent cardiac catheterization with a diagnosis of acute posterior myocardial infarction (MI) and required percutaneous coronary intervention (PCI) for acutely occluded obtuse marginal branch of circumflex artery (Cx) (Figure 1A). During the procedure, cardiac markers became available: troponin I 15 ng/mL (reference range 0 ng/mL to 0.06 ng/mL) and creatine kinase MB 177 U/L (reference range 7 to 25 U/L). The level of troponin I later rose to maximum >50.000 ng/mL before beginning to decline. The patient underwent PCI and a single bare metal stent was placed in the obtuse marginal branch of circumflex artery (Cx). We learned that he was diagnosed invasive papillary urothelial bladder cancer four months ago; on the first day of the first chemotherapy course with conventional doses of cisplatin and gemcitabine he presented to our hospital with acute posterior MI, then he had PCI application to Cx (Figure 1B). Due to the development of both event after chemotherapy; recurrent acute coronary syndrome associated with cisplatin was suspected. Switching of cisplatin to another chemotherapeutic was recommended to the medical oncologist.

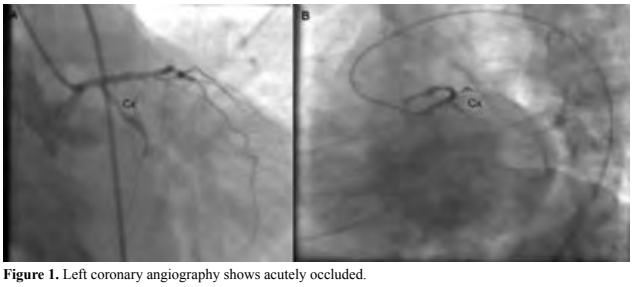

Figure 1. Left coronary angiography shows acutely occluded.

PO-144

Acute inferior ST segment elevation myocardial infarction and percutaneous coronary intervention in a young man receiving chemotherapy for testicular

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Introduction: Testicular cancer is a highly treatable malignancy in men. Systematic randomised trials have shown that Cisplatin, Etoposide and Bleomycin combination chemotherapy remains the mainstay of treatment. Cisplatin and Bleomycin is thought to be the most likely agents causing myocardial infarction (MI). MI seen just after Bleomycin first course chemotherapy is very rare.

Koroner arter hastalığı / Akut koroner sendrom

We present a case of treatment-related acute myocardial infarction in a young man just after first course of chemotherapy for testicular Mixed Germ Cell Tumor.

Case: A 36 year old man was diagnosed the Mixed Germ Cell Tumor as a result of right orchiectomy biopsy, one month ago. He received a combination of Cisplatin (35 mg) and Etoposide (175 mg) daily for five days. He had received Bleomycin (30 mg) on the second day. On the tenth day, he received second Bleomycin (30 mg) therapy and after one hour, he was consulted to cardiology department with sudden onset of chest pain. This was the first chest pain and he had no any other known disease as well any major cardiovascular risk factors. His blood pressure was 140/80 mmHg and the heart rate was 90 beats/min. The physical examination was normal. Electrocardiography demonstrated sinus rhythm and ST elevation in D2-D3 and aVF derivations, ST depression in D1-aVL derivations (Figure 1). The bedside echocardiographic examination showed hypokinesia in inferior segments of the left ventricle. He was immediately taken to the catheter laboratory for coronary angiography, with the resulting diagnosis of acute inferior myocardial infarction. Left coronary angiography demonstrated normal left main coronary artery, normal Left Anterior Descending artery (LAD) and normal Circumflex coronary artery (CX). On the right coronary angiography there was intracoronary thrombotic lesion in the mid segment of the Right Coronary Artery (RCA) (Figure 1a). A bare metal stent was implanted to RCA mid lesion (Figure 1b). The patient was discharged healthy.

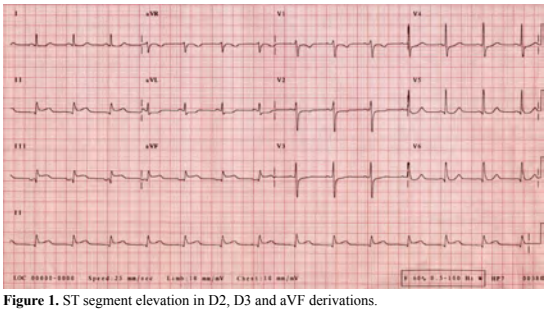

Figure 1. ST segment elevation in D2, D3 and aVF derivations.

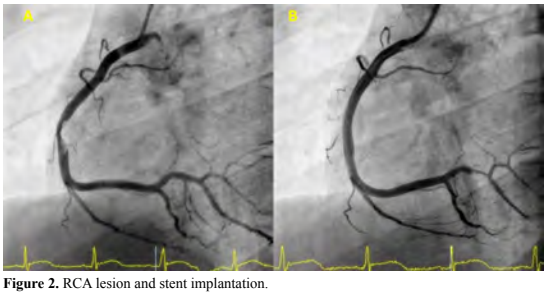

Figure 2. RCA lesion and stent implantation.

PO-145

A simple left anterior decending artery (LAD) lesion causing left ventriculer free wall rupture and total oclusion of femoral artery

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A 50 years old male patient without any known medical history and cardiovascular risk factor presented to the emergency department with a substernal chest pain that started 3 days ago. On admission the patient presented with a blood pressure of 70/50 mmhg and heart rate 100bpm. Electrocardiography showed subacute anterior myocardial infarction and normal sinus rhythm. In physical examination; juguler venous distension, his upper right leg was cold and pale and also he was complaining a serious right upper part of the leg pain and paresthesia. The pulse of femoral and popliteal arteries were not palpable. Taking into consideration these findings, we get suspicious about right femoral artery tromboemboli and consulted a cardiovascular surgeon. At the same time echocardiography administered in order to find the origin of the embolisation. İn parasternal long and short ax view; anterior, anteroseptum were severe hypokinetic and ejection fraction was %30. In apical 4 chamber view, there was a subacute partial free wall ruptur (the defect was about 0,7 cm from the apical wall) in which a moderate hemorrhagic pericardial efüzyon that covered with a 1 cm trombosed mass (coagulum) adjacent to left ventricle can be seen as well as aneurism in the apical wall (video 1). A moderate to large pericardial effusion measuring 1,9 cm in its greatest dimension at the apex. There was an augmented material flow in expiration and decreases with inspiration (pick E velocity in expiration: 0,8 m/s and in inspiration 0,4 m/s). Mitral inflow respiratory variation was about %50. İnside of the left ventricle there was severe Sec that explains the origin of the peripheral embolisation. (Video 2) Despite the inotropic agents blood pressure was not increasing greater than 70/60 mmhg. İABP administered from left femoral artery and he was undertaken to thorax and lower extremite BT angiography. Right internal iliac, femoral, popliteal, tibial, peroneal arteries were total occluded. Before the surgery, coroner angiograpy performed from his right radial artery to see the responsible lesion. Left anterior decending (LAD) midsegment was total occluded. Infarct related segment was supplying small amount of miyocardium. Surprisingly this simple lesion had caused both peripheral embolism and miyocardial rüptür. Stiff wire failed to penetrate proksimal lesion altough using extra back up cathater. The patient immedietly underwent an operation for right femoral embolectomy and repair of the rupture with a PTFE (polytetrafluorethylene)-patch. After following postoperative intensive care unit 47 days he discharged without any sequelae. Left ventricule free wall rupture is reported about %0,5-%2 of all myocardial infarctions (usally after 3-5 days later) and frequently with large anterior wall infarctions, woman and older (>70 years) age. But our patient was young, male and a small anterior wall infarction. About 10% patients are dying because of the rupture of the free ventricule in STEMI. Electrocardiographic signs suggestive of Hemopericardium include increased T wave amplitude and the reversion to normal of inverted T waves. So early diagnosis and emergency operation is very important for these patients. After anterior wall myocardial infarction the incidence of early trombus has been reported at rates 10-56%. LV trombus with systemic embolisation is a serios complication that resuting extremite loss if there is a delay in diagnosis. However, early operative intervention, has a high mortality rate, with a 30-day mortality rate of 15-25%.

PO-146

A case of double chambered right ventricle diagnosed by cardiac magnetic resonance imaging and catheterization

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A 20 year-old female patient presented to our clinics with the complaint of the increasing dyspnea on minimal exertion. Cardiac auscultation revealed a grade 3/6 systolic murmur over the left sternal border. TTE and TEE revealed evidence of a double chambered right ventricle (RV) across a prominent moderator band. There was no evidence of obstruction noted across the pulmonary outflow tract. The left ventricular systolic function was within normal limits with no valvular abnormalities. Cardiac magnetic resonance imaging revealed a hypertrophied muscle bundle dividing the RV into two chambers It was detected high pressure proximal and low pressure distal right heart chamber communicated each other with a narrow duct in catheterization. Maximal systolic pressure gradient was measured as 160 mm Hg between two chambers of right ventricle. There was no left-to right shunt. Left ventriculography taken from left anterior oblique position did not show any ventricular septal defect. Right ventriculography taken from right anterior oblique position showed that there was a double chambered right ventricle separated from each other with abnormal muscle bundle and dilated pulmonary artery (Figure). Double chambered right ventricle (DCRV) is a rare congenital heart defect in which the right ventricle is separated into a high pressure proximal and low pressure distal chamber. This defect is considered to be congenital and typically presents in infancy or childhood but has been reported to present rarely in adults. DCRV is typically found concomitantly with congenital cardiac disorders, most notably ventricular septal defect and subaortic stenosis. Due to its rarity and the difficulty of visualization, DCRV continues to be misdiagnosed. In conclusion, multimodality cardiac imaging using echocardiography,cardiac CT, cardiac MRI and cardiac catheterization is often required for complete characterization of complex congenital heart anomalies in adults like our case.

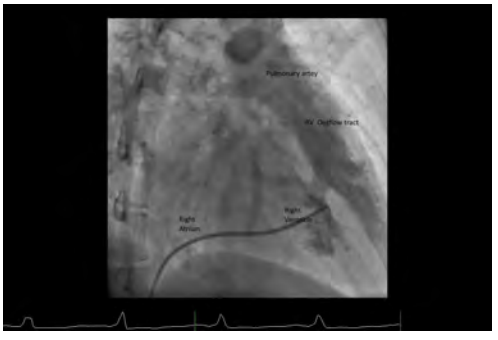

Figure 1. DCRV in RAO view; the appearance of 2 chambered RV in angiography.

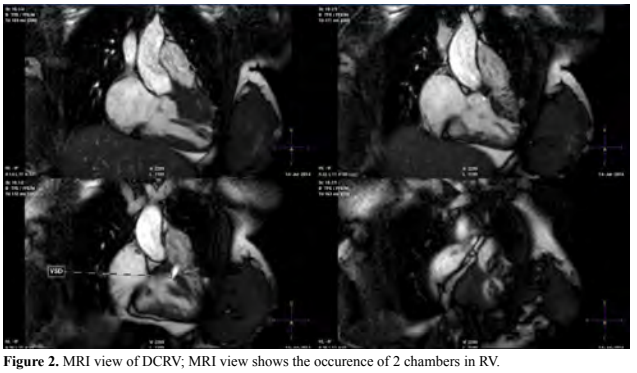

Figure 2. MRI view of DCRV; MRI view shows the occurence of 2 chambers in RV.

PO-147

Large atrial thrombus associated with subclavian vein catheter in a hemodialysis patient

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Catheter-related right atrial thrombus(CRAT) is rare but potentially life threatening complication of central venous catheters. This complication is usually underreported and the optimal treatment is controversial. Incidence of catheter related thrombosis is variable but when right atrial thrombus is present, it carries a mortality risk of %18 in hemodialysis patients and greater than %40 risk in non-hemodialysis patients. Surgical resection, anticoagulation and thrombolysis are the principle treatment strategies. We want to report a case of 64 year old diabetic patient, who is on hemodialysis for 4 months through right subclavian vein. She developed a giant right atrial thrombus adherent to the catheter tip. She was treated with heparin infusion and oral warfarin for three weeks, afterwards surgical resection was performed due to insufficient decrease in the size of thrombus. Right atrial thrombus associated with subclavian vein catheter is a very rare which makes our case interesting.

Kardiyak görüntüleme / Ekokardiyografi

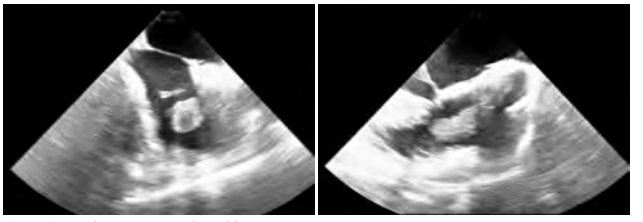

Figure 1. TEE image; transesophageal images.

PO-148

Koroner bypass operasyonu sonrası perikardiyal konstriksiyon fizyolojisine neden olan organize perikardiyal hematom

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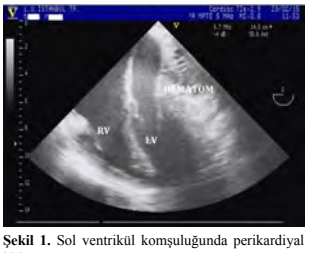
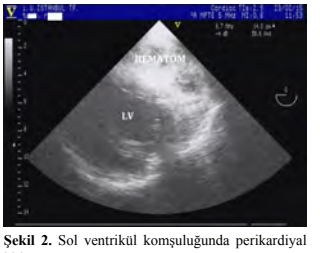
Yaklaşık bir yıl önce geçirilmiş koroner arter bypass operasyonu öyküsü olan ve kronik iskemik kalp hastalığı nedeniyle takip edilen 57 yaş erkek hasta, 1 yıldır eforla olan nefes darlığı şikayetinin giderek artması, 1 aydır her iki bacağında ve karında şişlik şikayetleri ile başvurdu. Hastanın yapılan fizik muaynesinde TA:100/60 mmhg, NDS:116/ritmik, sağ sinüs kapalı, sağ akciğer orta zonlara kadar, sol akciğer alt zonda ince krepitan raller duyulmakta idi. 3(+) pretibial ödemi mevcut olan hastanın batın bombe ve umblikus 3 parmak yukarıda ve assiti mevcut idi. Hastanın EKG si 100%dk, atriyal flutter ile uyumlu idi. Yapılan transtorasik ve transözefageal ekokardiyografilerinde sol ventrikül posterior duvar komşuluğunda perikard boşluğunu dolduran 3.2 cm kalınlığında organize thrombus izlenimi veren, yer yer düşük ekoik alanlar içeren, posterior duvarın diastolünü engelleyen kitle imajı izlendi (Şekil 1, 2). Ayrıca perikard konstriksiyonu lehine bulgular ve her iki atriyumda genişleme saptandı. EF %52 olarak ölçüldü. Toraks BT'de sağ akciğerde enfeksiyon, malignite ayrımı yapılamayan konsolidasyon saptandı. Hastaya PET-CT çekildi. PET-CT'de lenf nodları, plevral effüzyon ve parankimde FDG tutulumu saptanmadı ayrıca perikardiyal kitlenin organize hematom veya yabancı cisim ile uyumlu olabileceği düşünüldü. (Şekil 3,4,5). Hastanın kalp yetersizliği ve pnömoni tedavisi sonrası hemodinami ve koroner anjiyografisi yapılmak üzere kateter laboratuvarına alındı. Koroner anjiyografisinde bypass greftleri açık saptandı. Hemodinami çalışmasında konstriktif fizyoloji saptandı. Hasta kalp ve damar cerrahisi ile perikardiyektomi ve perikard boşluğundaki kitlenin rezeksiyonu yapılmak üzere operasyona verildi. İntraoperatif yapılan transözefageal ekokardiyografide kitlenin görünümü daha net izlenmiştir (Video 1). Hastanın operasyon sonrası kontrol ekosunda kitlenin izlenmediği görüldü (Şekil 6,7). Kitlenin patolojik incelemesi organize hematom ile uyumlu olduğu izlendi.



Şekil 1. Sol ventrikül komşuluğunda perikardiyal kitle.



Şekil 2. Sol ventrikül komşuluğunda perikardiyal kitle.

PO-149

Sık geçici iskemik atak hastasında nadir bir etiyoloji; Non kompaction kardiyomiyopati

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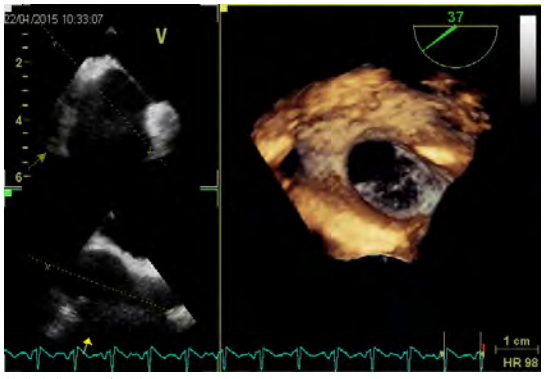
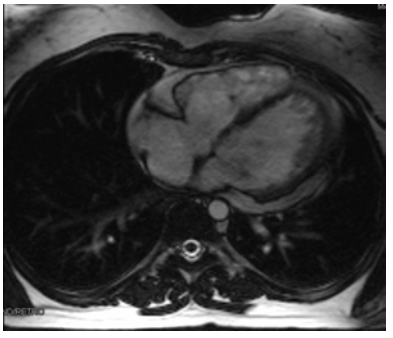
Non kompaction kardiyomiyopati intrauterine dönemde endomiyokardiyumun morfogenezinde duraklama sonucu olusan belirgin trabeküler yapı ve intratrabeküler girintiler ile karakterize, genelde sol ventrikülü tutan doğumsal kardiyomiyopatidir. Sıklıkla asemptomatik olmakla birlikte; kalp yetmezligi, ventrikuler aritmiler ve sistemik embolilerle karsimiza gelebilir. Tanısı ekokardiyografi ile genelde konulur.

Olgu: 28 yaş kadın olgu, uzun yıllardır kısa süreli çarpıntı ve senkop atakları mevcut. Bir yıl önce kısa süren, sol tarafında hissizlik ve güçsüzlük yakınması sonrasında çekilen Kranial MRG da; sağ frontoparietal bölgede embolik odaklar saptanmış. Takip kranial MRG'da, aynı bölgede iskemik enfarkt saptanmış. Etiyoloji açısından yapılan ve 3D TTE ve TEE de; biventriküler EF normal, sağ ventrikül (SağV) apikal gölgede trabekülasyon artışı, SağV genişlemiş,interatriyal septumda 1,5 cm çapında defekt saptandı (Şekil 1). Kardiyak MRG ve toraks Anjiyo BT çekildi;sekundum tip ASD lehine bulgular ve her iki ventrikül apikal kesimlerinde daha belirgin olarak izlenen trabeküler görünüm (Şekil 2) saptandı. Yirmi dört saatlik Holter EKG'sı de aritmi izlenmedi. Alt ekstremite doppler görüntülemede DVT lehine bulgu saptanmadı. Olguya mikroembolilere bağlı tekrarlayan geçici iskemik atak düşünülerek antikoagulan tedavi başlandı. Olgu noncompaction kardiyomiyopati ve eşlik eden sekundum tip ASD olarak değerlendirildi. Olgu antikoagulasyon ile takibe alındı.

Sonuç: Non-kompaction KMP hastaları progresif kalp yetmezliği kliniği, ölümcül olan ve olmayan artimiler ve sistemik emboli kliniği ile başvuran sıklıkla geç tanı alan olgulardır. Genç hastalarda sık geçici iskemik atakların etiyolojisi araştırılması önerilmektedir. Sıklıkla etiyoloji açıklanmamasına rağmen paradoksal emboliler, konjenital kalp hastalıkları ve kalp yetmezliği açısından değerlendirilmesi gerekir. Bu hastalarda nadir görülmesine rağmen non kompaction KMP akla getirilmesi ve tanı konulması durumunda aile taraması yapılması önerilir.



Şekil 1. 3D TEE de; ASD.



Şekil 2. Kardiyak MRG görüntüsü; kardiyak MRG da non-kompaction kardiyomiyopati.

PO-150

Akut arter embolisinin ilk septom olduğu sol atriyal miksoma

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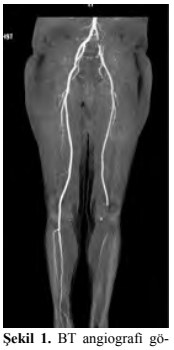
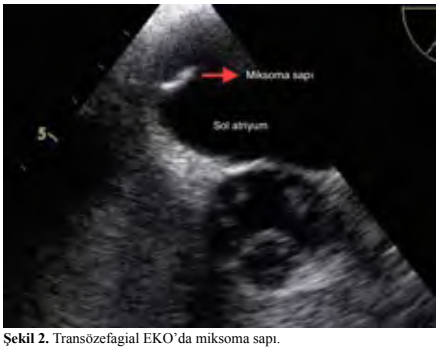
Kardiyak miksomalar kalbin en sık rastlanan iyi huylu tümörlerdir. Asemptomatik ve rastlantısal olarak görülebildiği gibi, nefes darlığı, senkop ve çarpıntıya yol açarak mitral darlığını taklit edebilirler. 62 yaşında erkek hasta sol bacakta ağrı şikayeti ile acil servise başvurdu.Fizik muayenesinde sol bacakta ısı kaybı ve solukluk mevcuttu, sol popliteal ve distal nabızlar alınamıyordu. Laboratuvar tetkikinde lökositoz saptandı.Bilateral alt ekstremite BT angiografide (Şekil 1): Sol ana iliak arter proksimalinde lümen kalibrasyonunda ileri derecede darlığa neden olan, emboli ile uyumlu yumuşak plak izlendi. Ayrıca sol popliteal arter total oklüde görünümdeydi. Akut arter embolisi tanısı ile acil operasyona alınarak embolektomi yapıldı. Emboli kaynağı araştırılırken yapılan transözefagial ekoda sol atriyumda interatriyal septumda köken alan embolize olmuş miksomaya ait olduğu düşünülen kitle sapı tespit edildi (Şekil 2). Histopatolojik incelemede emboli metaryalinin miksoma ile uyumlu olması üzerine operasyonla sol atriumda bulunan miksoma sapı çıkartıldı. Kardiyak miksomalar ortalama 50 yaş civarında görülür. Miksomaların %75'i sol atriyumda görülmektedir ve tipik yerleşim yeri interatriyal septumda fossa ovalis bölgesidir. Kadınlarda erkeklerden daha sıktır. Sol atriyal miksomalar, inme ve akut ekstiremite iskemilerinin nadir bir nedenidir.Bu durum miksomanın gerek kendisinin gerekse miksoma yüzeyindeki trombüs parçalarının distale giderek emboli komplikasyonlarına yol açmasından kaynaklanabilir. Miksomaların %50'sinden fazlasında tanı ancak sistemik embolizasyon olduktan sonra veya tümörün kalp kapaklarını kapatacak büyüklüklere erişmesi ile hemodinamik bozukluk oluşturmaya başlamasından sonra konulmaktadır. Bizim olgumuzda olduğu gibi, embolizasyona zemin hazırlayacak kardiyak aritmi, ateroskleroz gibi hastalıkları bulunmayan hastalarda periferik embolilerin nedeni olarak nadir de olsa kardiyak miksoma ile karşılaşılabileceği akılda tutulmalıdır.



Şekil 1. BT angiografi görüntüsü.



Şekil 2. Transözefagial EKO'da miksoma sapı.

Kardiyak görüntüleme / Ekokardiyografi

PO-151

İskemik mitral yetersizliği nedeniyle ring anuloplasti uygulanan bir hastada parsiyel ring ayrılması ve 3D transözafagiyal ekokardiyografi ile değerlendirilmesi

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Giriş: İskemik mitral yetersizliği, koroner arter hastalığı bulunan hastalarda mortalitenin önemli bir göstergesidir. Ciddi koroner arter hastalığı nedeniyle cerrahi revaskülarizasyon uygulanacak hastalarda orta ya da ciddi MY izlendiğinde sıklıkla tedaviye mitral ring anuloplasti eklenmektedir. Bununla birlikte mitral ring anuloplasti uygulanan hastalarda, uygun halka çapının seçilmesi son derece önemlidir. Düşük çaplı ring uygulamalarında cerrahi sonrası erken dönemlerde parsiyel ring ayrılmaları ve buna bağlı orta-ciddi MY gözlenmektedir. Bildirimizde, mitral yetersizliği nedeniyle ring anuloplasti uygulanan bir hastada parsiyel ring ayrılması ve 3D ekokardiyografi ile değerlendirilmesi sunulmuştur.

Olgu Sunumu: Nefes darlığı şikayeti ile dış merkezde değerlendirilen 68 yaşındaki hasta, yapılan tetkikleri sonucunda ileri derecede mitral yetersizliği saptanması üzerine kliniğimize yönlendirildi. Hastanın klinik hikayesinden 17 yıl önce 3 damar koroner arter bypass cerrahisi operasyonu yapıldığı ve yine 5 yıl önce tekrar 4 damar bypass cerrahisi yapıldığı öğrenildi. Aynı zamanda ikinci cerrahi sırasında saptanan orta derecede mitral kapak yetersizliği için ring anuloplasti (29 mm DURAN ring) yapıldığı öğrenildi. Kliniğimizde yapılan transtorasik ekokardiyografide ejeksiyon fraksiyonu %25-30 saptandı (Video 1,2), aynı zamanda ileri derecede MY izlendi. Transözafagiyal ekokardiyografide A2-P2 skalloplarından kaynaklanan ciddi MY izlendi (Video 3,4,5). Bununla beraber daha önce implante edilmiş olan anuler ringin anterior yaprakçıktan ayrıldığı ve kapağın santral bölgesine yer değiştirdiği gözlendi. Yapılan 3D incelemede mitral kapağa ait ringin yer değiştirmesine bağlı olarak çift orifis görünümü izlendi (Video 6,7). Hastanın daha önce iki kez açık kalp cerrahisi öyküsü olması ve EuroScore %19 olması nedeniyle kalp damar cerrahisi bölümünce tekrar operasyon planlanmadı. Medikal tedavisi düzenlenen hasta taburcu edildi.

Sonuç: Mitral anuler ring ayrılması olan hastalarda uygulanan tedavi biçimi genellikle açık kalp cerrahisidir ancak bu tedavi biçimi birçok hasta için yüksek risk taşımaktadır. Bu nedenle doğru tanı yöntemi ve mitral kapak yapısının ayrıntılı biçimde değerlendirilmesi son derece önemlidir. Özellikle mitral ring anuloplasti uygulanan hastalarda ileri MY saptanması halinde 3D ekokardiyografik inceleme kapak yapısı hakkında daha ayrıntılı bilgi sağlayabilir.

PO-152

İntrakardiyak kitle: Metastaz mı, hiperkoagulasyon zemininde trombüs mü?

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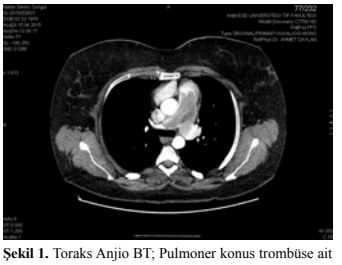
37 yaşında bilinen endometriyal sarkoma tanılı kadın hasta 2 yıl önce bu nedenle opere olmuş, kemoterapi ve anti-östrojen tedavi almış. Son 2 yılda efor dispnesi tarifleyen hasta şikayetlerinin artması ve pozisyonla değişen baş dönmesi, çarpıntı şikayetleri de olması nedenli dış merkez radyasyon onkolojisine başvurmuş. İzlemde kardiyolojiye konsulte edilmiş ve yapılan toraks anjio bt'de pulmoner emboli saptanması üzerine, heparin infuzyonu sonrası kumadinize edilmiş. Ayrıca yapılan TTE tetkikinde sağ ventrikülde ve her iki atriyumda PFO ilişkili trombus ile uyumlu görünüm izlenmesi üzerine hasta kliniğimize sevk edilmiş. Kliniğimizde tekrarlanan toraks anjio BT'de her iki atriyumda ve sağ ventrikül içerisinde trombüs ile uyumlu dolum defektleri, pulmoner konus sağ lateralde trombüse ait dolum defekti (Şekil 1) izlendi. Yapılan TÖE tetkikinde ise sağ ventrikül içinde farklı kesitlerden 2.5x3.3 cm, 4.5x2.5 cm çapında trombüs ile uyumlu hiperekojen görünüm ve İAS'de PFO ile uyumlu görünüm, sağ atriyumda PFO ile ilişkili 1.6*1.1 cm ve sol atriyumda PFO ile ilişkili 1.5x1.1 cm çapında trombüs ile uyumlu görünüm izlendi (Şekil 2), (Video 1). EKG'si sinüs ritminde olan hastanın yapılan karotis-vertebral arter, alt ve üst ekstremite arteriyel ve venöz sistem renkli doppler USG normal olarak raporlandı. Tromboza yatkınlık açısından yapılan mutasyon taramada ise protrombin G20210A'da, faktör13 V34L'de, GP3A L33P'de, MTHFR C677T'de heterozigot mutasyonlar saptandı. Pulmoner emboli, pulmoner hipertansiyon ve PFO ile ilişkili trombüs nedeniyle hasta kliniğimiz PAH konseyinde tartışıldı. İzlemde hastanın kumadinize olarak izlenirken romatolojik tetkiklerinin tamamlanması ve kontrol ekokardiyografi ile tekrar değerlendirilmesi kararı alındı. Hastanın tetkikleri devam ederken takip edildiği KDC servis izleminde kardiyopulmoner arrest gelişmesi üzerine KPR yapılarak yoğun bakıma alındı, ancak hemodinamik yanıt alınamaması üzerine hasta exitus kabul edildi.



Şekil 1. Toraks Anjio BT; Pulmoner konus trombüse ait dolum defekti.

Şekil 2. TÖE tetkiki; PFO ilişkili biatrial ve sağ ventrikül içi trombüs.

PO-153

Case report: a rare case myxoma localized to right ventricle presenting with syncope

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Cardiac myxoma is the most frequent benign tumor of the heart which is usually located in left atrium. Right ventricular myxoma is extremely rare. Hereby, we present a case of cardiac myxoma located in right ventricle of the heart. 54 years old male patient was presented to emergency department with syncope. At time of admission, systolic blood pressure was 80/40 mmHg, heart reate was 120 bpm and he was tachypneic. He was immediately consulted to cardiologist with preliminary diagnosis of pulmonary emboli. Meanwhile his troponin level was reported as three times high of the normal. There was no abnormality in other blood tests. Contrasted thorax CT revealed a right ventricular mass completely filling right ventricle cavity. Transthoracic echocardiography confirmed the right ventricular mobile mass and protruding into pulmonary artery. (Figure 1) Preoperative coronary angiography was performed. Diffuse vasculature was also observed angiographically. (Figure 1) Patient was referred to cardiovascular surgery and the mass was completely excised. Pathological examination of the excised surgical material was reported as benign cardiac myxoma. (Figure 2) 75% of primary cardiac tumors are benign, and 25% are malignant. 50% of the benign tumors are comprised by myxomas. Myxomas are intracavitary tumors. Left and right ventricular myxomas together have occurrence rates of 6-8%. Right ventricular myxoma is quite rare. Surgical excision should be performed soon after the diagnosis due to risk of valvular obstruction and pulmonary embolization. Patients should be followed up with annual echocardiography examinations for surveillance of recurrence.

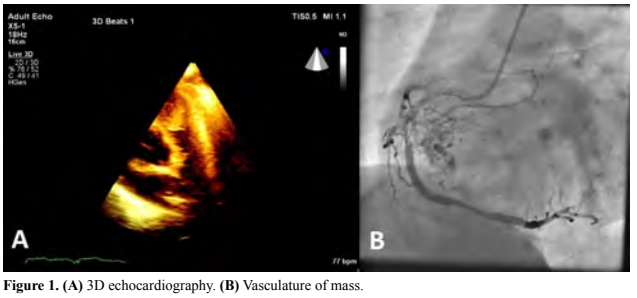

Figure 1. (A) 3D echocardiography. **(B)** Vasculature of mass.

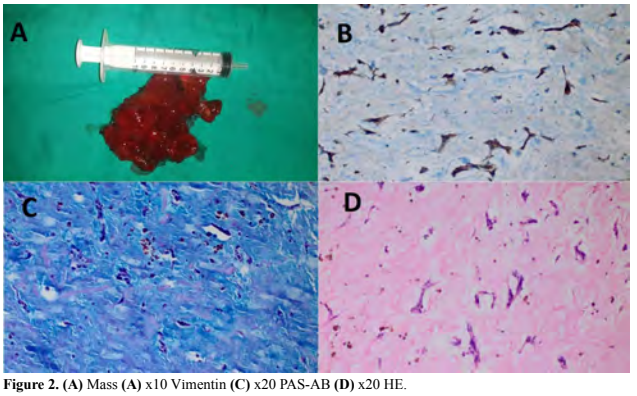

Figure 2. (A) Mass **(A)** x10 Vimentin **(C)** x20 PAS-AB **(D)** x20 HE.

PO-154

Treatment of left ventricular thrombus with apixaban

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[2]*İstanbul University Cerrahpaşa Faculty of Medicine, İstanbul*

A 68 year old man was admitted to our emergency department due to shortness of breath started about seven days. An electrocardiogram demonstrated left bundle branch block and the patient's laboratory revealed elevated myocardial necrosis marker levels. Transthoracic echocardiographic (TTE) examination showed a thrombus (1.3x0.6) in the apex of the left ventricle (Figure 1). The left ventricular ejection fraction was 25%. The coronary angiography revealed total occlusion of the ostial left anterior descending coronary artery. Medical treatment decision was taken. In addition to dual antiplatelet therapy, apixaban was administered as anticoagulant therapy for the left ventricular thrombus. The size of thrombus slowly decreased, and TTE performed 8 weeks after the initial apixaban administration demonstrated no thrombus without a thromboembolic event (Figure 2). This case shows that left ventricular thrombus can be resolved with apixaban treatment. Apixaban may be an effective alternative to vitamin K antagonist for some patients with left ventricular thrombus.

Kardiyak görüntüleme / Ekokardiyografi

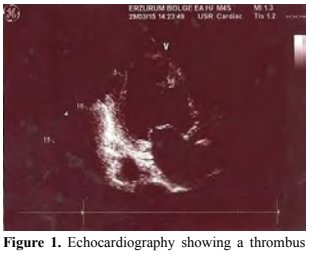

Figure 1. Echocardiography showing a thrombus in ventricle.

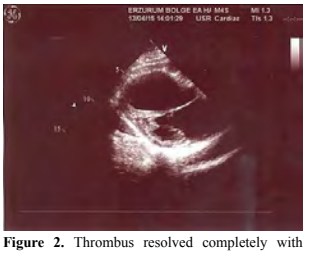

Figure 2. Thrombus resolved completely with apixaban.

Girişimsel kardiyoloji / Koroner

PO-155

Anomalous origin of right coronary artery from the mid-left anterior descending artery leading to coronary steal phenomenon and ischemia

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Bozok University, Faculty of Medicine, Yozgat

Case: A 50-year-old woman was admitted to our cardiology outpatient clinic with complaint of exertional dyspnea (NYHA class II) and atypical chest pain. Patient was not suitable for the treadmill stress test due to congenital hip dislocation. Therefore, we scheduled myocardial perfusion scintigraphy. A single photon emission computed tomographic (SPECT) study was performed with 20 mCi of technetium-99m methoxyisobutylisonitrile (Tc-99m MIBI) after a pharmacologic stress test with dipyridamole and again at the rest. On the images obtained after the stress, hypoperfusion was detected in the mid and basal segments of the anterior region of the heart. Perfusion of these segments returned to normal on the images taken at rest (Figure 1). These changes were interpreted as ischemia. As further investigation, coronary CT angiography was scheduled for the patient. Coronary CT angiography demonstrated a single coronary artery from the left valsalva sinus. An anomalous RCA was originating from the mid-LAD, after the first diagonal branch, coursing to right, anterior the pulmonary artery and then trajects downward into the right atrioventricular groove (Figure 2). The coronary arteries were free of atherosclerotic disease.

Conclusion: Single coronary artery, anomalous origin of the RCA from LAD, is a rare clinical entity. Although coronary anomalies are considered to be benign entities, they may cause myocardial ischemia and even sudden cardiac death. Coronary CT angiography may be beneficial in the diagnosis CAD and coronary anomalies. Coronary anomaly and accompanying ischemia is not a commonly encountered situation. Ischemia may be detected in a minority of these patients. In our patient, anomalous RCA may increase the load on LAD. Therefore, especially during prolonged physical activity, LAD could not satisfy the increased demand of blood flow for the myocardium. For this reason, ischemia on LAD region may be occurred.

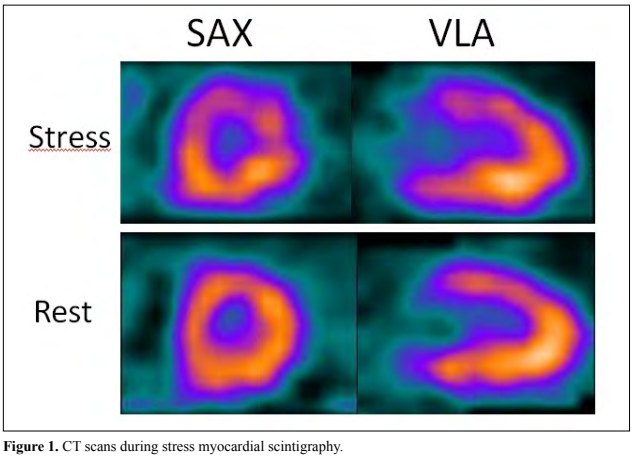

Figure 1. CT scans during stress myocardial scintigraphy.

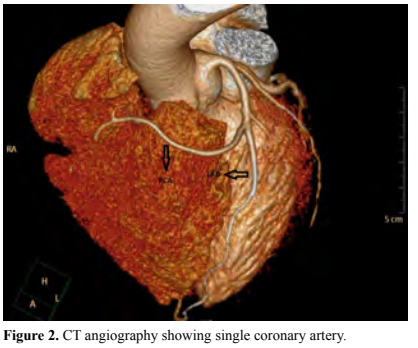

Figure 2. CT angiography showing single coronary artery.

PO-156

A case of coronary microfistulas; a newborn microfistula

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Introduction: Despite the fact that coronary artery fistulas are known to be rare congenital malformations, occasionally they appear as acquired solitary fistulas.

Case: A 56 year old male patient was admitted to chest pain. He was smoking and had hypertension. He was on metoprolol and ramipril.Coronary angiography was performed one year ago due to chest pain which was found to be normal (Figure 1). Grade III diastolic dysfunction with normal systolic function was seen on former echocardiography. The coronary angiography showed a microfistulas communication between the left coronary artery and the left ventricle cavity with no evidence of atherosclerotic disease (Figure 2).

Discussion: Coronary fistulas are rarely seen and incidence of coronary fistulas in general population is 0.002% but coronary angiographic series reveal an incidence of 0.3–0.8%(1,2). The mechanism is probably related to the diastolic pressure gradient caused by blood run-off from the coronary vasculature to a low-pressure receiving cavity. Coronary microfistulas can appear depend on pressure changing in fistula region or plague rupture on coronary microcanals. Many drugs of decreasing on left ventricular enddiastolic pressure (LVEDP) such as beta blocker, calcium channel blocker may trigger coronary microfistulas. We thougt coronary microfistulas were appeared due to decreased of LVEDP or micro plague ruptured in coronary microcanals.

Conclusions: Although coronary microfistulas are thought to be congenital, the way they are formed remains unknown. Its relationship with atherosclerosis has not been established. The possibility that plaque ruptures which may develop in coronary microcanals may in time lead to the formation of microfistulas should be remembered. Besides decreased LVEDP might be appeared existing coronary microfistulas. However, answers to these questions require more studies to be carried out.

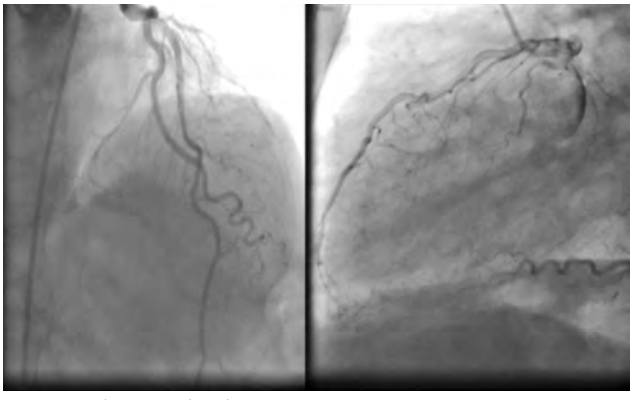

Figure 1. Normal coronary angiography.

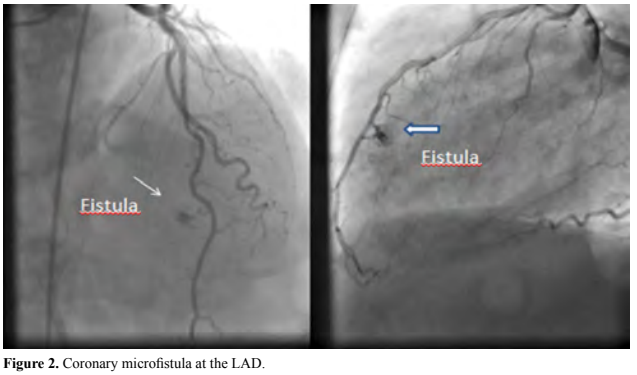

Figure 2. Coronary microfistula at the LAD.

PO-157

Cx osteal kritik lezyon için sol ana koroner artere yapılan Culotte Tekniği ile Tryton stent yerleştirilmesi ve bir yıllık sonucu

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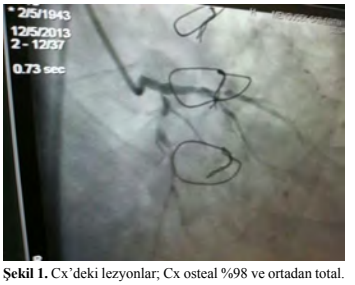
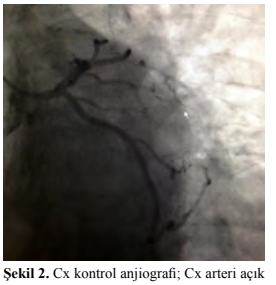
6 yıl önce 3 damar bypass operasyonu olan hasta CCS class 3 angina ile yatırılarak koroner anjiografi yapıldı. LAD osteal %40 ve ortadan total LIMA-LAD açık, Cx osteal de %98 kritik lezyon ve cx ortadan totaldi ancak antegrad dolusu vardi, Ao-Cx safen total izlendi, RCA total Ao-RCA safen açık izlendi. Cx nativ damara Sol ana koroneri (LMCA) de içerecek şekilde culotte teknigi ile bifurkasyon stent yapılmasına karar verildi. LMCA dan LAD ye uzanacak şekilde 3.5x3.0 Tryton marka stent yerleştirildi sonra Cx osteal lezyon twinpass micro kateter destegi ile fielder xt wire ile gecildi wire degisimi yapildiktan sonra ardisik PTCA yapildi sonra Cx ortadan itibaren stent yerleştirilmeye başlandı son stent LMCA dan tryton stentin başından Cx proksimali kapsıyacak şekilde yerleştirildi. LAD rewire yapıldı LMCA dan LAD ve CX e kissing ptca yapıldı son olarak LMCA ya 4.5x12 nc balon ile final POT yapıldı sonuç optimaldi. Bir yıl sonra yapılan koroner anjiografide LMCA nin ve CX in açık olduğu restenoz olmadığı görüldü. Sonuç olarak Tryton stentin ana koronerdeki stent yükünü azaltabileceginden seçilen vakalarda kullanabileceği düşünüldü.



Şekil 1. Cx'deki lezyonlar; Cx osteal %98 ve ortadan total.



Şekil 2. Cx kontrol anjiografi; Cx arteri açık ve restenoz görülmüyor.

PO-158

Ayrı ostiumlardan köken alan sağ çıkışlı Cx arter ve RCA arterin eş zamanlı tıkanmaları ile oluşan inferior miyokard infarktüsü olgusu

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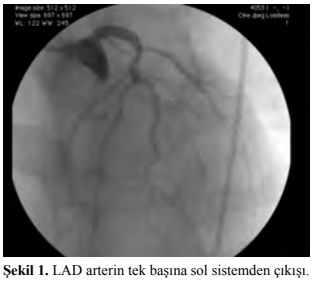
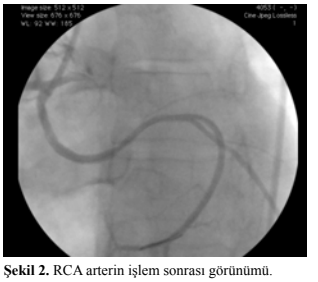
Daha önce bilinen kronik bir hastalığı olmayan 48 yaşında erkek hasta, 3 saatlik tipik göğüs ağrısı ile başvurdu. Çekilen EKG sinde inferior MI olduğu izlenen hasta primer perkütan koroner girişim yapılmak üzere kateter laboratuvarına alındı. Hastanın sol sistem enjeksiyonunda sadece LAD arterin izlenmekte olduğu görüldü (Şekil 1). LAD arterde herhangi bir ciddi darlık saptanmaması üzerine sağ koroner sistemi görüntülemesine geçildi. Sağ sistem enjeksiyonunda ise RCA ve Cx arterlerin ayrı ostiumlarda orijin aldığı ve RCA arterin total, Cx arterin de mid bölgeden subtotal olduğu görüldü (Video 1). Aynı seansta RCA ve Cx arterlere müdahale kararı alındı. RCA artere balon dilatasyon sonrası 2.75x15, Cx artere de 2.75x18 ilaç kaplı stent implantasyonları yapıldı (Video 2, Şekil 2). İşlem sonrası komplikasyon gelişmeyen hasta medical tedavisi düzenlenerek yoğun bakıma alındı. Üçüncü günde herhangi bir sorunu olmayan hasta ayaktan takip edilmek üzere taburcu edildi.



Şekil 1. LAD arterin tek başına sol sistemden çıkışı.



Şekil 2. RCA arterin işlem sonrası görünümü.

PO-159

A 52 Year-old women with diplopia after a myocardial infarction

Zeynep Yıldız[1], Şahin Avşar[1], Ahmet Öz[1], Adnan Kaya[2], Mehmet Bozbay[1], Ahmet Murat[1]

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[2]*Suruç Goverıtment Hospital, Şanlıurfa*

Background: The medial longitudinal fasciculus are cranial structures which help to move eyeballs together and their injury cause disturbed conjugate eye movements. Mesencephalon's vascular and demyelinating diseases can lead to this uncommon condition.

Case: A 52 year-old women was admitted to our emergency department for retrosternal pain two hours duration. Her medical history revealed that she had no risk factors for coronary artery disease except smoking for 15 years. Her electrocardiogram revealed ST segment elevation in leads D2, D3 and AVF with reciprocal changes in leads D1, aVL. She was taken to catheter laboratory and coronary angiography revealed occlusion of the proximal right coronary artery (RCA) with heavy thrombus burden. Circumflex and left anterior descending coronary arteries were normal.After balloon dilatation a 3.0x20 drug-eluting stent was implanted in proximal RCA. The day after she complained diplopia when she was looking her right side. Detailed neurological examination revealed horizontal gaze palsy on looking towards right side and impaired adduction in the left eye with nystagmus on right eye abduction. She had normal vertical eye movements and convergence.Her other neurological examination was found completely normal. Computed tomographic scans and magnetic resonance imaging did not show any abnormality. Patient was treated with enoxaparin, clopidogrel and aspirin. Four days later the patient recovered completely and was discharged a week after admission.

Conclusion: MLF syndrome or partial oculomotor nerve palsy (medial rectus palsy) was considered as the result of microembollus of medial longitudinal fasciculus (MLF) which take place in midbrain. This uncommon syndrome occurs if a lesion affects the horizontal gaze center. Like this case report this condition may not be displayed with magnetic resonance imaging in the early stages and if it is associated with an ischemic event it may be completely recover with medical treatment.

PO-160

Perkütan koroner girişim sonrası akut ST elevasyonlu miyokard infarktüsü şeklinde komplike olan dirençli vasospasm olgusunun başarılı yönetimi

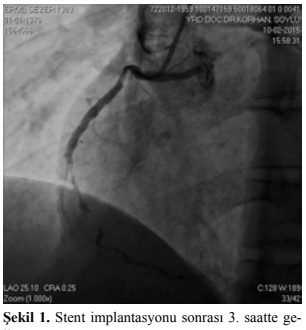
Metin Çoksevim, Kenan Çakmak, Ömer Gedikli, Korhan Soylu

Samsun Ondokuz Mayıs Üniversitesi, Samsun

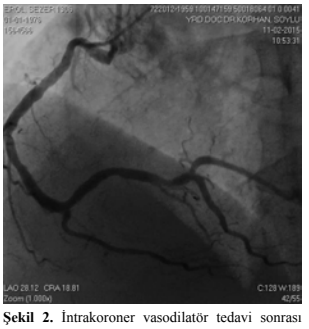
Vakamızda başarılı koroner girişim sonrası STEMI ile klinik prezentasyon gösteren şiddetli ve dirençli bir koroner vasospazm olgusu sunulmuştur.

Olgu: 40 yaşında erkek hasta son efor anginası ile başvurdu. Efor testinde angina ve lateral derivasyonlarda 3 mm ST depresyonu gelişti. Koroner anjiografide; RCA ortada %90 darlık, CX proksimalde %50 darlık ve LAD orta segmentte muskuler bridge saptandı. İntrakoroner 200 mcg perlinganit yapıldıktan sonra RCA için tekrar görüntü alındı. Aynı seansta RCA orta %90 darlık oluşturan lezyona 2,5x15 mm Brio marka balon ile predilatasyon yapıldıktan sonra 3x28 mm sirolimus salınımlı stent (Cre8) implante edildi.İşlem sonrası 3,5x20 mm balon ile post dilatasyon yapıldı ve optimal açıklık sağlandı. Koroner yoğun bakıma alınan hastada işlemden 3 saat sonra şiddetli parasternal ağrı ve soğuk terleme başladı. Elektrokardiyogramda D2, D3 ve aVF de 3 mm, ST elevasyonu, V1- 3derivasyonlarında 2 mm ST depresyonu saptandı. Akut stent trombozu düşünülerek acilen katater laboratuarına alındı. Anjiografide; stent açık ancak stent dışında yaygın spasm vardı. RCA'nın Acute Marginal dalın hem sonrasında tamamen tıkalı olduğu görüldü (Şekil 1). İntrakoroner perlinganit yapıldıktan sonra distal RCA akımının cılız şekilde oluşmaya başladığı görüldü. Lezyon bölgesine vasodilatör ajanların yeterince ulaşamadığı ihtimali düşünülerek mikrokateter aracılığıyla lokal olarak vasodilatör ajanlar uygulandı. Yaklaşık 80 dk boyunca RCA içerisine 4500 mcg intrakoroner perlinganit, 50 mg diltiazem ve 100 mcg adenozin yapıldı. Hasta semptomları gerilemiş olarak yoğun bakıma alındı. Kontrol anjiografide koroner spasm tamamen düzelmişti (Şekil 2). Bir ay sonra kontrole gelen hasta asemptomatik idi.

Tartışma: Bu olguda dirençli vasospasm olgularında uygulanan yüksek doz vasodilatör ajanlara rağmen hızlı klinik yanıt alınamayabileceği, böyle durumlarda özellikle bir mikro kateter yardımı ile lezyon içerisine vasodilatör ajanların ulaştırılmasının yararlı olabileceği gösterilmiştir.



Şekil 1. Stent implantasyonu sonrası 3. saatte gelişen spasm.



Şekil 2. İntrakoroner vasodilatör tedavi sonrası kontrol.

PO-161

Right coronary artery and aortic root dissection during percutaneous coronary intervention

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Introduction: Coronary dissection and its progression into aorta is an extremely rare and potentially life-threatening complication of percutaneous coronary intervention (PCI)(1). In this case report, we present a patient with right coronary artery dissection with retrograde progression to aorta due to the PCI.

Case Report: A 69-year-old male complaining of chest pain was admitted to our clinics. Because of unstable angina, coronary angiography was performed. The angiography showed critical luminal stenosis at the proximal and distal part of the right coronary artery (Figure 1). PCI was planned fort the right coronary artery (RCA) with the AL 2 guiding catheter. Guiding catheter was deep intubated until the proximal lesion. After the stenting of distal lesions, the proximal RCA was damaged by the guiding catheter causing a coronary dissection (Figure 1A). The following contrast injections caused the dissection to extend retrogradely to the aorta (Figure 2B). The distal flow of the RCA was not impaired. Finally, dissected segment of the RCA was stented from the ostium with a good angiographic result and the dissection of the sinus of Valsalva disappered, exept aortic wall opasification (Figure 3A,B). The patient was discharged after 7 days.

Discussion: The incidence of catheter-induced dissection and retrograde progression to the aorta estimated at 0,008% in diagnostic coronary angiography. However, the mortality rate is approximately 50%. Quickly diagnosis and treatment are very important to decrease this mortality. In our case, the initial dissection was type D according to The National Heart, Lung, and Blood Institute classification. Types C–F coronary dissections have high morbidity and mortality. Operators must be careful in type D and E dissections due to rapid progression. This case should call to the attention of the interventional cardiologist these issues: early diagnosis of coronary dissection, and the possibility of anterograde and retrograde progression of the dissection.

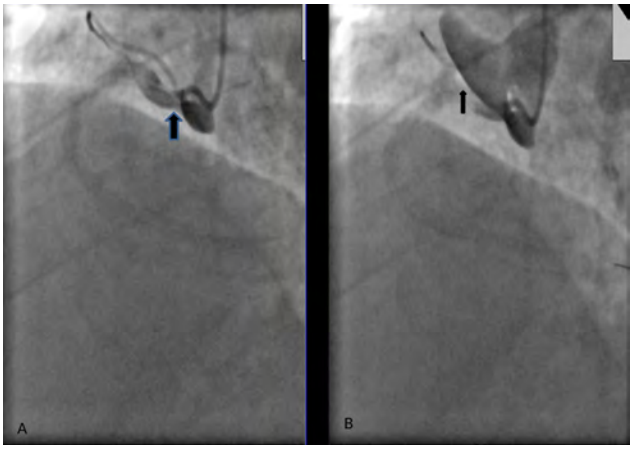

Figure 1. (A) Coranary dissection of the proximal RCA by AL2 guiding catheter. **(B)** Propagation of the dissection to the aortic root with contrast injection.

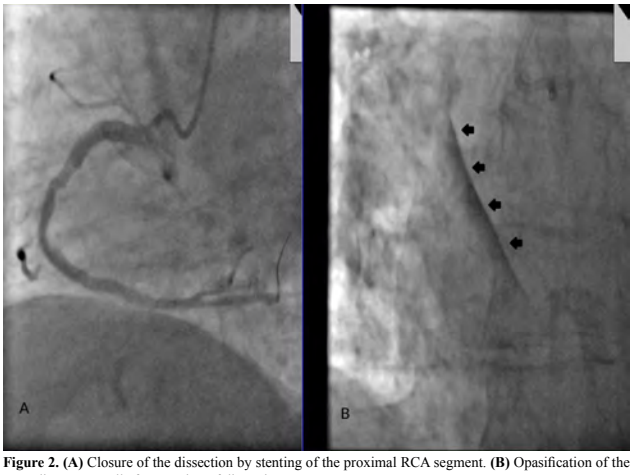

Figure 2. (A) Closure of the dissection by stenting of the proximal RCA segment. **(B)** Opasification of the ascending aorta wall after stenting of dissection.

PO-162

Sol ana koroner artere acil şartlarda girişim

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Amaç: Korumasız sol ana koroner (LMCA) perkütan koroner girişimleri (PCI) yüksek morbidite ve mortalite riski taşıyan işlemlerdir. Acil şartlarda kardiyojenik şoktaki miyokard enfarktüslü hastaların hemen her zaman tek seçeneği PCI'dır. Olgumuz gece nöbet şartlarında acil olarak hastanemize sevk edilen bir miyokard enfarktüsü olgusu olup hemodinamik olarak unstabil idi. Hasta yakınlarının onayı alınarak LMCA primer PCI işlemi yapıldı.

Olgu Sunumu: 70 yaşında erkek hasta üç gündür idrar çıkışında azalma ve genel durum bozukluğu ile PCI olanağı olmayan bir hastanenin acil servisine başvurmuş. Yapılan değerlendirmelerinde yaygın ön yüz ve inferor miyokard enfarktüsü ile uyumlu elektrokardiyografi bulguları saptanmış. Kreatinin 2.9 mg/dl olan olgunun yakınlarınında onayı ile kurumumuza acil primer PCI için sevki yapıldı. Acil servisimizde TA 77/42, nabız 122/dk ve derisi nemli olarak saptanan bilinci kapanma eğilimindeki hasta hemen koroner anjiyografi laboratuvarına alındı. Hastaya tikagrelor 180 mg ve asetilsalisilik asit 300 mg verildi. Unfraksiyone heparin 5000 IU IV bolus yapıldı. Dobutamin ve noradrenalin desteğine başlandı. Hastanın koroner anjiyografisinde LMCA %99 lezyon saptandı (Video 1). Hemen hasta yakınlarına bilgi verildi. Hastanın işlem sırasında hayatını kaybedebileceği ancak başka seçeneğinde o an için bulunmadığı belirtildi. Yakınlarının imzalı onamı alındı ve hemen işleme geçildi. Ek doz 5000 IU UFH yapıldı. 7F JL 4 katater ile LMCA'ya yaklaşıldı. LAD'ye doğru arkasında balon hazır şekilde PT-II MS tel ile geçildi. Cx'e işe başka bir PT-II MS teli gönderildi. Hemen balon LMCA'ya ilerletildi (2.5x20 mm). 15 atm'ye kadar pre-dilate edildi. Hastada herhangi bir aritmi olmadan balon indirilerek geri alındı. Akımın düzeldiği görüldü. Hemodinami hızla düzeldi ve TA 110/65 mmHg seviyelerine geldi Ardından 3.5x23 mm DES (Xience V) lezyona LAD'ye doğru implante edildi (Video 2). Cx teli implantasyon sonrası geri alındı ve stentin stratları arasından geçilerek tekrar Cx distaline indirildi. Proksimal optimizasyon tekniği ile 4.5 15 balon ile LMCA postdilate edildi. Ardından 3.0x25 mm balon LAD'ye ve 2.75x20 mm balon Cx'e doğru kissing yapacak şekilde LMCA ostiumuna hizalandı. Kissing balon işlemi sonrası optimal açıklık sağlandı ve işlem sonlandırıldı (Video 2). Ertesi gün hastanın idrar çıkışları arttı. İşlemden üç gün sonra servise alınan hastanın kreatinin değeri 1.6 seviyelerine geriledi. Toplam beş gün hastanede kalan hasta sağlıklı bir şekilde taburcu edildi Hastanın LAD lezyonu için elektif PCI planlandı.

Sonuç: Acil durumlarda LMCA primer PCİ işlemleri başarılı bir şekilde yapılabilir.

PO-163

Marijuana kullanımına bağlı ST segment yükselmeli miyokardiyal enfarktüsü ile başvuran hastada çoklu koroner arter trombüsü

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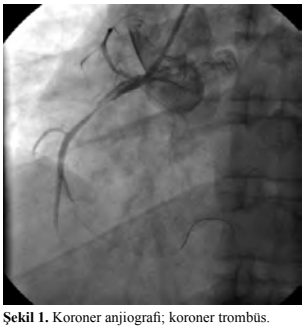
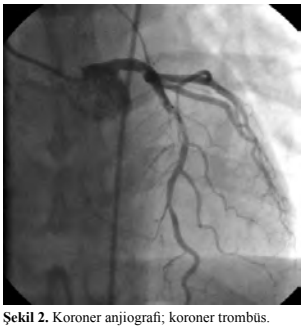
Akut miyokard enfarktüsü (ME) en sık aterom plağının rüptürü sonucu oluşan trombüsün koroner arter lümenini tıkamasıyla ortaya çıkar. Madde kullanımı özellikle genç yaştaki popülasyonda daha sık olarak görülmektedir. Esrar kannabinoid (KB) grubunda yer alan bir madde olup esrar kullanımının ME ile ilişkili olduğu bilinmektedir. KB'ler kalp hızı ve kan basıncında artışa neden olur. KB koroner arterlerde vazospazm ile birlikte karboksihemoglobin düzeyinin artmasına ve oksijen taşıma kapasitesinin azalmasına bağlı miyokardiyal oksijen sunum dengesinin bozulmasına yol açar. Ayrıca KB faktor VII aktivitesinde ve trombosit aggregasyonunda artışla ilişkilidir. 37 yaşında, KB kullanımı olan erkek hasta 2 saatlik göğüs ağrısı ile acil servise başvurdu. Akut İnferiyor ME tanısı ile koroner anjiyografi (KAG) laboratuvarına alındı. Yapılan KAG'de Sağ koroner arterde (RCA) tromboze lezyon balon ile predilate edildikten sonra metal kaplı stent implante edildi. Yoğun trombüs yükü nedeniyle tirofiban infuzyonu başlanarak işleme son verildi.Genç yaş grubunda akut ST yükselmeli ME daha nadir olarak görülür. Akut inferiyor ME nedeniyle hastanemize başvuran hastaya yapılan koroner anjiyografide sol ön inen arter (LAD) stent içinde, intermedier arter (İMA) osteali ve RCA ostealinde yoğun trombüs saptandı. RCA ostealindeki yoğun trombüs sonrası proksimalden tam tıkalı olan hastaya trombüs aspirasyonu sonrası stent işlemi yapıldı. Daha önceden madde bağımlılığı öyküsü olan ve hemodinamik parametreleri normal olan hastanın LAD ve İMA tromboze lezyonları için ek girişim düşünülmedi ve tirofiban infüzyonu başlandı. Yapılan kontrol anjiyografide LAD ve İMA trombüsünün tamamen kaybolduğu saptandı. Biz olgumuzda madde kullanımı sonucu gelişen ME yanısıra enfarkt ilişkili olmayan koroner arterlerde yaygın trombüs gelişebileceğini belirtmek istedik. Madde kullanımı plak rüptürüne bağlı trombüs oluşumu ve ME yanı sıra infarkt ilişkili olmayan koroner arterlerde trombüs oluşumuna yol açabilir.



Şekil 1. Koroner anjiografi; koroner trombüs.



Şekil 2. Koroner anjiografi; koroner trombüs.

PO-164

Left main coronary disease equivalent coronary microvascular dysfunction

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Case Report: 62 year-old man who has been followed with Type 2 Diabetes Mellitus for 12 years and hypertension for 20 years admitted to our hospital with dizziness and grayout after exertion. His symptoms had become frequent last one month and begun to be even one flight of stairs. His neurologic examination, Carotid Doppler Ultrasonography and cranial MRI was normal. Echocardiography was normal except mild left ventricular hypertrophy and grade 1 diastolic dysfunction. Exercise stress test was performed on the purpose of evaluation for ischemia and hemodynamic response to exertion. Horizontal ST depressions on inferolateral derivations were seen at fifth minute of test. At tenth minute, dizziness and grayout occurred and test was ended (figure 1-2). Even as his blood pressure was 117/78 at baseline, it dropped to 85/55 at ninth minute. We thought that hypotension after exertion and early ST depressions may be the cause of left main coronary or multivessel disease. We performed coronary angiography. Coronary arteries were normal (video 1-2). Invasive coronary flow reserve (CFR) was performed for differential diagnosis of microvascular dysfunction. LAD:1.62, Cx:4, RCA:4 was detected at invasive CFR. Our diagnosis was myocardial microvascular dysfunction involves heterogeneously in myocardia of left ventricle, which is limited to the area of LAD artery.

Discussion: Coronary microvascular dysfunction (CMD) is typical angina with evidence of myocardial ischemia in the absence of flow-limiting stenosis on coronary angiography. Coronary microvascular dysfunction often leads to a patchy distribution rather than to a limited area of ischemia, as typically seen in coronary artery disease. What makes our case interesting is CMD which confined to LAD territory as obstructive coronary artery disease and hypotension at stress test as LMCA or multivessel coronary disease without any significant stenosis.

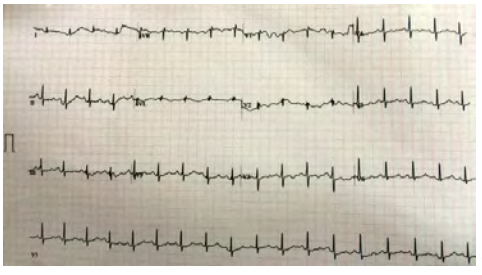

Figure 1. Exercise stress test at fifth minute.

PO-165

Klopidogrele karşı ticagrelor: Yaşam ve ölüm arasındaki ince çizgi

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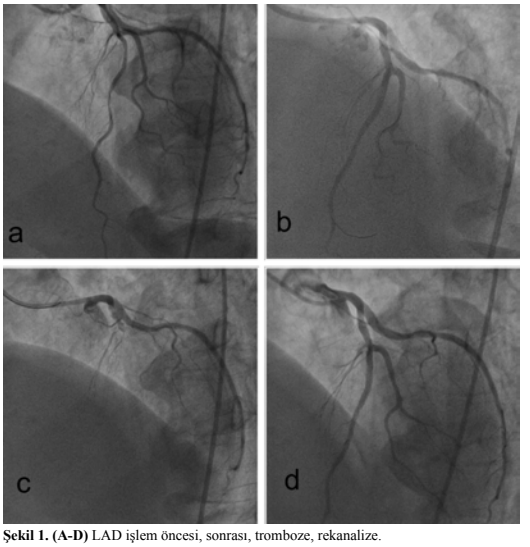
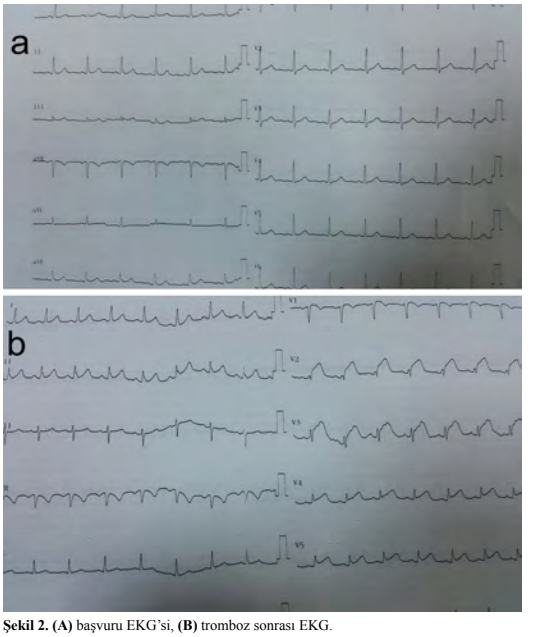
Uygun antiagregan seçimi stent trombozu komplikasyonunu engellemede önemli olabilir.

Olgu: Polikliniğimize başvuran 56 yaşındaki diyabetik hipertansif kadın hastaya anstabil angina pektoris tanısı ile koroner anjiyografi yapıldı. Hastanın fizik muayene, EKG, transtorasik ekokardiyografi ve laboratuvar değerleri normaldi (Şekil 2). Koroner anjiyografisinde LAD' %70 %60 %80 ardışık lezyonlar, Cx proksimalde %50 darlık, RCA normal saptandı. Hastaya işlem sırasında kullandığı asetil salisilik asit'e ek olarak 600 mg klopidogrel verildi ve 7500 IU unfarksiyone heparin IV tedavisi altında her üç lezyonu kapsayacak şekilde LAD arterine distalden proksimale 2,5x20 mm, 2,75x9 mm ve son olarak 3,0x24 mm DES (ilaç kaplı stent) yerleştirildi ve post dilatasyon yapılarak işlem sonlandırıldı. İşlemden 8 saat sonra akut stent trombozu gelişti (Şekil 2) ve LAD arterine yerleştirilen stentlerin proksimalden tromboze olduğu gözlendi. LAD'e yapılan girişimsel rekanalizasyon çabalarının netice vermemesi üzerine işlem TIMI 0 akım ile sonlandırıldı ve intraaortik balon desteği sağlandı. Klopidogrel kesilerek ticagrelor 180 mg yükleme dozu sonrası 90 mg 2x1 olacak şekilde ikili antiagregan tedavisi yeniden düzenlendi. Kontrol amaçlı yapılan 1 ay sonraki koroner anjiyografisinde stentlenen LAD arterin açık olduğu ve TIMI 3 akıma sahip olduğu gözlendi (Şekil 1).

Tartışma: Stent trombozu; uygulama sonrası sıklığı %0.5-2 arasında bildirilen, mortalitenin %45'e kadar yükseldiği önemli bir sorundur. Akut stent trombozu; stent uygulamasından sonraki 24 saat içerisinde stentin trombüsle kapanması olarak kabul edilmektedir.Tikagrelor yeni antitrombosit ilaçlardandır. PLATO çalışmasında stent trombozu açısından klopidogrelle karşılaştırılmış ve tüm hasta gruplarında stent trombozunu önlemede daha etkin olduğu gösterilmiştir. Olgumuzda tromboz sekizinci saatte gelişmiştir. Tikagrelor bu komplikasyon sonrası uzun vadede TIMI akımın korunmasında ilk tedavi seçeneği olarak düşünülebilir.



Şekil 1. (A-D) LAD işlem öncesi, sonrası, tromboze, rekanalize.



Şekil 2. (A) başvuru EKG'si, **(B)** tromboz sonrası EKG.

PO-166

LMCA distal ve LAD-D1 lezyonuna IVUS kılavuzluğunda JSBT metodu ile başarılı perkutan girişim

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69 yaşında erkek hasta polikliniğimize başvurdu. 1 hafta önce başka bir merkezde UAP tanısıyla KAG yapılmış ve By-Pas kararı verilmiş. Hasta operasyonu kabul etmemiş ve merkezimize PCI amaçlı refere edilmiş. KAG: LMCA distalden başlayıp D2 hizasına kadar uzanan kritik darlık mevcut. CX osteal bölgede %50 darlık mevcut (Dış merkezde %70 oarak rapor edilmiş). D1 proksimalde %60 darlık mevcut. RCA:Mid bölgede %70 darlık mevcut. Hastanın KABG'ı kabul etmemesi üzerine RCA, LMCA ve LAD ye PKG kararı verildi. 1.Seansta RCA ya başarılı PKG uygulandı. 2 hafta sonra LMCA girişimi planlandı. LMCA İşlem özeti: LAD, CX ve D1 GW ile geçildi. Daha sonra LAD D2 bölgesinden başlamak üzere IVUS yapıldı. Buna göre LAD osteal bölgenin ciddi hastalık içerdiği CX osteal bölgenin ise önemli darlık içermediği saptandı. Bu nedenle CX'e provizyonel yaklaşılmasına karar verildi. Öncelikle LAD' ye 2.5-15 bl ile predilatasyon uygulandı. Ardından JSBT (Jailed and semi-inflated baloon protection tecnique) metodu ile LMCA'dan D2 sonrasına uzanacak şekilde Xcience pro 3.0-38 mm stent implante edildi. Ardından balon koruma eşliğinde LAD'ye 3.5-15 NC balon ile postdlatasyon uygulandı. Daha sonra 4.0 bl ile LMCA ya postdilatasyon uygulandı. Daha sonra IVUS ile kontrol edildi. LMCA stent açıklığının yeterli olmadığı, CX osteal bölgenin ise etkilenmediği görüldü. Ardından 4.5-12 NC balon ile LMCA'ya postdilatasyon uygulandı. IVUS kontrolünde stent apozısyonunun yeterli olduğu görüldü. İşleme son verildi.

PO-167

Pulmonary arterial stent attempt for pulmonary arterial stenosis due to lung cancer

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Background: Stent approach for stenosis in pulmonary artery due to malignancy is a very rare condition accounting for only a few cases worldwide. We herein report a case with lung cancer whose symptoms were controlled by stent placement of pulmonary artery.

Case: A 69-year old man underwent a right lobectomy for non-small cell lung cancer on June 2012. The patient received four cycles of adjuvant cisplatin-vinorelbine chemotherapy. On November 2014, he presented with a three-week history of dyspnea. Examination showed a 3/6 pansystolic murmur and previous thoracic surgery. Chest computed tomography (CT)-angiogram showed a 45 mm thickening-mass surrounding pulmonary artery on the right and infiltrating heart. Also, main pulmonary artery was minimally dilated and right pulmonary artery was moderately stenotic. PET (pozitron emission tomography) revealed a 67x158 mm mass on the left lung, a new nodule on the inferior lobe of right lung and new paraaortic lymph nodes with an involvement among muscles on left gluteal region. Biopsy showed a non-small cell carcinoma. Transthoracic echocardiography reflected a severe pulmonary hypertension with a pressure approximately at 80 mmHg and also lateral side of left ventricle and left atrium were involved by the malignant mass. Pulmonary angiogram showed a 95% stenosis; the stenosis was first dilated by a 8 mm-balloon and then a 10 mm and 12 mm ballon inflation was done respectively, after balloon dilatation; a 10,0x39-mm left pulmonary stent was inserted (Omnilink Elite peripheral stent, Abbott vascular, Santa Clara, CA USA). First peak pulmonary artery pressure was 60 mmHg and after stent process the pressure decreased to a level of 40 mmHg finally. Dyspnea was improved after the process. He was begun to receive a doublet chemotherapy regimen including platinium and taxanes. On the second cycle, shortness of breath appeared again and a new thorax CT showed progression and echocardiography was compatible with ejection fraction decreasing from 55% to 35%. Because of progression after just first cycle of chemotherapy, he was thought to be resistant to sistemic therapy and was referred to palliative radiotherapy.

Discussion: Pulmonary artery compression due to malignancies particularly lung cancer has been reported in case reports. While first case of pulmonary artery stenosis due to malignancy was reported in 1984, first successful stent placement case report was performed in 2002 and among these only five cases were presented. Most of the cases showed signs of stenosis due to the first malignancy. Our case is an example of successful insertion of stent in pulmonary artery and the response of pressure of pulmonary artery did well locally. This is the first case for pulmonary artery stent implementation due to second primary lung cancer after approximately 2 and a half year diagnosis with first lung cancer.

PO-168

A case of renal embolia treated percutaneously,need not surgery

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A 64-year old male was admitteed to our emergency service with a several hours history of pain over the right flank region. A contrast enhanced spiral computed tomography (CT) was performed for a definitive diagnosis. Occlusion of the right renal artery was detected (Figure 1A) and also showed the left ventricular apical thrombus (Figure 1B). On emergency condition,we engaged a 7F Renal guiding catheter in the right renal artery ostium,then a 5F Judkins Righ (JR) diagnostic catheter was advanced through that renal guiding catheter to extract thrombus mechanically with manual aspiration. Some of thrombus was aspirated. In an attempt to recanalize the renal artery, 10 mg of recombinant tissue plasminogen activator was directly administrated into the renal artery. So we performed a balon angioplasty with a 3,0 mm x 20 mm semi-complian balon, distal embolic protection was not used during the procedure and there was no evidence of distal embolization in complete angiography.

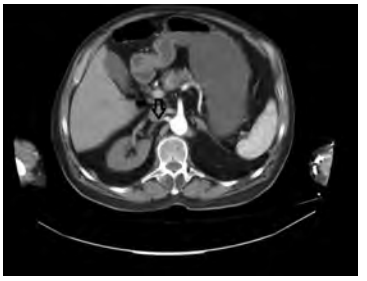

Figure 1. CT shows the obstructed right renal artery.

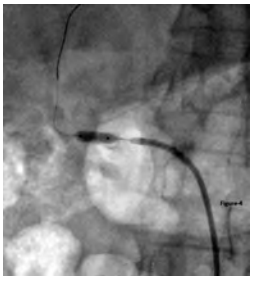

Figure 2. Baloon angioplasty procedure.

PO-169

Vertebrovertebral steal sendromunda periferik perkütan girişim

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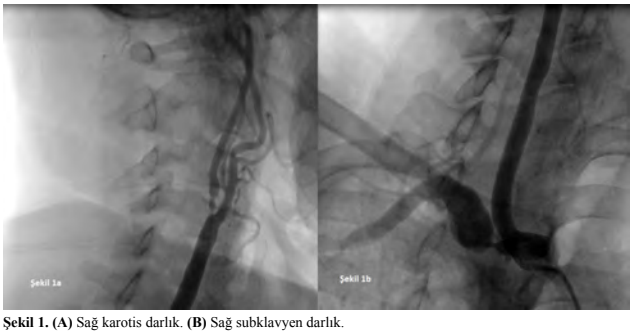
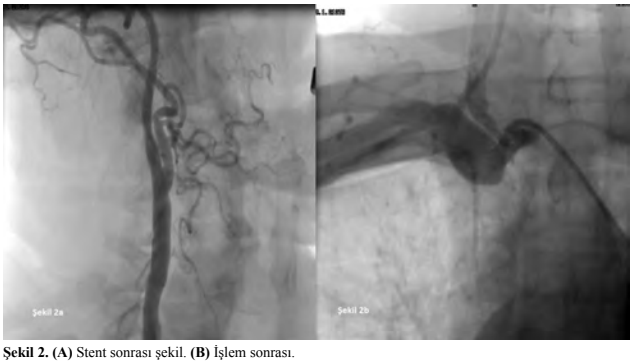
73 yaşında erkek hasta 1,5 yıl önce TİA sonrası yapılan bilateral karotis görüntülemede sağ karotis arterde kritik darlık saptanmış ve sağ karotis endarterektomi uygulanmış. 5 aydır olan başdönmesi şikayeti nedeniyle araştırılırken yapılan bilateral karotis DSA anjiyografide sağ internal karotis arterde kritik darlık saptanması üzerine kliniğimize refere edildi. Hastaya yapılan karotis anjiyografide sağ internal karotis arterde kritk darlık saptandı (Şekil 1a). Ayrıca üst eksremitede sağ-sol tansiyon farkı olması üzerine yapılan üst eksremite periferik anjiyografide sağ subklavyen arterde 80 mm hg grandyent oluşturan kritik darlık izlendi (Şekil 1b). Sağ vertebral arterin sol vertebral arterden dolduğu görüldü (Video 1). Konsey sonrası subklavyen ve karotis artere stentleme kararı alındı. Ayrı bir seansta sağ karotis arterdeki kritik darlığa 9,0x6,0x40 mm stent implante edildi (Şekil 2a) sağ subklavyen arterdeki kritik darlığa 6,5x12 mm balon-expandable stent implante edildi (Şekil 2b). Takiplerde baş dönmesi şikayeti gerileyen hasta medikal tedavisi düzenlenerek taburcu edildi.



Şekil 1. (A) Sağ karotis darlık. **(B)** Sağ subklavyen darlık.



Şekil 2. (A) Stent sonrası şekil. **(B)** İşlem sonrası.

PO-170

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 into the left subclavian artery distal to the occlusion. Percutaneus transluminal subclavian artery stenting was succesfully performed. The reduction of efor angina and claudication of arm was provided.

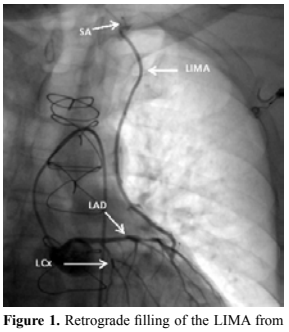

Figure 1. Retrograde filling of the LIMA from the LAD.

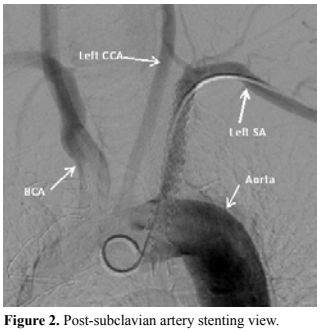

Figure 2. Post-subclavian artery stenting view.

PO-171

İntraabdominal aortik anevrizmalı bir hastada başarılı total oklüde SFA rekanalizasyonu

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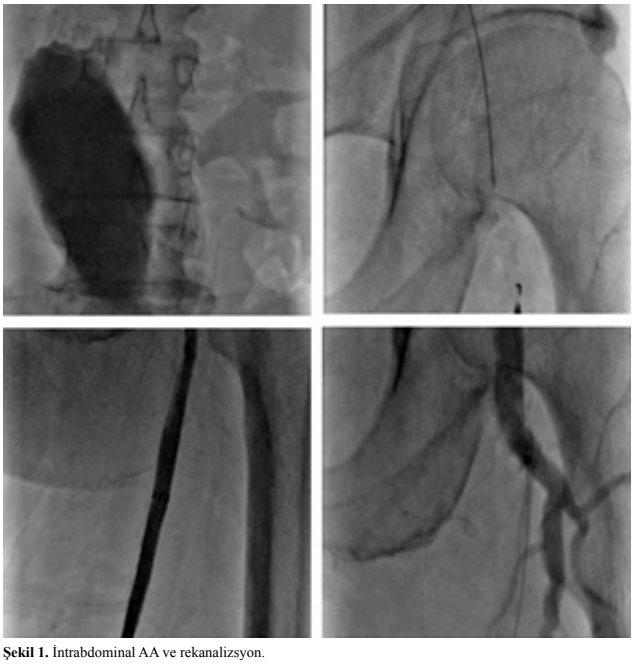
İzmir Üniversitesi Tıp Fakültesi, İzmir

73 yaşında erkek hasta yürümekle ortaya çıkan sağ bacakta kramp tarzında ağrı ile başvurdu. BT anjiyoda abdominal aortada trombüs içeren 55x43 mm çapında anevrizma mevcuttu. Periferik anjiyografide sağ SFA'nın proksimalinden oklüde olduğu izlendi. Total tıkalı SFA proksimalinden antegrade rekanalize edilmek amacıyla glidecath eşliğinde control wire ile geçilmeye çalışıldı. Ancak wire ilerlemedi. Ayrı seansta hasta prone pozisyona alındıktan sonra sağ popliteal arterden USG eşliğinde ponksiyon yapıldı. Retrograde olarak oklüzyonun proksimaline kadar control wire ilerlendi. Ancak proksimalde gerçek lümene ulaşılamadı. Gerçek lümen ulaşmak amacıyla Outback kateter lezyon proksimaline ilerletildi.Re-entry kanülü kullanılmadan önce gerçek lümene ulaşmak amacıyla öncelikle 0.014 inch distal uçdan açı verilmiş Conquest pro koroner tel ile geçilmeye çalışıldı. Distal uçtan çıkarılan telin proksimal kontrol injeksiyonundan lümende olduğu gözlendi. Outback kateter geri alındıktan sonra PTA ve stentleme işlemi başarı ile gerçekleştirildi.



Şekil 1. İntrabdominal AA ve rekanalizsyon.

Girişimsel kardiyoloji / Karotis ve periferik vasküler

PO-172

Successful carotid artery stenting despite distal tortuosity by using emboshield distal protection system

Ahmet Hakan Ateş, Uğur Arslan, Aytekin Aksakal, Mehmet Yaman, Huriye Yücel, İlksen Atasoy Günaydın

Samsun Training and Research Hospital, Samsun

Introductio: 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 buildup 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 angıoplasty was planned for treatment of carotid stenosis with distal tortuosity (Figure 1). After neurology consultation the patient was taken to angiography laboratory. 8F sheath was fixed to right femoral artery and 8F right coronary guiding catheter was placed to right common carotid artery. Later, the emboshield distal protection system was placed to distal region of lesion with support of extra support grand slam guidewire. Afterwards tapered self expandable carotid stent was impanted (6-8x 30 mm) and the post dilatation was done with 5x15 mm balon expandable balloon (Movie 1).

Discussion: Carotid angioplasty/stenting is a minimally invasive endovascular procedure that compresses the plaque and widens the lumen of the artery.

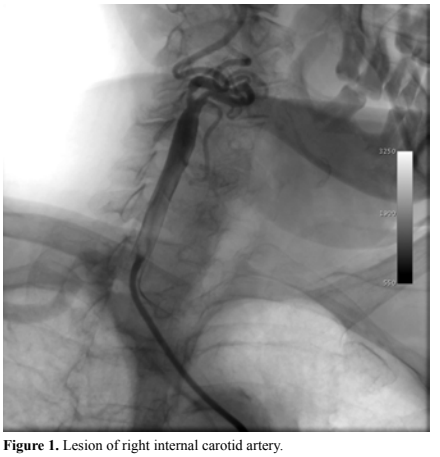


Figure 1. Lesion of right internal carotid artery.

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalıkları

PO-173

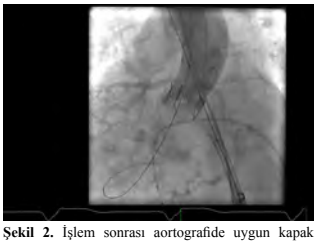
Dextrokardi ve aort stenozu bulunan bir hastada başarılı gerçekleştirilen TAVI işlemi

Hakan Erkan, Levent Korkmaz, Şükrü Çelik

Ahi Evren Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi, Trabzon

81 yaşında bayan hasta NYHA 3-4 septom ile hastanemiz acil servisine başvurdu. Hastanın bilinen dextrokardisi dışında kardiyak kökenli bir hastalık öyküsü yoktu. Hastanın fizik muayenesinde her iki akciğer bazal kesimlerinde ral ve sol 2. İnterkostal aralıkta sistolik karakterde üfürüm tespit edildi. Yapılan ekokardiyografide hastanın ileri aort stenozu olduğu ve aort kapak alanının 0.7 cm^2 olduğu saptandı. Hasta yoğun bakıma alınarak medikal tedavi altında semptomları gerilediken sonra kapağa yapılacak olası bir girişim açısından kalp ekibi tarafından değerlendirildi. Euroscoru 11 olarak hesaplanan hastaya TAVI yapılması kararlaştırıldı. TEE de aortik anulus çapı 19 mm olarak ölçüldü. Hastanın yapılan bilgisayarlı tomografisinde ve aort kökü anjiografisinde dextrokardisinin olduğu ve aort kavsinin olması gerekenin tam tersi şekilde olduğu izlendi (Şekil 1). Femoral arterden yapılacak TAVI işlemi açısından engel oluşturabilecek herhangi bir periferik vasküler hastalık saptanmadı. Hasta kateter labaratuarına alındı. Yapılan aortografi sonrasında sağ oblik -kadudal projeksiyonun kapak yerleşimi açısından en uygun düzlemin olduğu görüldü. Kalsifik aort kapağa süper sert tel üzerinden 20 mm balon yapıldıktan sonra 23 mm Edvards saphien XT kapak hızlı pace eşliğinde yerleştirildi. Yapılan kontrol aortografide kapağın uygun pozisyonda implante edildiği ve anlamlı bir aort yetersizliğinin olmadığı gözlendi (Şekil 2). Komplikasyon gelişmeyen hastanın femoral cut down kapatılarak işleme son verildi.

Şekil 1. Aortografide ters aort kavsi.



Şekil 2. İşlem sonrası aortografide uygun kapak yerleşimi.

PO-174

TAVİ'de vasküler komplikasyonları azaltmak için uygulanan modifiye cross over balon oklüzyon tekniğinde gelişen çok nadir bir komplikasyon: Emboli

Sina Ali[1], Mehmet Bilge[2], Yakup Alsancak[1], Ayşe Saatçi Yaşar[1], Zeynep şeyma Turinay[1], Murat Can Güney[1], Hakan Süygün[2], Melike Polat[2], Gamze Zengin[2]

[1]*Atatürk Eğitim ve Araştırma Hastanesi, Ankara*
[2]*Yıldırım Beyazıt Üniversitesi Tıp Fakültesi, Ankara*

Giriş: Periferik vasküler komplikasyonlar transkateter aortik valv implantasyonu (TAVİ)'nda mortaliteyi artıran önemli faktörlerden biridir. TAVİ uygulamalarının erken dönemleri ile karşılaştırıldığında şu anda vasküler komplikasyonlar azalmış olsa da yine de yüksek oranlardadır. Son zamanlarda vasküler komplikasyonların cross over balon oklüzyon tekniği ile önemli derecede azaldığı bildirilmektedir. Bildirimizde bu tekniğin modifiye şeklinin uygulanımı sırasında güvenlik kılıfının koparak iliyak artere embolizasyonu gelişen bir olguyu sunmaya çalışacağız.

Olgu: 84 yaşında ciddi semptomatik aort darlığı olan hastaya yüksek cerrahi risk nedeniyle TAVİ kararı alındı. TAVİ işleminin başında güvenlik amacı ile ilk olarak standart femoral arter giriş yerinin biraz distalinden olacak tarzda 5F kılıf yerleştirildi. Daha sonra floroskopi eşliğinde femur başının ortasından olacak seviyede arteriyel giriş sağlanarak öncelikle 6F kılıf daha sonra da sırayla iki adet Proglide kapatma cihazı yerleştirildi. Hemen sonrasında 9F kılıf yerleştirildi ancak geçiş esnasında biraz direnç hissedildi. 9F kılıf yerleştirildikten sonra Amplatz superstiff tel gönderilirken 9F kılıfın hemen distalinde daha önce konan 5F kılıfın bir kısmının koparak iliyak arter içinde kaldığı görüldü (Şekil 1). Bu sırada kasık bölgesinden kılıfın diğer kısmının dışarı çıktığı görüldü. Bunun üzerine 0.035 inch kalınlığında hidrofilik özelliği olan bir tel ile içeride kalan 5F kılıfın içinden geçilmeye çalışıldı ve bir müddet sonra bu sağlandı, (Şekil 2). Ancak 0.035 inch ile çalışacak periferik balonun şişirildikten sonra kılıf ile beraber 9F kılıfın içine girmeyeceği düşünülerek bu sefer 0.014 inch tel 5F kılıf içinden geçirildi. Tel üzerinden 2.5 mm koroner balon kılıfın üzerinden gönderilerek kılıf ve 6F klavuzun arasında şişirilerek 9F kılıf içine alınmaya çalışıldı ancak başarısız olundu (Şekil 3). Bunun üzerine snare ile kılıf bir müddet uğraş sonunda dışarıya alındı. Daha sonra standard tarzda 18 F kılıf yerleştirilerek işleme devam edildi ve Simetis 27 mm kapak başarılı bir şekilde implante edildi (Şekil 4).

Tartışma: Son zamanlarda TAVİ'de vasküler komplikasyonların cross over balon oklüzyon tekniği ile önemli derecede azaldığı bildirilmektedir. Bu teknikteki mantık; asıl femoral giriş yerinin biraz distaline tercihen 4F yoksa 5F kılıf konması daha sonrada normal giriş yerinin elde edilmesidir. İşlemin herhangi bir fazında veya daha sıkılıkla da TAVİ sonrası 18 F kılıfın çekilerek kapatma cihazlarının sütürlerinin ilerletilmesi sonrası herhangi bir istenmeyen vasküler komplikasyon gelişimi durumunda bu kılıf daha geniş kılıfların yerleştirilmesine olanak sağlar ve her türlü vasküler müdahele buradan yapılabilir. Sonuç olarak bu olgu modifiye cross over balon oklüzyon tekniği uygulanırken femoral artere girerken fonksiyon iğnesi ile ilk yerleştirdiğimiz güvenlik kılıfına girmemek için dikkatli olmamız gerektiğini göstermektedir.

PO-175

CoreValve biyoprotez kapak ile TAVI sonrası çok nadir bir komplikasyon: Abdominal aorta kapak embolizasyonu ve fonksiyonel ikinci aort kapak

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Giriş: Transkatater aort kapak replasmanı (TAVI), cerrahi açıdan yüksek riskli olan hastalarda tercih edilen etkin bir tedavi biçimidir.İşlem sırasında izlenen biyoprotez kapak embolizasyonu artan hasta sayılarıyla birlikte daha sık (CoreValve %2.6-8.1, Edwards Saphien %2-5.3) görülmektedir. Bildirimizde; ciddi aort darlığı ile takip edilen bir hastada TAVI işlemi sırasında meydana gelen kapak embolisi ve buna bağlı olarak ikinci bir biyoprotez aort kapağa sahip olan vaka sunulmaya çalışıldı.

Olgu Sunumu: Diyabet, hipertansiyon, kronik böbrek hastalığı ve koroner arter hastalığı (10 yıl önce üç damar by-pass) ile takip edilen 76 yaşındaki kadın hasta yeni gelişen nefes darlığı (NYHA 3) şikayeti ile polikliniğimize başvurdu. Yapılan transtorasik ekokardiyografisinde ejeksiyon fraksiyonu %60, ciddi aort darlığı (92/60 mmHg, AVA 0.5 cm^2) ve sol ventrikül hipertrofisi saptandı. Koroner anjiyografi sonucunda hastanın greftlerinin açık olduğu gözlendi ve kalp damar cerrahisi ile birlikte değerlendirilmesi sonucu cerrahi açıdan yüksek riskli olduğu düşünüldü (EuroScore %20). TAVI işlemine alınan hastaya 29 mm CoreValve biyoprotez kapak implantasyonu planlandı. Uygun biçimde aort kapağı geçilerek hızlı pacing altında (200 atım/dk) 22 mm balon ile predilatasyon yapıldı. Ardından 29 mm kapak sistemi uygun biçimde yüklenerek, sistem açılmaya başlandı (Video 1,2). Kapak açılımı sırasında kapağın ventrikül içerisine doğru yer değiştirdiği gözlendi (Video 3,4). Kapak geri çekilmek istendi ancak işlem sırasında kapak aort kökünden ayrıldı (Video 5). Kapağın tekrar sistem içerisine çekilerek dışarıya alınması planlandı, fakat sistemin mekanik hasar nedeniyle çalışmadığı ve kapağın sistemden kurtularak abdominal aorta seviyesine embolize olduğu ve sisteme ait olan hornun (sistemin uç kısımında bulunan piramit) kapak içerisinde olduğu görüldü (Video 6). Bununla birlikte kapağın fonksiyonel olarak aktif olduğu, distal akıma engel olmadığı ve hastanın hemodinamisinin stabil olduğu görüldü (Video 7,8,9). Hastanın klinik durumu göz önüne alınarak cerrahi için yüksek riskli olduğu düşünüldü ve işleme devam edilmesi planlandı. Ikinci bir 29 mm CoreValve kapak sisteme yüklenerek, abdominal aortada bulunan biyoprotez kapaktan geçilerek uygun biçimde yerleştirildi (Video 10). İşlem sonrasında aort kapakta 5 mmHg gradient ve hafif derecede AY izlendi. Ardından abdominal aortada bulunan kapak içerisinde bulunan hornun, yakalanarak femoral artere çekilmesi ve cerrahi olarak alınması planlandı, ancak kapaktan kurtarılan horn internal iliyak artere düştü. Semptomatik olmayan hastada klinik takip planlanarak işleme son verildi.

Sonuç: Operatör tecrübesi ile birlikte; kullanılan kapak türü, aortik anülüs ve uygun kapak çapı, aort yetersizliğinin ciddiyeti, yetersiz pacing hızı, kalsifikasyon yetersizliği ve kapağın sistemden ayrılmaması gibi sebebler kapak embolizasyonu için önemli faktörlerdir. Bildiğimiz kadarıyla, sunduğumuz vaka ilk olması bakımından önemlidir.

Girişimsel kardiyoloji / Kapak ve yapısal kalp hastalıkları

PO-176

PDA kapama işlemi sırasında kullanılan kapama cihazının pulmoner artere embolizasyonu

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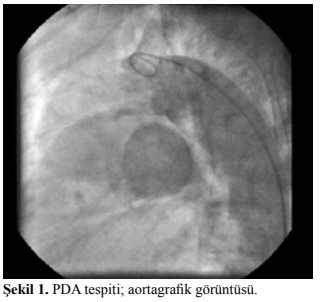
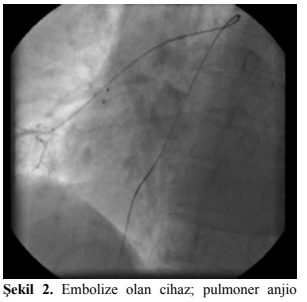
Kliniğe nefes darlığı şikayeti ile başvuran 71 yaşında bayan hastaya yapılan EKO incelemesinde sol ventrikül endsistolik ve enddiyastolik çapında genişleme ve patent duktus arteriosus (PDA) tespit edildi. Yapılacak kateter-anjiyo işlemi için sağ femoral arter ve ven kullanıldı. Kanülizasyon sonrası Pigtail kateter yardımı ile aortografi görüntüleri alındı. PDA tespit edildi (Şekil 1). Cihaz ölçümleri yapıldı. PDA 7F delivery sistem kullanılarak 0.35 inç 260 cm guide wire ile PDA geçildi. Akabinde 8x10mm PDA ocluder device (Amplatz Duct Occluder-ADO) implante edilmek istendi. Ancak cihaz serbest bırakıldığı sırada defekt alanından pulmoner sisteme geçerek embolize oldu (Video 1). Cihazın pulmoner arterdeki konumu tespit edildi ve snare kullanılarak eksternalize edildi (Şekil 2). Ardından 10x12 mm PDA occluder-ADO device pulmoner arter yolu ile defekt bölgesine konumlandırılarak implante edildi. Aortografik görüntüler alınarak işlem başarı ile sonlandırıldı (Video 2). Bu olgu ile 71 yaşında bile olsa PDA kapama işleminin perkütan yolla başarılı bir şekilde yapılabileceğini ortaya koymaya çalıştık. Ayrıca işlem sırasında oluşabilecek emboli gibi komplikasyonlara dikkat çekmek ve oluşabilecek bu tarz komplikasyonların nasıl üstesinden gelinebileceğine dair bir örnek sunmak istedik.



Şekil 1. PDA tespiti; aortagrafik görüntüsü.



Şekil 2. Embolize olan cihaz; pulmoner anjio görüntüsü.

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Ektazik koroner arter hastalığı ve iskemik mitral yetersizliği bulunan bir hastada PKG, MitraClip uygulanımı ve 2 yıllık takip sonucu

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Giriş: Koroner arter ektazisi (KAE), koroner arterlerde normal segmente göre lokalize damar genişlemesi olarak tanımlanır. Tedavi edilmeyen olgularda tekrarlayan akut koroner sendromlar izlenmekte ve kardiyomiyopati gelişebilmektedir. Bu hastalarda eşlik eden iskemik mitral yetersizliği (MY), sonraki dönemlerde ortaya çıkacak olan KY ve ölüm riskini göstermesi bakımından önemlidir. Bu durum özellikle düşük EF'li hastalarda çok daha belirgindir. Bildirimizde, KAE'si bulunan bir hastada meydana gelmiş ciddi MY için uygulanan MitraClip işlemi ve perkütan koroner girişimin 2 yıllık takibi sunulmuştur.

Olgu Sunumu: 10 yıldır koroner arter hastalığı ile takip edilen hasta 2 yıl önce tekrarlayan akut koroner sendrom atakları olması sebebiyle hastaneye yatırıldı. Daha önce yapılan koroner anjiyografisinde ektazik koroner arterler izlenen hastanın, ekokardiyografisinde ileri derecede iskemik MY saptandı (Video 1,2,3) (Şekil 1). Yapılan ekokardiyografisinde ejeksiyon fraksiyonu %15 (EDÇ:6.5, ESÇ:6.0) tespit edilen hasta kalp damar cerrahisi ile birlikte değerlendirildi ve hastanın cerrahi açıdan yüksek riskli olduğu düşünüldü. Tekrarlayan göğüs ağrıları olan hastaya perkütan koroner girişim planlandı. Belirgin KAE olan hastanın sağ koroner arterine (4.5x18 mm stent), LAD'ye (3.5x18 mm stent), sirkümfleks'e (2.75x16 mm ve 2.75x20 mm stent) implante edildi. İşlemden 1 ay sonra hasta MitraClip işlemi için tekrar hospitalize edildi. A2-P2 skallopuna tek klip yerleştirilerek 1+ ≤ MY ile işlemden çıkıldı. 2 yıl içerisinde KY ya da herhangi bir sebeble hastaneye yatış ihtiyacı olmadı. Düzenli olarak kontrol muayenelerine gelmeyen hasta atipik göğüs ağrısı yakınması ile tekrar kliniğimize başvurdu. İşlem öncesi NYHA fonksiyonel sınıfı 3 olan hastanın takiplerinde fonksiyonel kapasitesinde belirgin düzelme (sınıf 1-2) olduğu görüldü. Yapılan kontrol koroner anjiyografisinde stentlerin açık olduğu, kontrol ekokardiyografisinde 1+ ≤ MY olduğu izlendi (Video 6,7,8).

Sonuç: Ciddi MY ile beraber koroner arter hastalığı olan hastalarda standart tedavi koroner bypass cerrahisine ek olarak uygulanan kapak onarımı ya da replasmanıdır. Ancak bu durum ileri derecede bozulmuş sistolik disfonksiyonlu hastalarda yüksek mortalite oranlarını beraberinde getirmektedir. Bu vakamızda KAE nedeniyle tekrarlayan akut koroner sendrom yaşayan hastalarda revaskularizasyonun gerekliliğini, yine aynı hastada ileri MY nedeniyle uygulanan MitraClip tedavisinin 2 yıllık sonucunu bildirmeye çalıştık.

31. ULUSLARARASI KATILIMLI TÜRK KARDİYOLOJİ KONGRESİ OLGU SUNUMLARI YAZAR DİZİNİ

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