

## OS-01

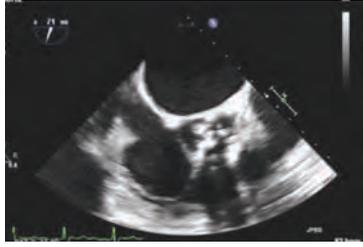
**Left main coronary artery obstruction caused by calcification on aortic valve after a successful transfemoral aortic valve implantation****Başarılı transfemoral aortik kapak replasmanı sonrası aort kapagı üzerindeki kalsifikasyona bağlı sol ana koroner arter tıkanıklığı**

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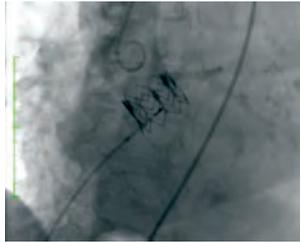
Transcatheter Aortic Valve Implantation (TAVI) is an effective and reliable treatment method in patients with severe aortic stenosis who present high surgical risks or in patients ineligible for surgical intervention. Despite this fact, various complications can develop as a result of this procedure. One of such complications is occlusion of left main coronary ostium due to calcification or by native valve cusp or by bioprosthesis. In some broad series, its prevalence in postoperative period after TAVI is determined to be %0.6. A 78 years old female patient diagnosed with severe degenerative aortic stenosis for 4 years. She had been receiving medical treatment for hypertension (HT), diabetes mellitus (DM), severe chronic obstructive pulmonary disease (COPD) and cardiac insufficiency. Results of the Transthoracic Echocardiography (TTE) showed severe aortic stenosis (mean gradient:59 mmHg), moderate tricuspid regurgitation, systolic pulmonary artery pressure 48 mmHg and left ventricular ejection fraction (EF) %65. The result of transesophageal echocardiogram (TEE) showed that there were large calcifications on and in the tricuspid valve structure of aortic valve. Under general anesthesia and with guidance of the TEE, the patient received Edwards Sapien XT 26 mm valve implantation, all accompanied with rapid pacing. An arcus aorta angiography showed that the right and left coronary arteries were non-selectively open and a mild AR. The procedure was considered successful. For 2 hours after the TAVI, the patient was hypotensive (TA:70/50) and bradycardia (42/min). On the ECG of the patient, intraventricular conduction delay and bradycardia were detected. The result of the TTE performed in CICU determined that the left ventricle EF of the patient was very low (%15) and no complication was observed on the valve. Upon development of cardiac arrest, cardiopulmonary resuscitation (CPR) accompanied by intrarterial monitoring started. The result of KAG accompanied by continuous CPR showed that the LMCA was completely obstructed due to calcification. Predilatation was performed with 3,5x12 mm and 4,0x12 mm balloons by means of breaching the calcification on the LMCA with a guide wire, all under CPR. After predilatation, 4,0x12 mm bare metal stent was implanted and thus full patency was obtained. Since the necessary blood pressure was not obtained, despite achieving coronary flow, peripheral femoral artery was taken to the femoral vein pump so as to reduce the work load of the heart with the aid of cardiovascular surgery. For three hours, intermittent off-pump performed yet required hemodynamic were not achieved. After a total of 7,5 hours, the patient still had asystole and was withdrawn from the pump and thus announced dead. In this case report a patient who suffered acute obstruction on left main coronary artery which was occluded by native valve calcification after TAVI and intervention after this occurrence is presented.



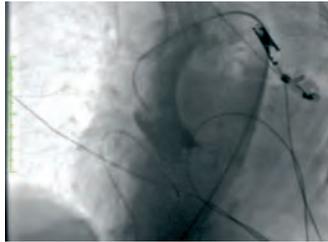
**Figure 1.** Transesophageal echocardiography image showing calcifications on the aortic valve.



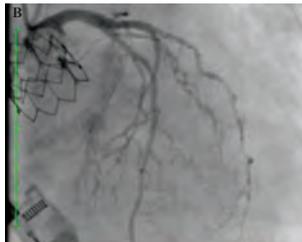
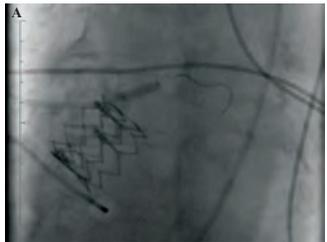
**Figure 2.** Coronal cardiac CT scan images showing calcific aortic valve and aortic annulus-left main coronary artery distance.



**Figure 3.** Fluoroscopic image of arcus aorta and aortic valve after aortic valve implantation.



**Figure 4.** Fluoroscopic image of arcus aorta and left main coronary artery obstructed with calcification.



**Figure 5.** Fluoroscopic image of stenting left main coronary artery (A) and patency of left main coronary artery (B).

## OS-02

**An unusual complication of a primary percutaneous coronary intervention****Primer perkutan koroner girişimin nadir bir komplikasyonu**

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A 76-year old female patient who had had coronary by pass surgery 8 months ago was referred to our hospital with the diagnosis of acute inferolateral myocardial infarction. she had been defibrillated due to ventricular fibrillation in her first hospital which have not primary coronary intervention (PCI) capability. Electrocardiography revealed minimal ST segment elevation in leads D2-D3-AVf and marked ST depression in leads V1-V3. She was immediately transferred to angiography laboratory immediately. Coronary angiography showed that left internal mammarian artery graft to left anterior descending artery and saphenous graft to right coronary artery was open. Circumflex artery was totally occluded. The patient received 10000 unit unfractionated heparin intravenously and 600 mg clopidogrel orally and 300 mg acetylsalicylic acid orally on the angiography table. The patient underwent successful coronary intervention and stent implantation of the circumflex artery. Thrombolysis In Myocardial Infarction-3 flow was ensured. This patient did not received glycoprotein 2b/3a antagonist. Then the patient was taken to coronary care unit for follow up. Examination revealed a swollen tongue with a marked blue-black discoloration suggestive of lingual hematoma just one hour after the procedure (figure 1a and b). The patient could not even close her mouth. We realized that she had bitten her tongue when she had had ventricular fibrillation. Clopidogrel and acetylsalicylic acid were continued. She remained under close follow up for airway obstruction, and the hematoma resolved gradually within several days (figure 2a and b). Lingual hematoma is a very very rare complication of primary PCI. This was the first case in which we encounter a lingual hematoma. We should keep in mind such an unusual complication when we deal with a patient who was defibrillated or resuscitated.



**Figure 1A and B**



**Figure 2A and B**

OS-03

### Percutaneous closure of an aortic prosthetic paravalvular leak with device in a patient presenting with heart failure

#### Kalp yetersizliği ile takrir olan bir hastada aortik prostetik paravalvuler kaçığın cihazla perkütan kapatılması

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**Introduction:** Paravalvular leaks are a well-recognized complication of prosthetic valve replacement. Perivalvular prosthetic regurgitation causes significant morbidity, and is associated with high perioperative mortality if open surgical repair is required. Paravalvular leaks manifest with symptoms of congestive heart failure, hemolysis, or in most cases, the combination of both. Percutaneous transcatheter closure of paravalvular leaks with specific device causes symptomatic improvement. We presented a case of transcatheter closure of aortic paravalvular insufficiency with Amplatzer Duct Occluder 2 device.

**Case:** A 57-year-old male patient presented with progressive dyspnea, New York Heart Association (NYHA) class III-IV and massive ascid was admitted to our clinic. He had a history of mitral valve replacement for serious mitral insufficiency and aortic valve replacement for severe calcific aortic stenosis in 2010. He had repetitive hospitalizations for decompensated heart failure for the period of three months. On examination he had a regular pulse rate of 82 beats/min, and blood pressure of 120/70 mmHg. The cardiopulmonary examination revealed a grade 2/6 early diastolic decrescendo murmur, clear prosthetic click and bilateral crackles in the lung bases. He had massive ascid, pretibial and scrotal edema. The transthoracic and transesophageal echocardiography showed mildly decreased left ventricular systolic function with left ventricular hypertrophy and a normally functioning metallic prosthetic valve however severe paravalvular insufficiency at the edge of the sewing ring and the native tissues around the valve was detected (Figure 1). Procedure was performed under transesophageal echocardiography guidance with general anesthesia. Right femoral artery puncture was performed and paravalvular aortic regurgitation was evaluated by aortography. Leak was passed by a hydrophilic terumo 0.35 inch guide wire and 5 F delivery catheter was placed to the left ventricle. Paravalvular aortic leak diameter was calculated using transesophageal echocardiography and 4 mm Amplatzer Duct Occluder II was chosen. ADO II was loaded into the delivery system and left ventricle side was opened first and aortic side was opened secondly. During the opening of aortic side, disappearance of leak was observed and device was released. (Figure 3). Complete disappearance of leak was confirmed by aortography after the 20th minutes of beginning. There was not any complication due to the procedure and the patient was discharged from the hospital following day.

**Conclusion:** Paravalvular aortic insufficiency is a complication of surgical aortic valve replacement and it is associated with poor prognosis. As in our case, paravalvular aortic leakage causes the persistence of congestive heart failure and percutaneous aortic leakage repair is a good alternative to the redo surgery.

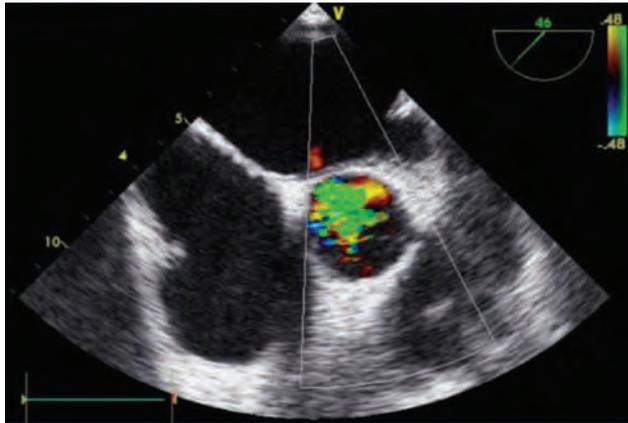


Figure 1. Transesophageal echocardiographic view of aortic prosthetic leak.

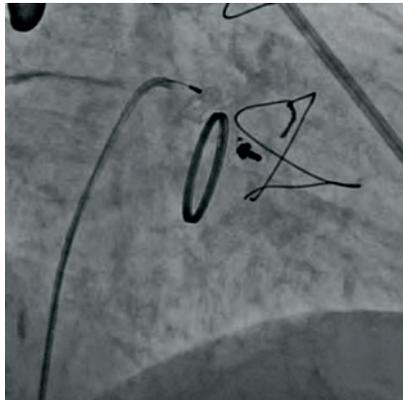


Figure 2. Releasing of the ADO II device.

OS-04

### Spontaneous coronary artery dissection in a healthy adolescent following consumption of caffeinated "Energy Drinks"

#### Sağlıklı bir ergende kafeinli "Enerji içeceği" tüketimi sonrası spontan koroner arter diseksiyonu

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A previously healthy 13 year old boy was admitted to our clinic after presenting with acute-onset, "crushing," mid-sternal chest pain over a period of about two hours. He had no history of diabetes, hypertension, hyperlipidemia, or cigarette smoking. He had no family history. He denied use of cocaine, amphetamines, hormones, steroids, alcohol, or other recreational drugs. The patient ingested an energy drink for the first time last night. About 8 hours after the high energy drink consumption the patient's chest pain started. Physical examination revealed a well-developed teen in moderate distress. His blood pressure was 120/70 mmHg, heart rate was 80 beats/min, and his respiratory rate was 16 breaths/min. Cardiac auscultation revealed an S4 gallop with a normal S1 and S2. The electrocardiogram (ECG) revealed sinus rhythm with 2- to 3-mm ST-segment elevations in leads II, III, aVF, and V3 through V5 (Figure 1A). The transthoracic echocardiography (TTE) showed left ventricular ejection fraction estimated to 0.54 and moderate apical hypokinesia. He had been given aspirin, subcutaneous enoxaparin, sublingual nitroglycerin, enalapril and metoprolol at presentation. After treatment the patient's chest pain relieved. Initial laboratory studies, within 4 hours of the onset of his symptoms, were normal white blood cell count, myoglobin level, creatine kinase MB fraction and mild elevated a troponin I level. The patient's chest pain decreased after medical treatment and for this reason we did not give thrombolytic therapy. After 4 hours of recorded ECG did not observe changes dynamically according to the baseline ECG (Figure 1B). The control troponin-I value after 24 hours increased. The dynamic T wave changes was observed in ECG recording leads V3-V5 (Figure 2). For these reasons the patient was transferred to a tertiary referral centre for coronary angiography. The left anterior descending (LAD) artery showed extensive dissection with visible tear from the distal part of the vessel. The TIMI (thrombolysis in myocardial infarction) flow grade was III (Figure 3). The right coronary artery and the circumflex artery were normal. Based on the morphology of the vessel with a dissection and TIMI III flow grade, we decided to manage this patient conservatively with close follow up. We continued low-molecular-weight heparin, antiplatelet therapy, and enalapril. A month later the control examination, the patient had no chest pain at all. Medical treatment was continued. Follow-up TTE revealed normal left ventricular function, with resolution of his apical hypokinesia. As a result, energy drinks may be one of the reasons leading to spontaneous coronary artery dissection. Energy drinks, especially for children as in this case can lead to serious adverse events. Should be considered consumption of energy drinks which was detected spontaneous coronary artery dissection in the young patients.



Figure 1. (A) eight hours after onset of chest pain 12-lead ECG showed (B) After 4 hours of recorded ECG did not observe changes dynamically according to the baseline ECG.

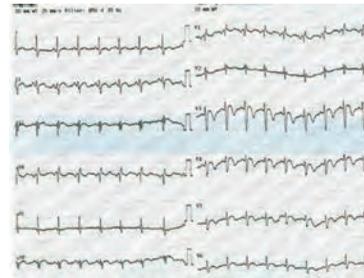


Figure 2. 12-lead ECG 24 hours after onset of chest pain showed leads V3-V5 was observed that dynamic T wave changes.

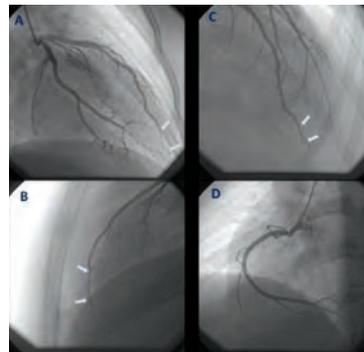


Figure 3. The left anterior descending artery showed extensive dissection (arrows) with visible tear from the distal part of the vessel and the circumflex artery were normal (A, B and C). The right coronary artery was normal (D).

OS-05

## Percutaneous mitral valve repair with the MitraClip system in a patient with severe mitral regurgitation caused by spontaneous papillary muscle rupture: the first case in the literature

**Spontan papiller kas rüptürünün sebep olduğu ileri mitral yetersizliği olan bir hastada MitraClip sistemi ile perkütan mitral kapak tamiri: Literatürdeki ilk vaka**

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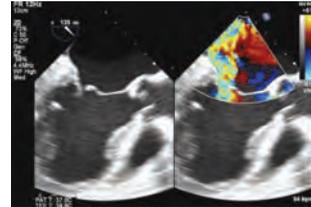
**Introduction:** Papillary muscle rupture (PMR) is usually associated with acute myocardial infarction. Spontaneous PMR, while unusual, has been noted to result from some causes. For patients with PMR, the standard therapy is surgical. We report, to the best of our knowledge, the first case of mitral valve repair with the MitraClip system of subacute severe mitral regurgitation (MR) caused by spontaneous PMR.

**Case:** A 73 year-old man with a history of chronic obstructive pulmonary disease, chronic hepatitis C, hypertension, DM and myocardial infarction in 2000 was referred to our hospital. He had been hospitalized 30 days earlier with a 1-week history of sudden onset of dyspnea with chest pain, paroxysmal nocturnal dyspnea, hemoptysis and decreased effort tolerance. Then, the patient was transferred to our institution. Initial examination at our hospital revealed the following: blood pressure 115/60 mm Hg, heart rate 92 bpm, bibasilar crackles on lung examination, and a holosystolic murmur at his apex. His initial ECG showed sinus rhythm and interventricular conduction defect. A transesophageal echocardiography demonstrated a part of calcified papillary muscle attached via chords to the anterior mitral valve leaflet (Figure 1). It showed also a posteriorly directed eccentric jet of severe MR and extensive calcification of both papillary muscles (Figure 2). A coronary angiography showed, a 40% distal left main stenosis, 70% proximal stenosis of the left circumflex coronary artery and a total occlusion of the mid right coronary artery with retrograde filling from the proximal right coronary artery from a collateral circulation. Since the cardiac surgery refused the patient due to the high-risk nature of surgery, the patient and his family were offered percutaneous repair of severe MR and informed consent was obtained. During the MitraClip procedure, we failed to grasp both leaflets at a central position. Finally, to stabilize anterior and posterior leaflets and to enable placement of target final clip, starting very close to the posteromedial commissure next to the central regurgitation jet, the first MitraClip was implanted. Then, the second MitraClip was directed towards the origin of the regurgitant jet mainly between A2 and P2 scallops and was implanted easily. In the end of the procedure, TEE demonstrated a significant reduction of MR grade from IV to grade I MR (Figure 3). On a control transthoracic echocardiography after the procedure, apical two-chamber view demonstrated chaotic echoes in the posteromedial papillary muscle (Figure 4).

**Discussion:** If PMR is untreated, the mortality could be as high as 80% during the first week and the mortality rate increases to 94% within 2 months. In our case, mitral valve repair with the MitraClip was selected because of the high-risk nature of surgery. This report indicates that severe MR with flail anterior mitral valve leaflet caused by spontaneous partial PMR could be treated by the MitraClip system.



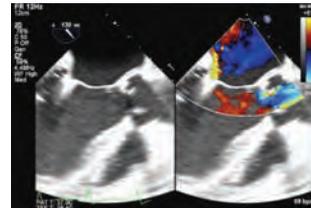
**Figure 1.** Transesophageal echocardiography demonstrates a part of calcified papillary muscle attached via chords to the anterior mitral valve leaflet moving like a whip in the left atrium during systole.



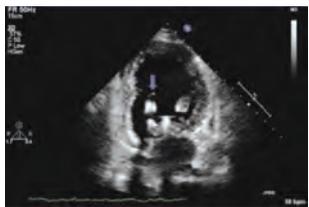
**Figure 2.** Transesophageal echocardiography indicates a posteriorly directed eccentric jet of severe MR from the middle scallops of both mitral leaflets (A2 and P2 segments) with flail anterior mitral valve leaflet.



**Figure 3.** Transesophageal echocardiography shows extensive calcification of both papillary muscles.



**Figure 4.** In the end of the procedure, transesophageal echocardiography demonstrates a significant reduction of MR grade from IV to grade I MR.



**Figure 5.** After the procedure, transthoracic echocardiography demonstrates chaotic echoes in the posteromedial papillary muscle in apical two-chamber.

OS-06

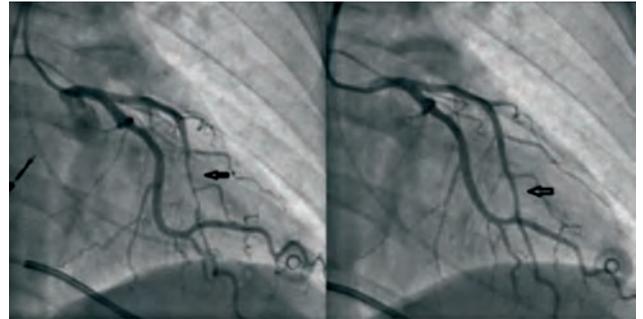
## Successful treatment of myocardial bridge with alcohol septal ablation in hypertrophic obstructive cardiomyopathy

**Hipertrofik obstruktif kardiyomiyopatiye alkol septal ablasyonu ile miyokardiyal köprünün başarılı tedavisi**

Murat Sunbul, Alper Kepez, Kursat Tigen, Okan Erdogan, Bulent Mutlu

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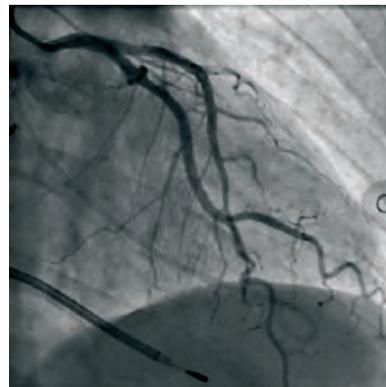
Hypertrophic obstructive cardiomyopathy (HOCM) is characterized by left ventricular (LV) hypertrophy of various morphologies, with variety of clinical manifestations and hemodynamic dysfunctions. Patients with HOCM usually have certain abnormalities including diastolic dysfunction, myocardial ischemia, mitral regurgitation, myocardial bridge (MB) and LV outflow obstruction related to excessive myocardial hypertrophy (1). These abnormalities can cause serious symptoms such as chest pain, palpitations, dyspnea, fatigue, and syncope due to myocardial ischemia and LV outflow obstruction. We present here a rare case of successful treatment of MB with alcohol septal ablation in hypertrophic cardiomyopathy which has not been reported previously. A 36-year-old man with a history of hypertrophic obstructive cardiomyopathy (HOCM) for ten years had recently presented with effort induced dyspnea and chest pain. Alcohol septal ablation was performed because of severe symptoms, including dyspnea and angina, five years ago and intracardiac defibrillator was implanted due to non-sustained ventricular tachycardia. Three years ago, the patient underwent coronary angiography due to anterior ischemia documented in myocardial scintigraphy, which revealed MB in left anterior descending artery (Figure 1). Recent transthoracic echocardiography showed asymmetric septal hypertrophy, a significant dynamic left ventricular outflow tract (LVOT) gradient of 89 mmHg at valsalva manoeuvre and systolic anterior motion of mitral valve. Alcohol septal ablation was performed due to recurrent severe symptoms. LVOT gradient was decreased and myocardial bridge was improved after alcohol septal ablation (Figure 2-3). The patient was discharged three days later and the symptoms were improved completely.



**Figure 1.** Right cranial view of left anterior descending and circumflex arteries during systole (left) and diastole (right) of myocardium.



**Figure 2.** Right cranial view; alcohol septal ablation was performed to third septal branch of left anterior descending artery.



**Figure 3.** Right cranial view; myocardial bridge improved remarkably after alcohol septal ablation.

## OS-07

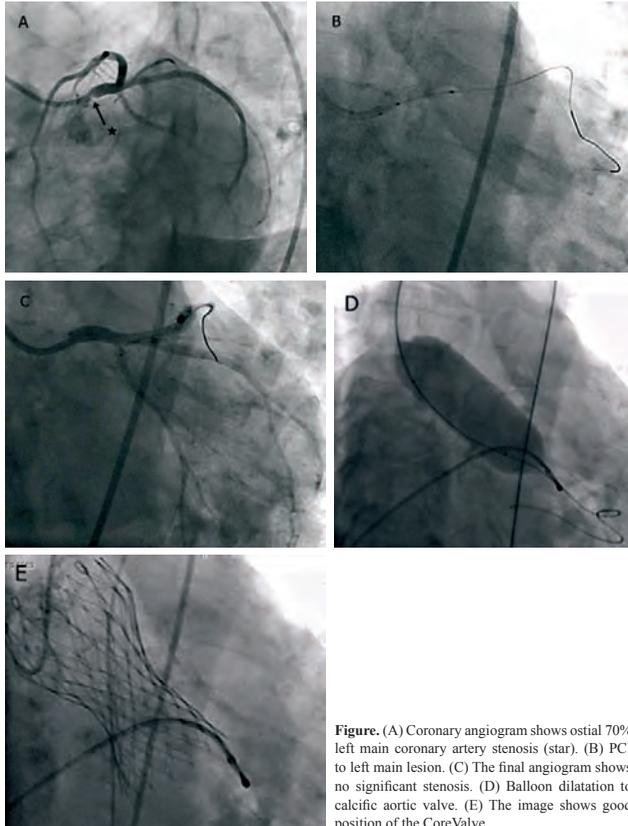
**Successful combined percutaneous coronary angioplasty for left main coronary disease and transcatheter aortic valve implantation: a case report****Başarılı kombine sol ana koroner arter için perkütan koroner anjiyoplasti ve kateter aortik kapak implantasyonu: Vaka sunumu**

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A 73-year-old male patient was admitted to our department with chest pain, syncope and dyspnea during daily physical activity. The patient had a medical history of hypertension, renal failure and chronic obstructive pulmonary disease (COPD). On examination, a 4/6 systolic ejection murmur was heard on aortic focus. The electrocardiogram showed sinus rhythm and findings of left ventricular hypertrophy. A transthoracic echocardiography revealed a calcified and immobilized aortic valve. Peak and mean aortic valve gradients were 83 mmHg and 48 mmHg respectively. His calculated aortic valve area was 0.70 cm<sup>2</sup> according to the continuity equation. Coronary angiography revealed a 70% stenosis on left main coronary artery (LMCA) ostium (Fig A). No significant stenosis were on other coronary arteries. Under the diagnosis of severe degenerative aortic stenosis (AS) and LMCA disease, the patient investigated for possible treatment options. The patients logistic Euroscore and Society of Thoracic Surgeons score was calculated 24.1% and 12.3% respectively. Because of high surgical risk score, percutaneous coronary intervention (PCI) and transcatheter aortic valve implantation (TAVI) considered to be the suitable treatment for the patient. A 7F sheath inserted to patients left common femoral artery (CFA). After cannulation of LMCA, a 0.014" guidewire was inserted to LAD. 3.5x12 mm zotarolimus eluting stent (Endeavor, Medtronic, USA) was deployed to ostial LMCA lesion (Fig B), after that stent dilated with 3.5x20 mm noncompliant balloon (NC Sprinter, Medtronic). The procedure was successful and the patient was hemodynamically stable after PCI (Fig C). The vascular access for CoreValve delivery catheter was obtained at the right CFA with standard percutaneous access techniques. Balloon dilatation of the stenotic aortic valve was performed with a balloon under rapid pacing using a temporary pacemaker (Fig D). Then a 29 mm CoreValve (Medtronic) was deployed at the aortic annulus under angiographic guidance (Fig E). Immediate post-procedural aortogram showed good position of the CoreValve. The patient was discharged from hospital five days later and he has been uneventful for 6 months of the follow-up.

**Discussion:** Patients with AS frequently have concomitant coronary artery disease, necessitating combined aortic valve replacement and coronary artery by-pass surgery which are associated with increased surgical risk. Treating concomitant CAD prior to TAVI appears to be reasonable, as severe CAD might have a negative effect on the safety of the TAVI procedure, especially because of the need for rapid pacing and anesthesia. Our case had unstable hemodynamics and high surgical procedure risk. Therefore we preferred both LMCA stenting and TAVI at the same procedure. While data regarding the outcome of combined TAVI with PCI are still insufficient, our case suggests that a combined percutaneous procedure is acceptable and safe, for selected high-risk patients with both coronary artery and aortic valve disease.



**Figure.** (A) Coronary angiogram shows ostial 70% left main coronary artery stenosis (star). (B) PCI to left main lesion. (C) The final angiogram shows no significant stenosis. (D) Balloon dilatation to calcific aortic valve. (E) The image shows good position of the CoreValve.

## OS-08

**Ablation of premature ventricular contractions of epicardial origin within the coronary sinus****Koroner sinüs içinden epikardiyal kaynaklı ventriküler erken vuru ablasyonu**Alptuğ Tokatlı<sup>1</sup>, Fethi Kılıçaslan<sup>2</sup>, Mehmet Uzun<sup>3</sup>, Bekir Sıtkı Cebeci<sup>3</sup><sup>1</sup>Golcuk Military Hospital, Department of Cardiology, Kocaeli<sup>2</sup>Florence Nightingale Hospital, Department of Cardiology, Istanbul<sup>3</sup>GATA Haydarpaşa Hospital, Department of Cardiology, Istanbul

**Introduction:** The quality of life may be significantly impaired in patients with ventricular premature contractions (VPC), especially when they are frequent. Besides, VPCs may cause significant complications such as ventricular arrhythmias and ventricular dysfunction. If there is clinical indication, endocardial radiofrequency (RF) ablation can be done successfully in most patients. But, VPCs may rarely be originate from epicardial site. In this article, we present a patient with VPC originating from an epicardial site that was ablated within the CS successfully.

**Case:** 31-year-old man was admitted to our hospital with palpitation. Physical examination was normal except for irregular pulse. ECG showed unifocal VPC's. VPCs were characterized by inferior axis and left bundle branch block pattern with a precordial transition zone at V3. Transthoracic echocardiography findings were within normal limits. Exercise test was normal except for VPCs. Holter ECG demonstrated very frequent, unifocal VPCs including couplets and bigemines despite antiarrhythmic therapy. We attempted electrophysiological study and catheter ablation for frequent VPCs. Patient was in sinus rhythm and had frequent VPCs at EP laboratory. A decapolar and a quadripolar catheters inserted into appropriate position. Basal intracardiac measurements were within normal limits. Because we could not induce ventricular tachycardia, we decided to map VPCs. An epicardial focus was suspected due to a pseudo delta wave in the precordial leads and a prolonged maximal deflection index (MDI= time from onset of QRS to the maximal deflection / QRS duration). The decapolar catheter was advanced to very distal within the CS (to anterolateral region). The earliest ventricular activation recorded at distal CS. A left coronary angiography was done and left aortic cusp was mapped by ablation catheter. Here, ventricular activity was earlier at distal CS than left aortic cusp. Local activation time was 50 ms earlier than ventricular activation at the ECG. No phrenic nerve stimulation was seen during high output pacing from distal CS. Coronary angiography was performed before ablation to show the relation between ablation site and epicardial coronary arteries. RF ablation (20 W; 60°C) was applied within the CS. VPCs were disappeared immediately. After RF ablation, the patient did not have any symptom and Holter ECG were normal 2 weeks later.

**Discussion and Conclusion:** Epicardial origin should be considered in patients who had unsuccessful endocardial ablation attempts and who has pseudo delta wave and prolonged MDI at the surface ECG. RF ablation can be applied within the CS successfully for VPCs of epicardial origin. During RF ablation within the CS, phrenic nerve and coronary artery damage can be avoided by performing high output pacing and coronary angiography.

**Giriş:** Ventriküler erken atımlar (VEA) özellikle sayıca fazla olduklarında semptomatik olarak hastanın hayat kalitesini önemli ölçüde bozabilmeleri yanında nadiren ventriküler disfonksiyon ya da ciddi ventriküler aritmiler gibi önemli komplikasyonlara sebep olabilmektedir. Klinik olarak endikasyon oluştursa hastaların çoğunda radyofrekans (RF) kateter ablasyon endokardiyal yoldan başarı ile uygulanabilir. Ancak nadiren VEA'lar epikardiyal kaynaklı olabilir. Yazımızda, KS içinden başarı ile RF ablasyonu yapılan epikardiyal kaynaklı bir VEA olgusu sunulmaktadır.

**Olgu:** 31 yaşında erkek hasta çarpıntı ve ritim düzensizliği şikayetleri ile hastanemize başvurdu. Fizik muayene aritmik nabız dışında normal idi. EKG'de unifokal VEA mevcuttu. VEA'lar, sol dal bloğu örneğinde ve inferior akslıydı. Transizyon bölgesi V3 derivasyonunda idi. Transtoraksik ekokardiyografi bulguları normal sınırlardaydı. Eforlu EKG testi VEA'lar dışında normal sınırlardaydı. Antiaritmik tedavi altında iken yapılan Holter EKG'de zaman zaman bigemine ve couplet gelen, çok sayıda, unifokal VEA izlendi. Hastaya elektrofizyolojik çalışma ve kateter ablasyon planlandı. Hasta sinüs ritiminde ve sık VEA'lar olduğu halde hemodinami laboratuvarına alındı. KS ve His kateterleri yerleştirildi. Bazal intrakardiyak ölçümler normal sınırlardaydı. Taşikardi uyarılamadığı için VEA'ların haritalanmasına karar verildi. EKG'de pseudo-delta dalgası olması ve maksimal defleksiyon indeksinin (MDI= QRS' in başlangıcından maksimal defleksiyonuna kadar geçen süre / QRS süresi) uzamış olması sebebiyle epikardiyal odak olabileceği düşünüldü. KS kateteri, KS içinde mümkün olduğunca distale, anterolateral bölgeye ilerletildi. VEA'lar esasında erken ventriküler aktivite KS distalindeydi. Sol koroner angiografi yapılarak RF kateteri ile sol aort küspisi maplendi. VEA esasında KS distalinde sol aort küspisten daha erken ventriküler aktivite vardı. KS distalinde VEA esasında yüzey EKG'deki ventriküler aktivasyondan 50 msn daha önce lokal ventriküler aktivasyon mevcuttu. KS distalinden yüksek outputla yapılan uyarı ile frenik sinir uyarısı olmadı. RF öncesi yapılan koroner anjiyografi ile ablasyon bölgesi ile major epikardiyal koroner arterlerin uzaklığı kontrol edildikten sonra bu bölgeye KS içinden RF uygulandı (20 Watt, 60 derece). VEA'ların kaybolduğu izlendi. RF kateter ablasyon uygulamasından sonra hastanın çarpıntı yakınması olmadı ve kontrol Holter EKG normal olarak bulundu.

**Tartışma ve Sonuç:** Özellikle endokardiyal yaklaşımla başarı sağlanamayan ve yüzey EKG'de pseudo delta dalgası varlığı ve uzamış MDI olan hastalarda epikardiyal odak aklı gelmelidir. Epikardiyal kaynaklı VEA'lara KS içinden başarı ile RF ablasyonu uygulanabilir. KS içinden ablasyon yapılırken koroner anjiyografi yapılarak koroner arterlere ve yüksek outputla uyarı yapılarak frenik sinire zarar vermekten kaçınılabilir.



**Figure 1.** ECG showing ventricular premature contraction with left bundle branch block and inferior axis. **Figure 2.** After successful ablation there is no ventricular premature contraction on surface ECG. **Figure 3.** Successful epicardial ablation side.

OS-09

**An interesting cause of third degree atrioventricular block: cause or coincidence?****Atriyoventriküler bloğun ilginç bir nedeni: Sebep ya da rastlantı?**

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We present a case of reversible third degree atrioventricular block in a young athlete without underlying heart disease. The patient is a healthy, 23-year-old man who presented with dizziness. ECG showed third degree atrioventricular block. He had no occupational exposures to solvents, did not use alcohol, recreational or herbal drugs. The patient repeatedly denied experimentation with anabolic steroids. He was an enthusiastic swimmer and had been only taking whey protein supplements for 4 weeks prior to the development of symptoms. A detailed evaluation for etiologies of third degree atrioventricular block including cardiac MR failed to show any causes. He had dramatic clinical and electrocardiographic improvement with recovery to sinus rhythm 10 days after first admission while discontinuation of the nutritional supplements. Holter monitoring 10 days after first admission and after discharge showed no atrioventricular block with an average heart rate of 76. The temporal relationship of whey protein consumption within several weeks of atrioventricular block can implicate it as the culprit agent or only coincidence. But clinicians must be more careful when evaluating atrioventricular block especially in athletes and may pay attention about a commonly used and reportedly safe whey protein supplements usage.



Figure 1. Third degree atrioventricular block at admission.

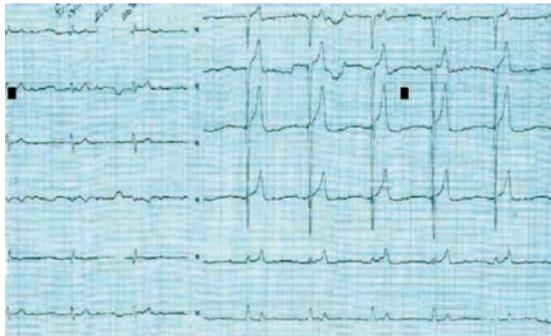


Figure 2. Third degree atrioventricular block one day after admission.

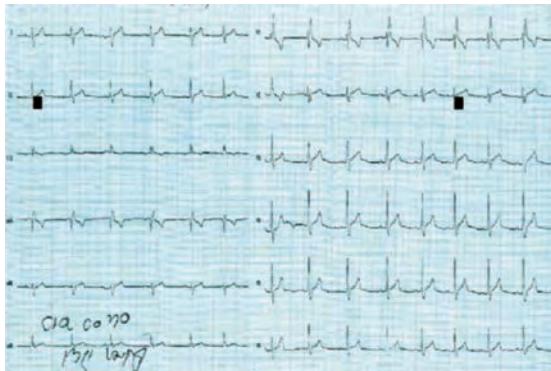


Figure 3. Recovery to sinus rhythm 10 days after first admission.

OS-10

**Association of persistent left superior vena cava and atrial fibrillation: ablation strategies depending on the anatomical variations****Persistan sol süperior vena kava ve atriyal fibrilasyon ilişkisi: Anatomik varyasyonlara bağli ablasyon stratejileri**

Emin Evren Özcan, Gabor Szeplaki, Bela Merkely, Laszlo Geller

Semmelweis University, Heart Center, Budapest, Hungary

In some rare cases, LVSC may persist and become a source of the AF episodes. Misinterpretation due to persistent LSVC and the approaches that might be used during pulmonary vein isolation (PVI) in presence of anatomical variations will be discussed in this presentation. A 61 years old male patient with history of PVI one year ago due to paroxysmal AF was referred to our unit for reablation because of the frequent drug resistant episodes. In the beginning of the intervention, during insertion of CS catheter, it stepped out of the heart shadow and the electrical signals disappeared. The trace of catheter was consistent with persistent LSVC. After insertion of CS catheter, we continued standard PVI protocol by using electroanatomic mapping (Ensite NavX mapping system, St. Jude Medical, Minneapolis, MN, USA). The second potentials were observed to elongate and disappear in the recordings of the left superior pulmonary vein (LSPV) during the wide area circumferential ablation of the left sided veins. However, localized sharp potentials just behind the far-field signals were notable (Figure 1A). These were thought to be far-field signals resulted from the anatomical contiguity of LSVC and LSPV (Figure 2A,2B). The potentials became prominent with advancing the circular multielectrode catheter from the ablation line toward distal part of the vein. Despite pacing from the LSPV at the lowest output capturing the vein, exit block could not be demonstrated (Figure 1B). It was difficult to understand whether it was originated from a gap at the ablation line or from the far-field capture resulted from the anatomic contiguity, without activation mapping of both sides. Moreover, this might also be caused by the connections between LA or LSPV and persistent LSVC which may show a propagation similar to the far-field capture. Pacing from the CS catheter localized in the LSVC-CS junction was capturing LSPV. Therefore, we decided to map LSVC. Circular multielectrode catheter was retrogradely introduced into LSVC through CS (Figure 2C). The mapping process was introduced from the CS junction and the catheter was advanced into the LSVC up to the level without electrical activity. Besides local sharp LSVC potentials following the far-field LA potentials (Figure 1D) premature ectopic beats with the earliest activation in LSVC were observed during the mapping (Figure 1E). Thereupon, LSVC was isolated, applying circumferential ablation at CS junction. Local sharp potentials disappeared and the ectopic pulses were not conducting to LA (Figure 1F). The pacing performed from LSVC with maximal energy did not capture LA. Then, ablation catheter was advanced into the LA again and exit block was demonstrated with pacing from LSPV. The circular catheter was inserted into LSPV through the same long sheath and the potentials persisting following PVI were observed to disappear (Figure 1C). The intervention was successfully completed without any complication.

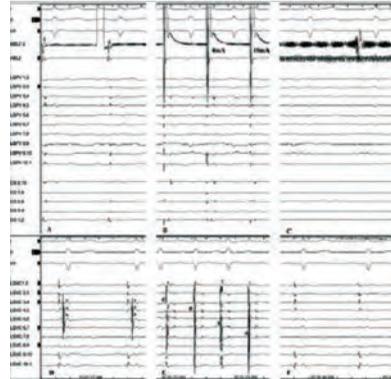


Figure 1. A,B,C are recordings from LSPV and D,E,F are from LSVC. (A) Sharp far-field signals resulted from the anatomical contiguity of LSVC. (B) Pacing from the LSPV at the lowest output capturing the vein, exit block could not be demonstrated. When output was increased to 10 mA LSVC was captured directly and far-field signals disappeared. (C) After isolation of LSVC sharp potentials disappeared. CS catheter is in the distal LSVC. LSVC recordings during sinus rhythm (D) and ectopic beats (E). Note both ectopic beats (#) and left atrial far-field signals are preceding surface p wave (E). Following isolation LSVC potentials (\*) abolished and only far-field LA signals remained (D and F).

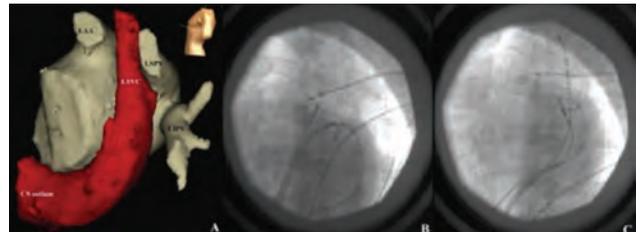


Figure 2. A) Computed tomography image integrated to NavX map. Note the anatomical contiguity between LSVC and LSPV. Fluoroscopic images in left anterior oblique projection demonstrating catheter positions. (B) Ablation and circular multielectrode catheters are in LSPV and CS catheter distal is in the LSVC-CS junction. (C) Ablation and circular multielectrode catheters are in LSVC and CS catheter is in the distal LSVC.

### Ventricular tachycardia based long QT without hypocalcemia after using of ibandronic acid

#### İbandronik asid kullanımı sonrası hipokalsemi olmaksızın uzun QT'ye bağlı ventriküler taşikardi

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**Introduction:** Long QT syndrome is important because it triggers life-threatening arrhythmias. Long QT syndrome can be divided into two forms as congenital and acquired. Various drugs may cause acquired long QT syndrome. Bisphosphonates are the agents widely used during osteoporosis treatment. A case of acquired long QT syndrome degenerated into malign arrhythmia associated with the use of ibandronic acid is presented.

**Case Report:** A female patient at the age of 57 years with complaint of palpitation applied to the external centre in an emergent state. She was transferred to our centre after beginning of medical cardioversion with intravenous amiodarone upon the detection of VT (figure 1). At the first admission the patient was on normal sinus rhythm with ventricular bigeminy extrasystole and her corrected QT interval was measured as 0, 53 seconds (figure 2). She has no associated family history and no cardiac risk factor other than HT. She has had no coronary artery disease from coronary angiography and had been using bisoprolol, valsartan and amlodipine previously. In addition, ibandronic acid treatment for her osteoporosis had been administered for two weeks. Routine biochemical tests of the patient were detected in normal range. Acquired long QT syndrome associated with ibandronate was especially taken into consideration for the patient. A series of ECG monitoring was performed during 2 weeks after the discontinuance of ibandronic acid medication and it was observed that ventricular extrasystoles disappeared and QTc interval returned to the normal value measured as 0, 42 seconds (figure 3).

**Discussion:** The top limit for duration of QTc interval according to heart rate is usually given as 0.44 seconds. Prolongation of ventricular action potential duration is seen as prolongation of QT interval in superficial ECG and is important in the sense that it causes torsades de pointes, one of the life-threatening arrhythmias. Long QT syndrome can be classified as congenital and acquired. Acquired form can be induced by antiarrhythmic agents, tricyclic antidepressant drugs, non-sedative antihistamines and antibiotics. It may also be caused by electrolyte abnormalities such as hypokalemia, hypocalcemia and hypomagnesemia, fasting, lesions of the central nervous system, apparent bradyarrhythmias, cardiac ganglionitis, and mitral valve prolapse. In our case, after medical cardioversion with amiodarone, ibandronate therapy causing long QT syndrome was discontinued and electrolytes were frequently monitored. During follow-up, the patient's QTc interval came back to normal range. In addition, there was no VT induced with programmed ventricular stimulation during diagnostic electrophysiologic study after the normalization of QTc. Consequently, a series of ECG monitoring should be performed on patients treated with bisphosphonates. Ibandronic acid may cause ventricular tachycardia after prolongation of QT even without development of any electrolyte abnormality.

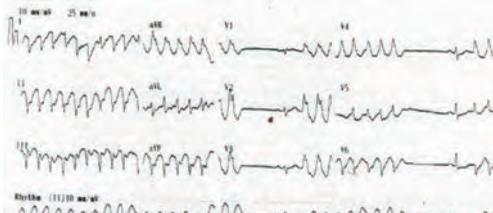


Figure 1. Monomorphic ventricular tachycardia and captured sinus beat seen on the surface 12 leads ECG took at first admission.

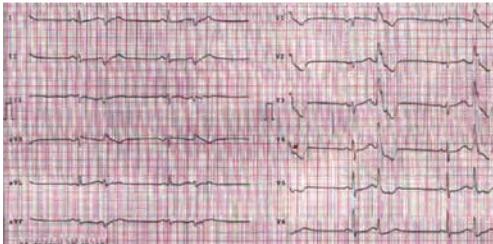


Figure 2. Normal sinus rhythm with ventricular bigeminy extrasystole and prolonged QT interval measured as 0, 53 seconds, after medical cardioversion.

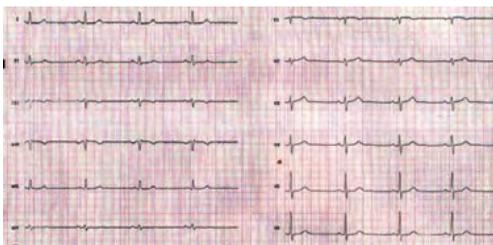


Figure 3. Corrected QT interval returned to the normal value measured as 0, 42 seconds after 2 weeks from the discontinuance of ibandronic acid medication.

### Pneumopericardium and pneumomediastinum following radiofrequency catheter ablation: a case report

#### Radyofrekans kateter ablasyonunu takiben pnömoperikardiyum ve pnömomediastiniyum: Vaka sunumu

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A 68-year-old man with a history of symptomatic paroxysmal Atrial fibrillation (AF) had failed prior pharmacologic therapies and he underwent two times percutaneous radiofrequency ablation and once cryoablation in last two years. Two weeks later after the last RFCA procedure, he was admitted to emergency department with chest pain, and was diagnosed with pericarditis, requiring oral steroids. Seven days later he returned to the emergency room with dyspnea and chest pain that was more severe when supine. Physical examination revealed distant heart sounds, blood pressure 90/50 mmHg and heart rate 125 bpm. On admission laboratory results showed highly elevated renal and inflammatory parameters (creatinin clirens: 2.4 mg/dl, C-reactive protein 39 mg/dL, white blood cell count of 14,500 cells/ $\mu$ L with a leftward shift). The patient was admitted to the internal medicine service with a diagnosis of acute renal failure. On chest x-ray, widened mediastinum, increased cardiothoracic index, mediastinal air-fluid level, visible edge of the pericardium due to increased radiolucency were determined. (Figure-2). Four days before chest x-ray was normal. (Figure-1). So, patient examined by department of chest diseases. Computed tomography (CT) scan of the chest showed air and fluid in the pericardial space and in mediastinum. (Pneumopericardium and Pneumomediastinum) (Figure 3-4). Patient was consulted with cardiology. On echocardiogram ejection fraction (EF) was 55%, pericardial effusion coat around the heart the largest side 2 centimeter. But, there was no sign of cardiac tamponade. According to the chest x-ray and echocardiography findings the patient diagnosed pneumopericardium and pneumomediastinum because of esophago-atrial fistule (AEF) and recommended emergency operation. Cardiac surgery was insufficient for this operation in our hospital. So, the patient referred to other hospital. While the patient was transporting in ambulance, symptoms of dyspnea was increased and finally become cardiopulmonary arrest. Intubated and CPR was started.. There was no response after 45 minutes CPR and the patient died. AEF is a rare but life-threatening complication after percutaneous catheter ablation, with a mortality rate of 80%. AEF after radiofrequency ablation for atrial fibrillation has a median time to appearance of 15 days. AEF must be remembered complication after cardiac ablation. A high chance of survival with early diagnosis and treatment of AEF.



Figure 1. Normal mediastinum, normal cardiothoracic index.



Figure 2. Widened mediastinum, an increased cardiothoracic index, mediastinal air-fluid level is present, visible edge of the pericardium due to increased radiolucency.

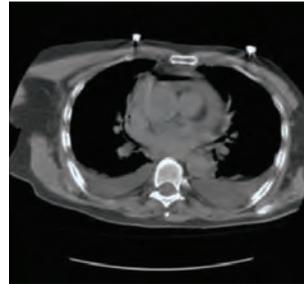


Figure 3. Air and fluid in the pericardial space and in mediastinum. ( Pneumopericardium and Pneumomediastinum). Opacities as lung atelectasis across the pericardium.

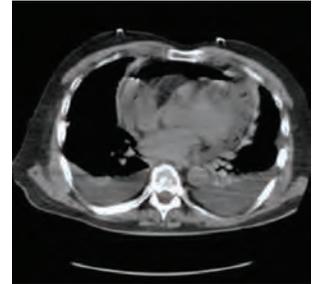


Figure 4. Air and fluid in the pericardial space and in mediastinum. ( Pneumopericardium and Pneumomediastinum). Opacities as lung atelectasis across the pericardium.

## OS-13

## Radiofrequency catheter ablation of supraventricular tachycardia in two pregnant women: Ablation without fluoroscopic exposure

## İki gebe kadında supraventriküler taşikardinin radyofrekans kateter ablasyonu: Floroskopik maruziyet olmaksızın ablasyon

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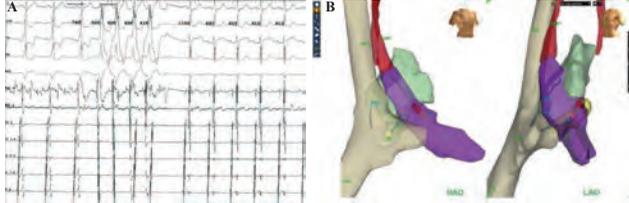
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Radiofrequency catheter ablation (RFCA) offers definitive therapy for vast majority of patients with supraventricular tachycardia (SVT). Conventional RFCA requires the use of fluoroscopy for determination of cardiac anatomy and navigation of the catheters. Total fluoroscopy time may be considerably high for some cases. In certain patient groups that fluoroscopy is relatively contraindicated (eg. pregnant, children), electroanatomic mapping systems may be used for catheter navigation. We are presenting our clinical experience in two pregnant women with SVT who had RFCA using only electroanatomic mapping system instead of fluoroscopy.

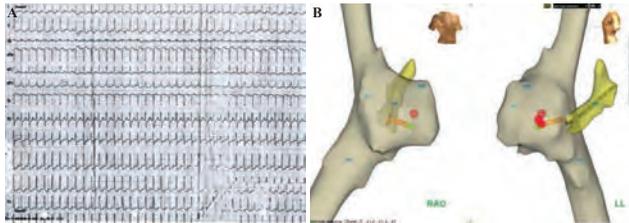
**Case 1:** A 27 years old pregnant woman (21th gestational week) admitted to our outpatient clinic with the complaint of palpitation episodes despite metoprolol therapy. ECG revealed SVT. Resting ECG showed preexcitation compatible with left lateral accessory pathway. Patient was taken to the EP lab in sinus rhythm. Right atrium (RA) and coronary sinus (CS) anatomy was constructed using the EnSite NavX™ (St. Jude Medical, St Paul, MN, USA) electroanatomic mapping system. CS catheter was introduced to the CS thereafter. Foramen ovale was patent. RF catheter was advanced from patent foramen ovale and left atrial (LA) anatomy was constructed. Maximal preexcitation was produced by atrial pacing and mitral annulus (MA) was mapped. RF was administered to the lateral MA region that has the closest AV conduction. However, there was no success. Then, RF catheter was advanced retrogradely to left ventricle under guidance of the En-Site system. Atrioventricular reentrant tachycardia (AVRT) was induced by catheter manipulation. MA was mapped during AVRT. The closest VA conduction was located on lateral MA. RF application here terminated the tachycardia. Eventually, after RFCA, there was no preexcitation, VA conduction was decremental and no AVRT was inducible.

**Case 2:** RFCA was advised for 21 years old pregnant woman (30 th weeks of gestation) who had several SVT attacks despite metoprolol treatment. RA and CS anatomy was constructed by EnSiteNavX system. Tricuspid valve and His region was determined. AH jump and SVT was induced by programmed atrial stimulation. Atrioventricular nodal reentrant tachycardia was diagnosed using electrophysiologic maneuvers. RF catheter was advanced to the slow pathway region and RF was applied. Junctional beats were observed during RF applications. After RFCA, programmed atrial stimulation was completely normal (no jump and no echo beat) and no SVT was inducible.

In conclusion, successful SVT ablation without fluoroscopic guidance is possible by using the En-Site system in pregnant women. Zero fluoroscopic exposure was utilized during RFCA in these patients. To the best of our knowledge, our patients are the first reported adult cases in our country that had successful RFCA using only the En-site system.



**Figure 1.** (A) Disappearance of preexcitation during RFA. (B) Complete electroanatomic mapping of the heart from right anterior oblique (RAO) and left anterior oblique (LAO) views. AORT:ascending aorta, CS: coronary sinus, HIS: His bundle, IVC: inferior vena cava, LA: left atrium, LV: left ventricle, PFO: patent foramen ovale, RA: right atrium, RF: RF catheter on lateral mitral anulus, SVC: superior vena cava.



**Figure 2.** (A) Supraventricular tachycardia episode. (B) Electroanatomic mapping of right atrium and coronary sinus from right anterior oblique (RAO) and left lateral (LL) views. Red points represent sites of ablation. CS: coronary sinus, HIS: His bundle, IVC: inferior vena cava, RA: right atrium.

## OS-14

## Comparison of microvolt T wave alternans and electrophysiologic testing for arrhythmic risk stratification in patients with ischemic or nonischemic dilated cardiomyopathy

## İskemik veya non-iskemik dilate kardiyomyopati hastalarda aritmik risk sınıflaması için mikrovolt T dalga alternansı ve elektrofizyolojik testin karşılaştırılması

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**Objectives:** The aim of this study was to compare Microvolt T-wave alternans (MTWA) and electrophysiologic study (EPS) for arrhythmic risk stratification in the clinical CRT-Pacemaker (CRT-P) or CRT-Defibrillator (CRT-D) implantation in patients with ischemic or nonischemic dilated cardiomyopathy.

**Background:** T wave alternans has been proposed as a powerful tool for identification of patients at high risk for ventricular arrhythmias and sudden cardiac death in ischemic or nonischemic dilated cardiomyopathy.

**Methods:** The study population consisted of 30 patients with dilated cardiomyopathy (EF<= 35%) and LV dyssynchrony confirmed by 3-D transthoracic echocardiography. Patients with heart failure symptoms (NYHA II-IV) who were referred for implantation of CRT-P or CRT-D were included in the study. MTWA test was performed using Cambridge Heart II, 2005-5013 system. EPS was performed in all patients by standard techniques. Patients were treated according to the EPS result.

**Results:** MTWA test was found positive in 15 patients, indeterminate in 4 and negative in 11 patients. EPS was positive in 17 and negative in 13 patients. Moreover EPS was also positive in patients with positive MTWA results and negative in patients with negative MTWA results. Two of the 4 patients with indeterminate MTWA results were further identified as normal by EPS.

**Conclusion:** This study shows that MTWA was successful in identifying arrhythmic risk and comparable to electrophysiologic testing. EPS was especially useful in discriminating indeterminate MTWA results. In conclusion, we may suggest that patients with a negative MTWA test could therefore receive only CRT-P.

## Elektrofizyoloji-ablasyon / Electrophysiology-ablation

## OS-15

## Pulmonary vein isolation using the novel cryoballoon: a comparison of first and second generation

## Yeni kriyobalon kullanarak pulmoner ven izolasyonu: Birinci ve ikinci jenerasyonun karşılaştırılması

Bülent Köktürk, Alexander Yang, Päsler Marcus, Bansmann P.m., Hoppe Christian, Horlitz Marc

Krankenhaus Porz am Rhein, Köln

**Background:** Cryoballoon ablation is effective in pulmonary vein isolation. A novel redesigned second generation Cryoballoon (Arctic front Advance©) was compared to the original first generation CryoAblation catheter (Arctic front©).

**Methods-Results:** Cryoballoon ablation of patients with atrial fibrillation (AF) was performed by one transeptal approach. The primary end point was a complete pulmonary vein isolation confirmed by a so called Achieve-Catheter© and secondary endpoints included complications, AF recurrences, procedural dates and lesion formation using biomarkers and navigator gated, free breathing, delayed enhancement cardiac MRI (DE-MRI). One day after the procedure blood samples including blood concentration of cardiac troponin I (cTnI), creatinine kinase (CK) and myocardial bound for CK (CK-MB) were obtained. Follow-up with 7-day Holter monitoring was performed after 3 months and are scheduled for 6 and 12 months after the procedure. 63 consecutive patients (Arctic front © group/group 1: 32 patients; 23 males, age 60 (44 to 79) years, paroxysmal AF: n=26, persistent AF: n=6 / Arctic front Advance © group/group 2: 31 patients; 17 males, age 62 (35 to 77) years, paroxysmal AF: n=26, persistent AF: n=5) underwent cryoablation. The primary endpoint was achieved in all patients. No major complication occurred in neither of the groups. Median procedure duration was 135 (115-215) minutes in the first generation group compared to 134 (80-160) minutes in the second generation group. The median energy application time was in Arctic front group © with 45 (21 to 80) minutes longer than in the Arctic front Advance © group, in which 33 (20 to 44) minutes energy application in median was necessary to isolate all pulmonary veins. However, the lesion formation in the Arctic front Advance © group seems to be greater as in the Arctic front © group shown by the significant higher cTnI level [group 2:6,3 (1,6 to 14,8; group 1: 3,5 (0,2-13,1); p=0,036] and greater necrosis formation in DE-MRI in group 2. After a follow-up time of 83 (6 to 185) days 25 patients (78,1%) in group 1 and 28 patients (90,3%) in group 2 were free of AF recurrence.

**Conclusion:** The novel Cryoballoon (Arctic front Advance ©) is as safe and effective as the old one. However, it seems to perform greater lesion formations which is reflected by a higher cTnI level and can be visualized by DE-MRI although less energy application time is necessary for a complete pulmonary vein isolation. This is probably associated with a better clinical outcome.

OS-16

## Thrombus and left atrial spontaneous echo contrast formation during percutaneous mitral valve repair with the MitraClip system of severe mitral regurgitation: the first cases in the literature

### İleri mitral yetersizliği için MitraClip sistemle perkütan mitral kapak tamiri sırasında trombüs ve sol atriyal spontan ekokontrast oluşumu: Literatürdeki ilk vaka

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**Introduction:** Mitral valve repair with the MitraClip is a new promising therapeutic option for symptomatic severe mitral regurgitation (MR). In spite of its beneficial effect, theoretically, Mitraclip may have an acute, harmful effects especially in severe MR patients with atrial fibrillation (AF). However, to the best of our knowledge, left atrial spontaneous echo contrast (LASEC) and thrombus formation during repair with the MitraClip of severe MR has not been well documented in the literature. Here we present, two unique cases, a thrombus formation on the septal puncture site, and LASEC formation during the procedure.

**Case:** The first case was a 75-year-old man with a left ventricular function of 25%. His medical history included AF and severe MR. Using fluoroscopic and transesophageal echocardiography (TEE) guidance, the MitraClip device was directed towards the origin of the regurgitant jet. The clip was retracted until both leaflets are grasped and then closed to coapt the mitral leaflets. In the end of the procedure, TEE demonstrated a significant reduction of MR grade from IV to trace residual MR. However, when the leaflets were grasped, marked LASEC was observed during TEE (Figure 1). LASEC was clearly absent immediately before grasping the leaflets in this case (Figure 2). He received warfarin after the procedure. At one month follow up, he was clinically stable and had a mild degree of MR. The second patient was a 43-year-old man with a left ventricular function of 15%. His medical history included paroxysmal AF and severe MR. During the procedure, the patient developed AF but recovered in the end of the procedure. After the procedure, TEE demonstrated a significant reduction of MR grade from IV to I residual MR. Immediately after the guide catheter removal from the interatrial septum, TEE demonstrated a mobile thrombus seemed to be attached to the interatrial septum at the septal puncture site and mild LASEC (Figure 3). The patient was managed with anticoagulation because of the high-risk nature of surgery. The TEE performed on the 5th postoperative day demonstrated no interatrial septal thrombus (Figure 4).

**Discussion:** The mechanism underlying the increase in LASEC after the reduction of MR by Mitraclip procedure in our patients could be the disappearance of marked MR jet agitating blood stasis in left atrial cavity. Another possibility is that the acute increase in left ventricular afterload induced by removing the low-impedance regurgitant flow may have contributed to LASEC formation. In our second case, most probably Mitraclip procedure led to interatrial thrombus formation by the disappearance of severe MR jet agitating blood stasis. However, endocardial damage during septal puncture and the duration of the Mitraclip procedure may have contributed to a hypercoagulable state. This report shows that thrombus and SEC formation in the LA may occur during percutaneous mitral valve repair with the MitraClip system of severe MR.



Figure 1. TEE shows marked left atrial spontaneous echo contrast when the leaflets were grasped.

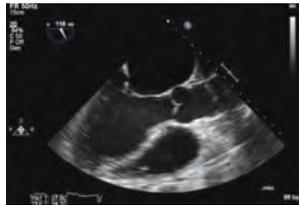


Figure 2. TEE shows no LASEC clearly immediately before grasping the leaflets.

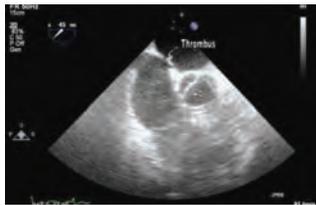


Figure 3. TEE demonstrates a thrombus moving back and forth between the left and right atrium and mild left atrial spontaneous echo contrast in the short axis view.



Figure 4. On the 5th postprocedural day, TEE demonstrates resolution of the interatrial septal thrombus.

OS-17

## “Home-made snare” for retrograde CTO intervention with rendezvous technique

### Buluşma tekniği ile retrograd KTO girişimi için “ev yapımı snare”

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**Introduction:** 68 years old Malay gentleman with history of dyslipidemia, chronic smoker and anterior myocardial infarction missed thrombolytic in July 2011. Noted to have 95% Proximal LAD lesion which was stented with Yukon Stent and CTO RCA. He was in CCS class II.

**Description of problem:** Long RCA CTO from mid to distal RCA and patent previous LAD stent. He opted for percutaneous coronary intervention.

**Procedure, technique and equipments used:** PCI to CTO RCA Right and Left femoral arteries punctured and two 6Fr Sheaths inserted. JR4 guide engaged the RCA and XBLAD 3.5 guide engaged the LCA. Simultaneous left and right coronary artery angiogram showed long CTO RCA from prox/mid to distal RCA with collateral to RCA filling-up retrogradely from LAD artery. Septal channel from LAD connecting to PDA artery identified as a target for retrograde intervention. v) “Home-made” Snare Using 20mm bent of the soft end of Sion blue wire with Minitrek 2.0\*15 balloon (both already used earlier) dilated at 10atm at distal JR4 guide. This entrapped the distal 20mm on Sion blue wire; with gentle push of the wire it creates a wide-loop outside the JR4 guide to be used as a snare (I called it “Home-made” snare). The retrograde Fielder FC wire successfully snared into JR4 guide. vi) Rendezvous technique (Kissing microcatheter) Antegradely Finecross microcatheter advanced into JR4 guide. The retrograde Fielder FC wire then successfully entered into antegrade Finecross. Both antegrade and retrograde microcatheter meet at proximal RCA; with antegrade Finecross being advanced into RCA with simultaneous retrograde Finecross being pulled backward slowly into mid to distal RCA. However the JR4 guide unfortunately was very unstable despite being anchored with anchor balloon Sapphire 1.5\*15 at Conus branch. The guide was very unstable and came out. vii) Wire Externalization Subsequently AL1 6F Guide engaged RCA and the retrograde Fielder FC re-snared with “Home-made” snare. Fielder FC wire was trapped with Minitrek 2.0\*15 and retrograde Finecross advanced into AL1 guide. RG3 330mm wire externalized via RFA distal end of AL1 guide. viii) Ballooning and Stenting Antegradely Antegradely via RG3 externalized wire Minitrek 2.0\*20mm advanced along the CTO lesions and predilated sequentially from 10 to 14 atm. Then the long CTO lesions stented from PDA to mid/proximal RCA with 3 overlapping stents: Biomatrix 2.5\*28 (14atm), Biomatrix 2.75\*36 (18atm) and Biomatrix 3.0\*36 (20atm). The final angiogram showed a very good result with a brisk TIMI 3 flow.

**Results:** During post procedure 8 months follow up, he was asymptomatic with functional class I. He was advised for a long term dual antiplatelet therapy; with a minimal of 1 year. “Home-made” Snare for retrograde CTO intervention is an easy technique to learn, to apply clinically and it is safe at the same time with the benefit of cutting the cost of snare.

OS-18

## Successful renal denervation of a patient with ineligible renal artery anatomy by anchoring technique: an alternative attempt

### Uygun olmayan renal arter anatomisi olan bir hastada çapalama tekniği ile başarılı renal denervasyon: Alternatif girişim

Sinan Dagdelen, Ali Buturak, Yasemin Demirci, Aleks Degirmencioglu

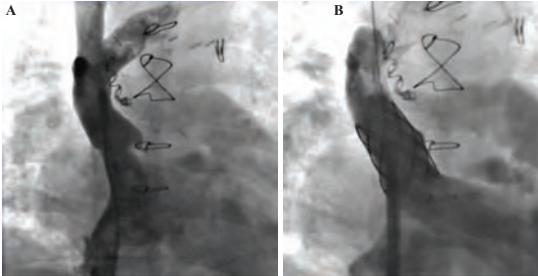
Acibadem University School of Medicine Department of Cardiology, Istanbul

Percutaneous transcatheter renal sympathetic denervation is a partly new and promising treatment for refractory hypertension. A 54-year-old male with more than 10-years history of resistant hypertension was referred to cardiology outpatient department. Despite the lifestyle modifications and pharmacological therapy with five different anti-hypertensives, the patient's systolic and diastolic arterial blood pressures were uncontrolled. Renal denervation was planned after renal angiography. Left renal artery was successfully ablated. After right renal artery cannulation with a 6Fr IMA guiding catheter (45 cm, Cordis, Johnson & Johnson, USA), the Symplcity™ radiofrequency catheter (Medtronic Inc., Minneapolis, MN, USA) was tried to be advanced through the lumen of the guiding catheter, but it failed. Renal guide catheter was coming away from the ostium and falling back to the aorta lumen as the ablation catheter was touching the renal arterial wall (Video 1). Guide catheter was exchanged with a 6 Fr RDC-1 renal guide catheter (55 cm, Cordis, Johnson & Johnson, Warren, NJ, USA) but this attempt also did not work out. Afterwards, we decided to advance a soft 0.014 inch guidewire (180 cm, Asahi Inteco Co, Thailand) and keep it inside the lumen of right renal artery during renal ablation procedure. Ablation delivery catheter could be advanced to distal right renal artery after guide catheter was stabilized and anchored via keeping the soft 0.014 inch guidewire inside the lumen (Video 2). We meticulously kept the tip of the ablation catheter away from the guidewire during the right renal artery denervation to prevent ineffective ablation. Right renal artery was successfully ablated following the guiding catheter stabilization (Video 3). The patient was discharged with a medication of amlodipine and bisoprolol 10 mg once daily two days after the intervention. Catheter based renal denervation is a safe and effective procedure which improves resistant hypertension with great and persistent decreases in systolic and diastolic blood pressures. This novel treatment option is usually performed quickly and safely in patients with eligible renal and aorta anatomies. In cases of high acute renal artery take off, short renal artery trunk or S shaped kinking of the aorta and iliac arteries, the renal denervation procedure may be challenging. The patient presented here, had a severely angulated and double curved right renal artery ostium which complicated the guiding support and advancement of the delivery ablation catheter. Anchoring the guiding catheter technique may be used as an alternative attempt in cases of ineligible renal artery anatomy impeding the delivery catheter advancement and guiding support in renal denervation procedures.

## OS-19

**Successful stenting of systemic venous pathway stenosis after double switch repair for congenitally corrected transposition of great arteries in children: a case report****Çocukta konjenital düzeltilmiş büyük arter transpozisyonu için "double switch" operasyonu sonrası sistemik venöz yol stenozunun başarılı stentlenmesi**Arda Saygılı<sup>1</sup>, Ahmet Arnaz<sup>2</sup>, Yusuf Yalçınbaş<sup>2</sup>, Tayyar Sarıoğlu<sup>3</sup><sup>1</sup>Acıbadem Hospital, Pediatric Cardiology, İstanbul<sup>2</sup>Acıbadem Bakırköy Hospital Cardiac Surgery, İstanbul<sup>3</sup>Acıbadem University, Cardiac Surgery, İstanbul

A 8 year old boy with previous shunt operation for corrected transposition of great arteries, ventricular septal defect, pulmonary stenosis and multiple aorto-pulmonary collaterals artery had undergone corrective surgery. In early postoperative journey there is a clinics of vena cava superior obstruction. Cardiac catheterisation 72 hours after the surgery showed that he had systemic venous baffle stenose: between the caval vein and right atrium. Stent was successfully implanted percutaneously. The stenose was relieved immediately. Her symptoms quickly disappeared and extubated rapidly. Follow-up show excellent maintenance of patency.

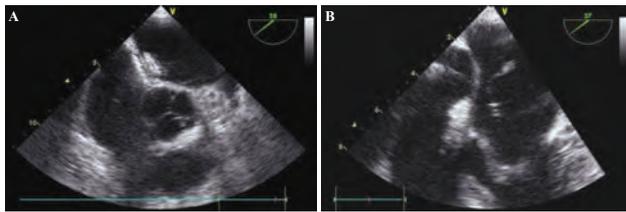


**Figure 1.** (A) Angiocardiogram of the patient with superior vena cava (SVC) and systemic venous baffle obstruction after the Senning procedure. (B) Angiocardiogram of the patient after the stent procedure.

## OS-20

**Successful lysis of a mobile left atrial disk thrombus on an amplatzer atrial septal defect occluder by the slow infusion of a low-dose tissue plasminogen activator****Amplatzer atriyal septal defekt tkayıcısının sol atriyal diski üzerindeki hareketli trombüsün düşük doz doku plazminojen aktivatörü yavaş infüzyonu ile başarılı lizisi**Arda Saygılı<sup>1</sup>, Selçuk Görmez<sup>2</sup>, Erkan Ekicibaşı<sup>2</sup>, Yasemin Demirci<sup>3</sup><sup>1</sup>Acıbadem Hospital, Pediatric Cardiology, İstanbul<sup>2</sup>Acıbadem Hospital, Cardiology, İstanbul<sup>3</sup>Acıbadem University, Cardiology, İstanbul

A 46-year-old man was admitted to our hospital for suspicion of transient ischemic attack with a subacute left retinal arterial thrombosis. The patient was anticoagulated with warfarin and a diagnosis of PFO was made. Closure were attempted with Amplatzer atrial septal occluder. Follow-up transesophageal echocardiography control demonstrated a mobile thrombus on the left side of the Amplatzer atrial septal occluder after device release. The thrombus was successfully treated with an infusion of heparin, and 25 mg recombinant tissue-type-plasminogen activator was slowly infused over a 24-hour period, resulting in complete lysis after 24 hours. The patient was asymptomatic and without a detectable thrombus at follow-up.



**Figure 1.** (A) Transesophageal echocardiography shows the Amplatzer septal occluder. (B) TOE shows thrombus formation on the left atrial edge of the Amplatzer septal occluder.



**Figure 2.** TOE shows no thrombus on the device after unfractionated heparin and rt-PA were infused.

## OS-21

**Digital artery thrombo-embolism after coronary angiography: complication or coincidence?****Koroner anjiyografi sonrası dijital arter tromboembolisi: Komplikasyon ya da rastlantı?**Sedat Koroğlu<sup>1</sup>, Erdiç Eroğlu<sup>2</sup>, Hüseyin Nacar<sup>3</sup><sup>1</sup>Afsin State Hospital, Cardiology Department, Kahramanmaraş<sup>2</sup>Sutcu Imam University Faculty of Medicine, Cardiovascular Surgery Department, Kahramanmaraş<sup>3</sup>Sutcu Imam University, Faculty of Medicine, Cardiology Department, Kahramanmaraş

A 42 years old man admitted to outpatient cardiology clinic with pain in his right index finger. In his past medical history a diagnostic coronary angiography via right femoral artery for typical angina pectoris was carried out one week ago. There were non-critical lesions on all coronaries and a medical therapy including acetylsalicylic acid 100 mg, nebivolol 5 mg and atorvastatin 10 mg was given to him. On physical examination the index finger had a purple color and it was cold (Figure 1A). On colored Doppler ultrasonography of right hand there was thrombus on right index digital artery. He was hospitalized and heparin infusion was started immediately. Then, totally 1.500.000 units of streptokinase with a regimen of 250.000 units in 30 minutes and the remaining dose 100.000 units/hour was administered. At the end of the therapy the ischemic symptoms and findings of index finger was resolved (Figure 1B). The patient was discharged with acetylsalicylic acid, cilostazol and low molecular weight heparin. There was no similar report in the current literature. Our diagnosis was thromboembolism and we excluded cholesterol embolization because the ischemia was resolved after thrombolytic therapy. We need more cases and scientific evidences concerning that this is a complication of coronary angiography.



**Figure 1**

## OS-22

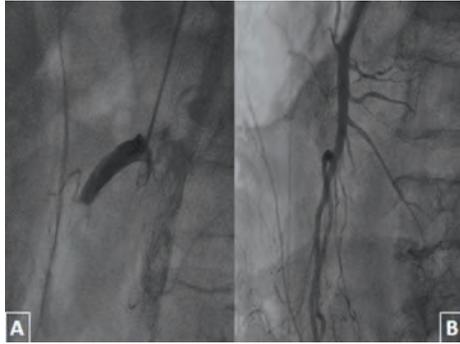
**Successful recanalization of acute superior mesenteric artery ischemia with balloon angioplasty and aspiration embolectomy****Akut süperior mezenterik arter iskemisinin balon anjiyoplasti ve aspirasyon embolektomi ile başarılı rekanalizasyonu**

Ercan Erdoğan<sup>1</sup>, Mehmet Akkaya<sup>1</sup>, Ahmet Bacaksız<sup>2</sup>, Abdurrahman Tasa<sup>1</sup>, Murat Turfan<sup>1</sup>, Osman Sönmez<sup>1</sup>, Musa Sahin<sup>2</sup>, Seref Kul<sup>1</sup>, Emin Asoglu<sup>1</sup>, Emrah Sevgili<sup>1</sup>, Mehmet Akif Vatankulu<sup>1</sup>, Mehmet Ergelen<sup>1</sup>, Ömer Göktekin<sup>1</sup>

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<sup>2</sup>Yüzüncü Yıl University, Faculty of Medicine, Cardiology, Van

Acute mesenteric ischemia (AMI) is a life threatening vascular emergency that is described sudden reduction of intestinal perfusion lead to bowel ischemia and infarct. Contemporary results report mortality ranges from 60% to 90%. Superior mesenteric artery (SMA) embolism is the most common (40-50%) cause of AMI. Early diagnosis is essential as delay in diagnosis can result in bowel infarction and irreversible damage. Therapeutic options include pharmacologic, endovascular treatment and surgery. A 71-year-old male admitted to the emergency department for acute worsening of pre-existing abdominal peri-umbilical pain, nausea, and vomiting. His past medical history was arterial hypertension and paroxysmal atrial fibrillation (AF). Transthoracic echocardiography showed a non-obstructive hypertrophic cardiomyopathy with 61% ejection fraction and no evidence of thrombi in the left atrium. All his vital signs were within normal limits. Also, we did not detect any sign of peritonitis with physical examination. Emergent computed tomography (CT) angiography showed complete occlusion of the proximal portion of the main stem of SMA due to thromboembolism. Conventional angiography of the aorta and the SMA was carried out using a left brachial access. This confirmed an occlusion of the proximal SMA (Figure 1A). Initially, a hydrophilic 0.035-inch guide wire is passed deep into the SMA and we performed balloon angioplasty (4.0×40×135 Fox Cross balloon) after IV infusion of 5000 IU heparin. A 8F guiding catheter is placed in the proximal SMA embolism. Aspiration was applied manually to the sheath simultaneously with sheath withdrawal, thus achieving aspiration of the clot (Figure 2). Angiography after repetitive aspiration showed nearly complete thrombi removal from the SMA main trunk. Final angiogram showed completely restored perfusion to the ischemic bowel through the patent main stem of SMA (Figure 1B). The patient got complete pain relief after the procedure. The most common source of an embolus is cardiac usually secondary to AF. Less common causes are mural thrombus following an acute myocardial infarction, paradoxical emboli through septal defects, cardiomyopathies, valvular diseases, endocarditis, and atrial myxoma. Severe abdominal pain is the cardinal symptoms of AMI. Plain films are not helpful in most cases. However they are useful to exclude other causes of acute abdomen like perforation and obstruction. CT is often used for diagnosis. Contrast-enhanced CT can show occlusion of SMA, frequently accompanied with findings suggestive of bowel ischemia, including pneumatosis intestinalis, bowel wall thickening, ileus and bowel dilatation. Angiography can define the location and origin of the arterial occlusion and provide the potential for intervention if mesenteric ischemia is diagnosed prior to ischemic bowel necrosis. Endovascular treatment of acute SMA occlusion provides a good alternative to open surgery.



**Figure 1.** Successful percutaneous treatment of thromboembolic occlusion on proximal trunk of superior mesenteric artery (SMA) with balloon angioplasty and aspiration embolectomy. A: SMA angiography showing a complete thrombotic occlusion of the proximal portion of the main stem of SMA due to a large amount of thromboembolism; B: Completely recanalized main stem of SMA after repetitive aspiration embolectomy.



**Figure 2.** Expelled embolic material.

## OS-23

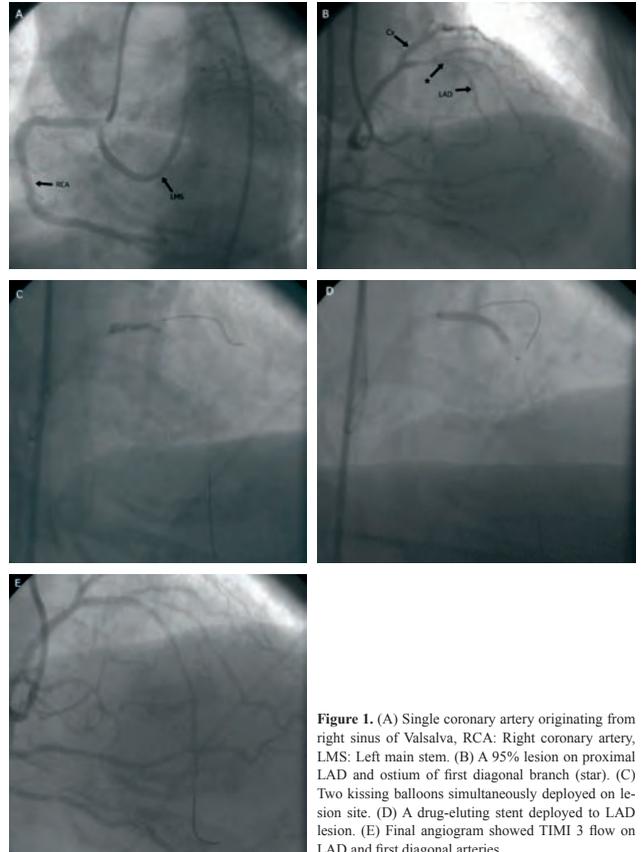
**Single coronary artery from the right sinus of valsalva and primary percutaneous intervention for bifurcation lesion on anomalous artery****Sağ sinüs valsalyadan kaynaklanan tek koroner arter ve anormal arterdeki bifurkasyon lezyonu için primer perkütan girişim**

Yılmaz Ömür Otlu<sup>1</sup>, Adil Bayramoğlu<sup>1</sup>, Mehmet Sait Altıntaş<sup>2</sup>, Şiho Hidayet<sup>1</sup>, Ramazan Özdemir<sup>1</sup>

<sup>1</sup>Department of Cardiology, Inonu University Faculty of Medicine, Malatya

<sup>2</sup>Department of Cardiology, Karaman State Hospital, Karaman

A 72 year-old female patient was admitted to our emergency unit with chest pain which had begun 2 hours before admission. She was diagnosed with diabetes mellitus about ten years ago and she had no history of previous coronary artery disease. Examination revealed blood pressure of 135/85 mmHg, pulse of 80 bpm. Heart and respiratory auscultation findings were normal. Electrocardiography revealed sinus rhythm and 3-mm ST segment elevation in leads V2-6. Under the diagnosis of acute anterior myocardial infarction, the patient was immediately sent to the coronary angiography laboratory. The left main coronary artery could not cannulated at left sinus of Valsalva. Coronary angiography revealed a single coronary artery originating from right sinus of Valsalva; a short main stem bifurcated right coronary artery and left main stem that finally divided left anterior descending and circumflex arteries. There was 95% lesion on proximal LAD and ostium of first diagonal branch, and TIMI flow was 1. Two separate 0.014 guidewires (Champion, SP Medical, Denmark) inserted to anomalous LAD and diagonal branches and two 2.0x20 mm old plain balloons (Sprinter Legend, Medtronic, USA) advanced over the guidewires. Both balloons were introduced in the bifurcation and simultaneously deployed. Then a 2,75x30 mm zotarolimus eluting stent (Endeavor, Medtronic) deployed to LAD lesion. Final angiogram showed TIMI 3 flow on LAD and first diagonal arteries. Contrast-induced nephropathy developed two days after procedure, so that multislice cardiac tomography could not be examined. She was discharged 7 days after the procedure and has been uneventful for 6 months of the follow-up. To our knowledge, there have only two reported PCI for bifurcation lesions on anomalous coronary arteries in the literature (Stevens et al 2008 and Das et al 2006). Both reports were introducing elective PCI procedures. Our case was different from both cases because primary PCI applied for a bifurcation lesion on an anomalous coronary. The selective cannulation of anomalous artery can be time consuming, and importance of this situation increases in acute conditions. In conclusion, the possibility of an anomalous coronary artery should kept in mind to reduce time loss during primary PCI in the "absence" of suspected artery.



**Figure 1.** (A) Single coronary artery originating from right sinus of Valsalva, RCA: Right coronary artery, LMS: Left main stem. (B) A 95% lesion on proximal LAD and ostium of first diagonal branch (star). (C) Two kissing balloons simultaneously deployed on lesion site. (D) A drug-eluting stent deployed to LAD lesion. (E) Final angiogram showed TIMI 3 flow on LAD and first diagonal arteries.

OS-24

### Relationship between dietary patterns and hypertension: Khorramabad study

#### Beslenme biçimi ve hipertansiyon arasındaki ilişki: Khorramabad çalışması

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**Introduction:** Hypertension is a common public health problem in developed and developing countries. In Iran one in four adults has high blood pressure approximately. Untreated hypertension leads to many degenerative diseases, including heart failure, end stage renal disease, and peripheral vascular disease. It is often called a “silent killer” because people with hypertension can be asymptomatic for years and then have a fatal stroke or heart attack. Many factors can be effective on high blood pressure incidence one of these predisposing factors could be belongs to dietary pattern. The aim of this study is to determine the relationship between hypertension and Dietary patterns among Iranian adults.

**Material-Methods:** In this cross-sectional study, 973 participants (237 males and 736 females) involved which were Khorramabad's residences and were selected with multistage cluster and randomized systematic sampling. Food intake assessments were measured by a validated, 201 food items, self-administrated, semi quantitative food frequency questionnaire. Blood pressure was gained by a nursing expert, twice after the participants sat for 15 min. Dietary patterns were identified by factor analyzes. Hypertension was identified as systolic blood pressure (SBP) higher than 130 ml/Hg and/or diastolic blood pressure (DBP) higher than 85 ml/Hg.

**Results:** Three major dietary patterns that were identified by factor analyzes were traditional dietary pattern (TDP), Healthy dietary pattern (HDP), and Western dietary pattern (WDP). The prevalence of hypertension in this study was 28.5%. There weren't any significant relationship between all dietary patterns with Mean±SD of blood pressure except HDP with DBP (P-value 0.007). But the risk of hypertension was significantly correlated with WDP and HDP (P-value 0.004 and 0.043 respectively) and the overall prediction power of model were 71.7% and 75.6% respectively. Whereas the overall prediction power of model for TDP was 75.3% and there weren't any significant relation between TDP and risk of hypertension.

**Debates:** This study's results showed that WDP could increase the risk of hypertension whereas HDP could decrease this risk. But TDP couldn't make any changes about this conspectus. But the cross-sectional studies have some limitations so for showing the exact association between dietary patterns and hypertension needs some follow up studies in near future.

OS-25

### Deep neck infection induced Takotsubo cardiomyopathy and accompanying idiopathic hypereosinophilic syndrome with Loeffler endocarditis: a case report

#### Derin boyun enfeksiyonunun tetiklediği Takotsubo kardiyomyopati ve eşlik eden Loeffler endokarditi ile idiyopatik hipereozinofilik sendrom: Olgu sunumu

Harun Kundi, Mustafa Çetin, Emrullah Kızıltunç, Müslüm Şahin, Zehra Güven Çetin, Hülya Çiçekçiöglü, Ender Örneker, Feridun Vasfi Ulusoy

Ankara Numune Education and Research Hospital, Department of Cardiology, Ankara

Takotsubo cardiomyopathy is a syndrome characterized by transient left ventricular apical or mid-apical ballooning without significant coronary artery stenosis. It is predominantly seen in postmenopausal women with emotional stress and clinically mimics acute coronary syndrome. Idiopathic hypereosinophilic syndrome (IHS) is described as eosinophilic infiltration of various organs, especially heart without presence of eosiniphila reasons like parasitic infections or allergies. A 72 year old female patient followed by otorhinolaryngology clinic with deep neck infection was consulted to our cardiology department with typical sudden chest pain. She had elevated cardiac markers on her blood work and on electrocardiography new onset ST segment elevations at anterior derivations was present. The patient referred to coronary angiography, no significant coronary artery stenosis has shown. On left ventriculography she had apical ballooning (figure 1) so that the patient thought to be Takotsubo cardiomyopathy induced by deep neck infection. On her transthoracic echocardiography (TTE), left ventricular apex was aneurysmatic and ejection fraction (EF) was 40-45%. The patient discharged with her medical therapy arranged. On her second month outpatient clinical control TTE revealed endocardial thickening and fibrothrombotic obliteration of the right ventricle and a large immobile thrombus in the right atrium with normal ranged EF (figure 2). On colour Doppler moderate tricuspid regurgitation was seen. The patient was hospitalized again and low molecular weight heparin therapy was started. Low extremity venous doppler ultrasonography and pulmonary computerized tomography angiography has shown no other evidence of thrombus. The patient also had 65% eosinophilia (11.500/ $\mu$ l) rate on her complete blood count. After exclusion of all other hypereosiniphila reasons she had diagnosed IHS with Loeffler endocarditis and started steroid therapy. On 24th hour of steroids her eosiniphila rate decreased to 0,2%. Warfarin was started with the intention to keep International Normalized Ratio 2-3 on her follow up. In this case, first interesting feature is Takotsubo cardiomyopathy was progressed after deep neck infection. The second feature is after diagnosis of Takotsubo cardiomyopathy very short timely new developing IHS with Loeffler endocarditis was presented in the same patient. On our review of literature deep neck infection induced Takotsubo cardiomyopathy or concomittance of Takotsubo cardiomyopathy-IHS with Loeffler endocarditis has not been reported in same patient before.

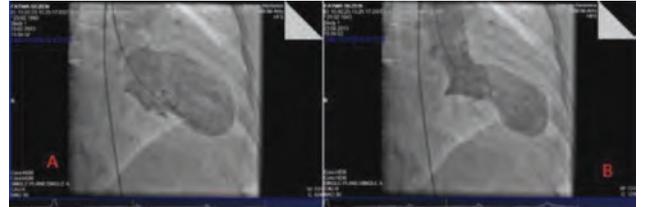


Figure 1. End-diastolic (A) and end-systolic (B) left ventriculograms show apical akinesis and basal hyperkinesis.

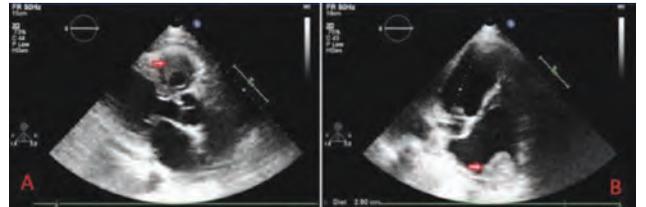


Figure 2. Two-dimensional echocardiogram shows endocardial thickening and fibrothrombotic obliteration of the right ventricle (A) and a large immobile thrombus in the right atrium (B).

## OS-26

### Multiple congenital left ventricular diverticula associated with stroke İnmeye neden olan çok sayıda konjenital sol ventrikül divertikülü

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Congenital ventricular diverticula, is usually the outward pouch formation of the ventricle (usually the left ventricle). An isolated congenital left ventricular diverticulum is a rare, usually asymptomatic cardiac abnormality which can cause major complications such as; systemic thromboembolism, infective endocarditis, cardiac rupture, heart failure, arrhythmia and sudden death. Large diverticula can be diagnosed with 2-dimensional echocardiography, but it has limited use with small diverticula. In this case diagnosis can be established by transesophageal echocardiography, left ventriculography or magnetic resonance imaging. We report a 33 year-old female patient, who admitted with sudden onset right-sided hemiplegia and dysarthria, who had multiple congenital left ventricular diverticula which was detected by echocardiography.

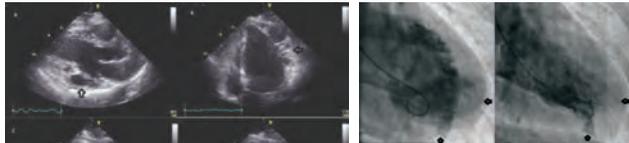
**Case:** A 33 year-old female patient was referred to our clinic for the search of etiology of ischaemic stroke. There was no history of any heart disease. On physical examination, her blood pressure was 120/70 mmHg and heart rate was 86 bpm. Cardiac examination was normal. The 12-lead electrocardiogram showed sinus rhythm of 86 beats per minute, T wave abnormality in leads V3-4 and incomplete left bundle branch block. Transthoracic echocardiography (TTE) showed; prominent trabecular meshwork and deep trabecular sinusoids in the posterolateral wall consistent with diverticulum (Figure 1). The coronary angiography was performed to exclude the possibility of a coronary artery disease. Epicardial arteries were normal. Left ventriculography revealed multiple contractile diverticulum that originated from the lateral wall and left ventricle apex (Figure 2). Cardiac magnetic resonance imaging (MRI) was performed. Diverticula were detected on the lateral wall of the left ventricle with a dimension of 7.0\*2.5 cm and 1.2\*1.0 cm on the apex (Figure 3). The case was presented to the cardiology- cardiovascular surgery council with the diagnosis of multiple congenital left ventricular diverticula. Follow up with medical therapy was recommended and warfarin, metoprolol and ramipril were initiated. Outpatient follow-up was recommended and at the end of one-year follow-up no cardiac symptoms or systemic embolism was discovered.

**Conclusion:** With this case we want to emphasize that detailed and careful transthoracic echocardiography should be performed in patients who attend with systemic embolism, keeping in mind that the etiology can be left ventricular diverticulum.

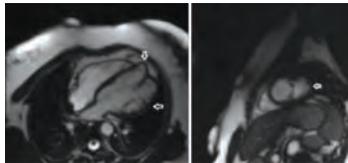
Konjenital ventrikül divertikül, ventrikülün (genellikle sol ventrikülün) dışı doğru keseleşmesidir. İzole konjenital sol ventrikül divertikülü nadir görülen bir kardiyak anomali olup genellikle asemptomatik iken; sistemik tromboemboli, endokardit, kardiyak rüptür, kalp yetersizliği, aritmi ve ani kardiyak ölüm gibi ciddi komplikasyonlara neden olabilir. İki-boyutlu ekokardiyografi ile büyük çaplı divertiküllerin tanısı konulabilir iken, küçük divertiküllerde kullanımı sınırlıdır. Bu durumda transözofageal ekokardiyografi, sol ventrikulografi veya manyetik rezonans görüntüleme ile tanı konulabilmektedir. Bu yazıda, ani başlangıçlı sağ hemipleji ve dizartri şikayeti ile başvuran ve ekokardiyografide çok sayıda konjenital sol ventrikül divertikülü saptanan 33 yaşındaki bir kadın hasta sunuldu.

**Olgu:** 33 yaşında kadın hasta iskemik inme etiyolojisi açısından araştırılması için kliniğimize yönlendirildi. Özgeçmişinde bilinen bir kalp hastalığı öyküsü yoktu. Fizik muayenesinde; kan basıncı 120/70 mmHg, kalp hızı 86 atım/dk olarak saptandı. Kardiyak muayenesi normal olarak saptandı. 12 derivasyonlu elektrokardiyografisinde ritim sinus, kalp hızı 86 atım/dk, V3-4 derivasyonlarda T negatifliği ve inkomplet sol dal bloğu mevcuttu. Yapılan transtoraksik ekokardiyografi'sinde (TTE), posterolateral duvarda içerisinde belirgin trabeküler ağ ve derin trabeküler sinüzoidler bulunan divertikül ile uyumlu bulgular saptandı (Resim 1). Koroner arter hastalığını dışlamak için koroner anjiyografi yapıldı. Epikardiyal arterler normal olarak saptandı. Sol ventrikulografide, sol ventrikül apikali ve lateral duvarda kontraktıl divertiküller ile uyumlu görünüm izlendi (Resim 2). Kardiyak manyetik rezonans görüntüleme yapıldı. Sol ventrikül lateral duvarında 7.0\*2.5 cm boyutlarında, apikalde 1.2\*1.0 cm boyutlarında divertiküller saptandı (Resim 3). Olgu, çok sayıda (multipl) konjenital sol ventrikül divertikülü tanısıyla kardiyoloji-kalp damar cerrahi konseyine sunuldu. Medikal tedavi ile izlem önerilen hastaya; warfarin, metoprolol ve ramipril başlandı. Poliklinik takibi önerilen hastanın, bir yıllık takip sonunda herhangi bir kardiyak semptom veya sistemik emboli izlenmedi.

**Sonuç:** Biz bu vakada, sistemik emboli ile başvuran hastalarda etiyolojide sol ventrikül divertikülünde olabileceğini ve bu nedenle bu hastalarda ayrıntılı ve dikkatli bir şekilde transtoraksik ekokardiyografi yapılması gerektiğini vurgulamak istedik.



**Figure 1.** Transthoracic echocardiography. Diverticulum, originating from the posterolateral wall (black arrows) (A): Parasternal long axis (B): Apical 4 chamber (C): Parasternal short axis (D): Prominent trabecular meshwork and deep intertrabecular sinusoids (black arrow), Apical 4 chamber.



**Figure 3.** Cardiac magnetic resonance imaging. Multiple diverticula, originating from the posterolateral wall and apex of the left ventricle (A): Apical 4 chamber view (B): mid-basal short axis view.

## OS-27

### Polymorphisms of the serotonin transporter gene and their relation to personality traits in patients with coronary artery disease Koroner arter hastalığı olan hastalarda serotonin transport genindeki polimorfizmler ve bunun kişilik özellikleri ile ilişkisi

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**Background:** Polymorphisms of the serotonin transporter gene (5-HTT) have been associated with both mental illness and myocardial infarction. Although many studies have analysed the correlations between personality factors and coronary artery disease (CAD), the importance of the personality factors and character traits and the role of the 5-HTT polymorphisms in CAD development and manifestations are still debatable.

**Objective:** To determine whether the 5-HTT gene polymorphisms (5-HTTLPR and STin2 VNTR) are associated with CAD and to check possible association of the 5-HTT gene polymorphisms and personality traits in patients with CAD.

**Methods:** In this case-control study, Of a total 279 male CAD patients, angiographically documented, we genotyped 126 patients of <55 years old and 125 healthy control individuals for DNA polymorphisms in these genes. Personality traits were assessed with the temperament and character inventory (TCI), which are designed to assess the four dimensions of temperament (novelty seeking [NS], harm avoidance [HA], reward dependence [RD] and persistence [P]) and the three dimensions of character (self-directedness [SD], cooperativeness [C] and self-transcendence [ST]). Blood samples were collected from each participant, and the 5-HTT polymorphisms have been investigated by polymerase chain reaction (PCR). The observed genotype frequencies are consistent with Hardy-Weinberg equilibrium.

**Results:** The CAD patients scored higher on HA (p<0.01) and scored lower on NS (p<0.05), SD (p<0.01) and C (p<0.01) than that of the controls. The SS, LS, and LL genotypes were 33.3%, 54.8% and 11.9%, respectively, for cases versus the controls (p<0.05). An association of STin2 VNTR with CAD was not shown. After adjustment for age and personality traits by using multi-variable logistic regression, the odds ratio for CAD associated with the SS genotype was 4.0 (95% CI 1.06-15.04, p<0.05).

**Conclusions:** The SS genotype of the 5-HTT polymorphism, but not the STin2 VNTR, is associated with a higher risk of CAD. This may play an important role in the expression and modulation of complex traits and behavior in patients with CAD or may serve as predictors of therapeutic response.

### A new treatment modality for resistant hypertension: carotid baroreceptor stimulation- the first case from Turkey

#### Dirençli hipertansiyonun tedavisinde yeni bir yöntem: karotid baroreseptör stimülasyonu- ülkemizden ilk olgu

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The prevalence of hypertension appears to be around 30-54% of the general population in Turkey but only one third of the patients are under control. Hypertension is defined as resistant to treatment when a therapeutic strategy that includes appropriate lifestyle measures plus a diuretic and two other antihypertensive drugs belonging to different classes at adequate doses. It is associated with high risk of cardiovascular morbidity and mortality. Carotid baroreceptor stimulation has recently been reported to reduce blood pressure in resistant hypertensive individuals. Here we reported our first case treated with carotid baroreceptor stimulation method in Turkey.

Ülkemizde her üç erişkinden birinde görülen hipertansiyon tedavisinde için pek çok yöntem kullanılmaya rağmen kan basıncı kontrol oranı %30'u geçmemektedir. Bir diüretik olmak üzere en az üç antihipertansif ilaçla kan basıncının >140/90 mmHg olması şeklinde tanımlanan dirençli hipertansiyon, artmış kardiyovasküler morbidite ve mortalite riski ile ilişkilidir. Karotid baroreseptör stimülasyonu, dirençli hipertansiyon tedavisinde kullanılabilecek yeni bir yöntemdir. Burada ülkemizde bu yöntemin uygulandığı ilk olgu sunuldu. 40 yaşında bayan hasta kontrolsüz hipertansiyon ön teşhisiyle kliniğimize müracaat etti. Hastanın her iki koldan ölçülen kan basıncı 200/110 mmHg idi. On beş yıldır hipertansiyon öyküsü olan hastaya yaklaşık bir sene evvela renal denervasyon tedavisi uygulanmıştı. Çoklu antihipertansif tedavi ve renal denervasyona rağmen evre 3 hipertansiyonu olan hastanın kan basıncının kontrol altına alınmasına yönelik karotid baroreseptör stimülasyonu cihazı yerleştirilmesine karar verildi. Kardiyoloji anjiyografi laboratuvarına alınan hastaya genel anestezi altında işleme geçildi. Hastanın boynu, hiperekstansiyonda hafifçe sola çevrilerek pozisyon verildi. Sternokleidomastoid adaleye paralel 4 cm'lik oblik boyun insizyonu ile cilt ve cilt altı dokusu geçildi. Platysma geçilerek sternokleidomastoid adale laterale doğru ekarte edildi. Sağ A. carotis communise (CCA) ulaşıldı, arter dönülerek askıya alındı. Kraniyale doğru CCA takip edilerek karotid bifurkasyonuna ulaşıldı. A. carotis interna (ICA) ve externa ayrı ayrı dönüldü. ICA, bulbustan itibaren 2 cm serbestleştirildi. ICA 2 cm distal posteriordan başlanarak lateral, anterior, anteromedial on ayrı baroreseptör stimülasyonu noktası sırayla karotid baroreseptör stimülasyonu cihazının (Barostim Neo, CVRx, Inc., Minneapolis, USA) elektrodu ile uyarıldı (Figür 1). Uyarılmasıyla sistolik kan basıncında %10 düşüş sağlanan noktalar tespit edilerek işaretlendi. En fazla kan basıncı düşürücü etkinin sağlandığı noktaya (ICA'nın anterior bölümü, 3. bölge) pil elektrodunun tespit edilmesine karar verildi. Tespit öncesi tekrar stimülasyonla kontrol edildikten sonra tek tek 6.0, 11 mm yuvarlak çift iğneli prolen sütür ile saat 5-7-12-3-9 pozisyonlarından adventisyaya tespit edildi. Elektrodun leadi, yaklaşık 3 cm altından CCA'nın ön yüzüne tespit edildi (Figür 2). İncisura jugularis üzerinden 1 cm'lik kesileyle cilt altına inildi. Kelly klemp ile girilerek daha önceden hazırlanmış 10 cm nazogastrik tüp cilt altından geçirildi. İçerisinden lead incisura jugularisten çıkarıldı. 4 cm'lik sağ infraklaviküler transvers insizyonla pectoral adele ve cilt arasında pilin yerleştirileceği cilt altı cebi hazırlandı. Tekrar bu insizyondan incisura jugularise Kelly klemp ilerletilerek nazogastrik tüp ve içerisinden pil leadi pil cebine taşındı. Leadin ucu pile bağlandı. Pil aktive edilip efektif olarak çalıştığı görüldü. Karotid insizyon bölgesine hemovac dren yerleştirildi. Katlar anatomisine uygun olarak kapatıldı.



Figure 1. Stimulation of different sites of internal carotid artery with Barostim Neo device.



Figure 2. Implantation of carotid baroreceptor stimulation device electrode to the internal carotid artery.

### Giant pulmonary artery trunk and subsegmental branches aneurysm with Eisenmenger syndrome; a rare result of secundum type atrial septal defect

#### Eisenmenger sendromu ile dev pulmoner arter gövdesi ve subsegmental dal anevrizması: Sekundum tip atrial septal defektin nadir bir sonucu

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**Introduction:** Eisenmenger syndrome is characterized with pulmonary hypertension, reversal of flow and cyanosis due to congenital cardiac defect with previous left-to-right shunt converted into right-to-left shunt. Atrial septal defect(ASD), total anomalous pulmonary venous return, large ventricular septal defect, large patent ductus arteriosus, truncus arteriosus may cause Eisenmenger syndrome.

**Case Presentation:** A 48-year-old woman presented with long-standing exertional dyspnea, chest pain and cyanosis. She had diagnosis of ASD 9 years ago and operation was suggested, however patient refused surgical or percutaneous treatment. On her initial evaluation at our hospital, the examination findings were as follows: blood pressure was 110/70 mmHg, pulse 116 beats/minute(atrial fibrillation), respiratory rate 26/minute with O2 saturation 58%. Chest auscultation revealed bibasilar diminished respiratory sounds with rhoncus. On cardiac examination, 3/6 systolic murmur at the left sternal border with tachyarrhythmia. Bilateral +++/+++pitting edema with hepatomegaly found during physical examination. Chest x-ray revealed cardiomegaly with mediastinal enlargement (figure 1). An echocardiogram showed marked right atrial and ventricle enlargement with 40 mm ASD and elevated right ventricular systolic pressure at 80 mmHg (figure 2). Parasternal short axis view demonstrated giant main pulmonary artery with enlarged right and left pulmonary artery. The main pulmonary artery was 82 mm, left pulmonary artery 46 mm and right pulmonary artery 40 mm (figure 3). Patient treated with vasodilator agents and intravenous furosemide, oral spironolactone and hydrochlorothiazide. Thorax computerized confirmed giant pulmonary artery trunk including enlargement of subsegmental branches (figure 4-7). Patient was treated symptomatically and discharged from hospital.

**Discussion:** Pulmonary arterial trunk aneurysm has been a very rare entity. A giant pulmonary arterial trunk aneurysm due to Eisenmenger syndrome has seldom been reported. Rare case reports indicate pulmonary artery dissection in young patients with Eisenmenger syndrome had been treated with heart-lung transplantation. Medical treatment including endothelin receptor antagonists, phosphodiesterase type-5 inhibitors, prostacyclin, and prostacyclin analogs have proven beneficial in patients with Eisenmenger syndrome. In our case we preferred medical treatment because of high risk in surgery. The case we reported demonstrated giant pulmonary artery trunk aneurysm including subsegmental branches due to ASD. Similar cases were seldom reported in the literature.



Figure 1. Chest x-ray.

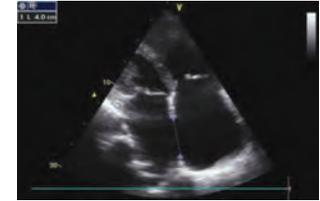


Figure 2. Transthoracic echocardiogram demonstrating atrial septal defect.



Figure 3. Parasternal short axis view demonstrating giant pulmonary artery trunk aneurysm.

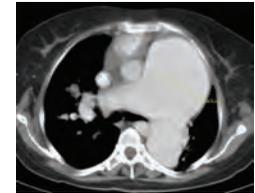


Figure 4. Computerized tomography image with giant main pulmonary artery aneurysm.

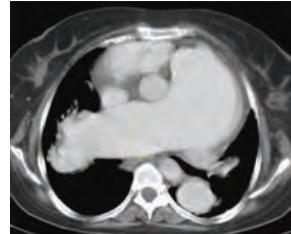


Figure 5. Computerized tomography demonstrating right pulmonary artery aneurysm.

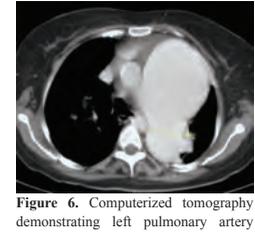


Figure 6. Computerized tomography demonstrating left pulmonary artery aneurysm.

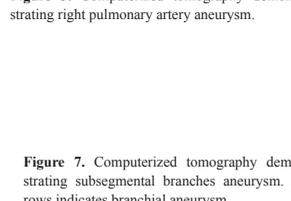


Figure 7. Computerized tomography demonstrating subsegmental branches aneurysm. Arrows indicates branchial aneurysm.

**Recurrent stress-induced cardiomyopathy: a case report****Tekrarlayan stresle tetiklenen kardiomyopati: Olgu sunumu**

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A 56-year-old female with history of hypertension, dyslipidemi was admitted to the emergency department with chest pain after learning of her mother's death. Pain was mainly retrosternal, continuous, with no aggravating or relieving factors. Physical examination was normal. Electrocardiography showed T-wave inversion on leads V1-V6 (Fig. 1a). Laboratory findings showed a significantly elevated serum level of troponin I (level was 2.39 µg/L, normal <0.1µg/L) but only slightly elevated creatin kinase (CK) and CK-MB. Transthoracic echocardiography showed basal hyperkinesis, mid to distal dyskinesis left ventricular chamber with systolic dysfunction (30% left ventricular ejection fraction). On coronary angiography we did not find coronary stenosis and ventriculography was akinesis of anterolateral, apical, and inferoapical regions, with an ejection fraction of 30% (Fig. 2a-2b). The patient was discharged in stable condition on acetylsalicylic acid (ASA) 100 mg, angiotensin converting enzyme inhibitor (ACEI) (perindopril 5 mg daily) and a beta-blocker (metoprolol 50mg daily). A follow-up echocardiography at 2 weeks showed left ventricular ejection fraction of 55% with no wall-motion abnormalities. Four months later, on learning about the sudden unexpected death of her sister, she developed left-side chest pain. The patient presented to the emergency department. Electrocardiography showed T-wave inversion on leads V1-V6 (Fig. 1b). Laboratory findings showed a significantly elevated serum level of troponin I (level was 1.28 µg/L, normal <0.1µg/L). The coronary angiography was normal and similar to previous ventriculography finding. The patient was discharged in stable condition on ASA 100 mg, beta-blocker (metoprolol 50 mg daily), and ACEI (perindopril 5mg daily). A follow-up echocardiography at 4 weeks showed an ejection fraction of 58% with no wall-motion abnormalities.

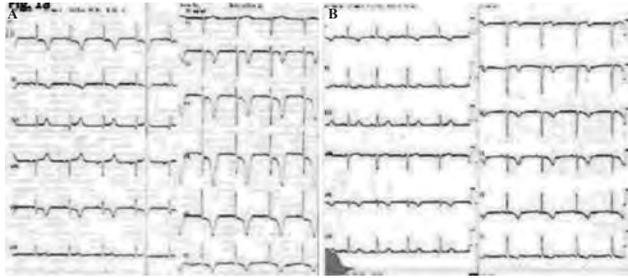


Figure 1. Sinus rhythm, significant for deep anterior T wave inversions.



Figure 2. Systolic and diastolic frames from left ventriculography illustrating akinesis of mid and apical segments.

**Hyperimmunoglobulin E syndrome (Job's syndrome) and endocarditis with intracardiac abscess****Hiperimmunoglobulin E sendromu (Job sendromu) ve intrakardiyak abse ile endokrdit**

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The hyperimmunoglobulin E or Job's syndrome is a rare disorder characterized by chronic dermatitis with recurrent sinopulmonary and skin infections and high levels of serum IgE. Patients with Job's Syndrome are often predisposed to recurrent staphylococcal abscesses usually involving skin and lungs but these infections generally lack the classical signs of acute inflammation like tenderness, erythema and warmth, so the term 'cold abscess' is used to describe this syndrome. Cardiac involvement of HIES is extremely rare with only limited numbers of cases with infective endocarditis and coronary aneurysms. We report the case of a 39-year-old patient with HIES who had methicillin-sensitive staphylococcus aureus endocarditis involving mitral and aortic valve with abscess formation: a pathognomonic lesion for Job's syndrome. The patient was previously healthy except for recurrent skin abscesses since childhood and also had no history of structural cardiac disease. Transesophageal echocardiography (TEE) revealed two vegetations attached to anterior and posterior mitral leaflets both 0.8x0.4 cm in size (figure 1). Image of a probable old abscess containing septa formation, located in the aortic annulus near to the left coronary cusp is established (figure 2). There was a fistula tract formation in the myocardium close to the circumflex artery territory, between the abscess and LVOT appearing like a "wind-sock" (figure 1) Color Doppler exhibited no blood flow at systole with near total collapse but apparent flow in diastole within the "wind-sock" (figure 3, 4). The patient was treated successfully with antibiotic treatment followed by successful surgical correction. According to this patient, it is essential to evaluate immune competence in a patient with recurrent skin abscesses and no known predisposing factor for endocarditis. Early diagnosis and proper management of Job's syndrome might prevent an undesirable and rare complication: endocarditis.



Figure 1. Transesophageal Echocardiography Vegetations on the mitral valve and wind sock appearance in LVOT.



Figure 2. Transesophageal Echocardiography. Image of a probable old abscess containing septa formation, located in the aortic annulus near to the left coronary cusp.

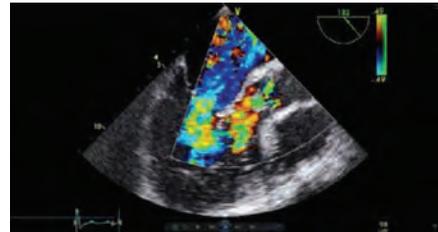


Figure 3. Transesophageal Echocardiography, color Doppler. Apparent flow in diastole within the "wind-sock".

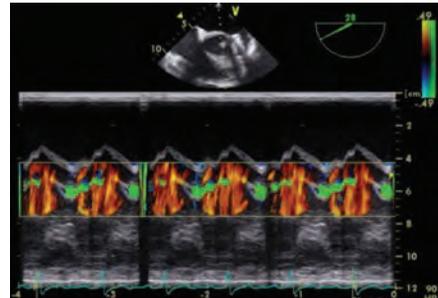


Figure 4. Transthoracic Echocardiography, color Doppler. No blood flow at systole and apparent flow in diastole within the "wind-sock".

### Coronary cascade between right coronary artery and left circumflex coronary artery in a patient with normal coronary arteries

#### Normal koroner arterleri olan hastada sol sirkumfleks arter ile sağ koroner arter arasındaki koroner kaskad

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A 41 year-old-male admitted to emergency room with typical chest pain and palpitation. He had no risk factors for atherosclerosis other than smoking. The electrocardiogram revealed supraventricular tachycardia with 170 bpm (fig 1). After achieving the normal sinus rhythm, his chest pain relieved. Cardiac biomarker was positive for Troponin I. Patient was hospitalized and coronary angiography and electrophysiologic study (EPS) were performed. Selective catheterization of the left main coronary artery did not show any significant luminal abnormality both at the left anterior descending artery (LAD) and left circumflex (LCX) (Fig 2). Also, the selective catheterization of the right coronary artery (RCA) in a left anterior oblique view did not reveal any significant stenosis. However, at later phase of the RCA angiogram, LCX was clearly visualized by the retrograde filling despite the absence of any significant stenosis in LCX (fig 3). In EPS, dual atrioventricular (AV) node physiology was detected and AV nodal reentrant tachycardia was induced and ablated successfully thereafter. Patient's chest pain and slightly elevated cardiac biomarkers were attributed to the supraventricular tachycardia which may increase myocardial oxygen demand. Intercoronary artery communication (ICC) is a rare variant of the coronary circulation. "Open ended circulation" or "coronary cascade" are also used to describe this rare entity. It is defined as an open-ended circulation with uni- or bi-directional blood flow between two coronary arteries. Two major types of the condition have been described: communication between the circumflex artery CX and the RCA in the posterior AV groove which was more common (as in our patient), and communication between left anterior descending and posterior descending artery in the distal interventricular groove. Two additional types of ICC were described between the large diagonal and PDA as well as between the ramus intermedius and LAD. Compared with collaterals, ICCs are larger in diameter, extramural, and straight. Furthermore, the structure of an ICC is typical of an epicardial coronary artery, with a well-defined muscular layer. Coronary artery flow in ICC is usually unidirectional. In case of bidirectional flow, the flow is generally from the right to left coronary artery in the absence of underlying CAD. Intercoronary arterial connections are thought to be embryonic in origin. The true prevalence of ICC is unknown and has been estimated to be approximately 2.3 per 100,000 populations. So far, we have identified only one patient with ICC at 2002. Ten year later another patient with the same angiographic entity was detected among the 48750 angiograms. The functional significance of this large anastomotic connection between normal coronary arteries is unclear but it can be speculated that in case of any significant stenosis in either of connected arteries, they have a potential role in protecting the myocardium at risk.

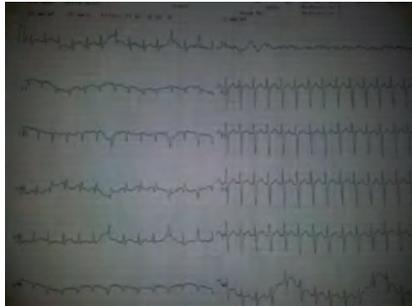


Figure 1. Electrocardiography showed supraventricular tachycardia with heart rate 170 bpm.

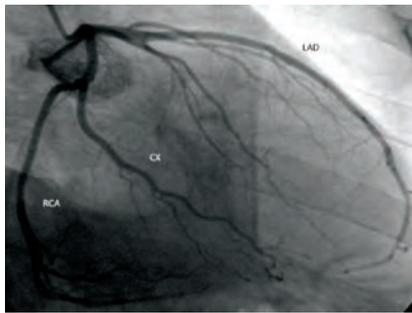


Figure 2. Left main coronary artery imaging showed normal epicardial coronary arteries without any other abnormality. (LAD: left anterior descending artery, CX: left circumflex artery, RCA: right coronary artery).

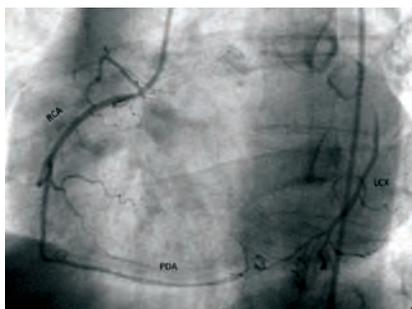


Figure 3. Selective RCA imaging showed retrograde filling of LAD simultaneously. (LAD: left anterior descending artery, LCX: left circumflex artery, RCA: right coronary artery).

### A rare complication of percutaneous coronary intervention: disruption of the dislodged stent by a loop snare technique

#### Perkütan koroner girişimin nadir bir komplikasyonu: Yerinden ayrılan bir stentin halka snare tekniği ile alınması

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During percutaneous coronary intervention, coronary stent dislodgement or embolisation before deployment is a rare but life-threatening complication that sometimes requires emergency surgical treatment. A 47-year-old man presented with unstable angina one year after first percutaneous coronary intervention (PCI). One year ago, she underwent coronary angiography that revealed 80% stenosis in the first obtuse marginal branch of the circumflex artery (Cx-OM1) and PCI was performed. She had risk factors of hypertension and hyperlipidemia. Her pulse rate was 92 beats/min and arterial blood pressure was 145/85 mmHg. Other physical examination findings were all normal. Admission electrocardiography showed normal sinus rhythm and 1-2 mm ST-segment depression on precordial leads. Angiography was performed using 7 F Judkins left and right catheters, which showed non-critical lesion of the LAD and RCA, 70% stenosis in the of the circumflex artery (Cx), 80% stent restenosis in the Cx-OM1 (Figure 1). We decided to perform PCI for the critical occlusion. A 0.014-inch floppy guide wire (CholCE, Boston Scientific, Minnesota, USA) was advanced to the Cx. After predilation of the Cx stenotic lesion with a 2.0x20-mm balloon dilatation catheter, a 2.5x25-mm sirolimus-eluting stent (Carlo S, Balton, Polska) was advanced but failed to cross the proximal Cx artery due to significant proximal angulation. The undeployed stent was dislodged while crossing the angulated segment between the left circumflex and left main coronary artery (Figure 2). The balloon was pulled out. A 1.5x15 mm Sprinter Legend balloon dilatation catheter (Medtronic) was advanced using a guiding catheter and the balloon was not pushed through the inside of the slipped stent. The dislodged sirolimus-eluting stent was caught with 4 mm Andra snare in the LMCA. However, 4mm snare distal ring was broken during traction. Unfortunately, stent was caught again with 20 mm snare (Figure 3A-3B) and the dislodged stent was finally disrupted during strongly traction (Figure 4A-4B). The dislodged sirolimus-eluting stent was surgically removed, and the patient underwent coronary artery bypass grafting successfully. To our knowledge, there have been no reports on dislodgement and disruption of a sirolimus-eluting stent by a loop snare technique. There is a rising trend for drug-eluting stents in PCI. The numbers of stent loss during PCI has decreased in recent years, probably due to improvements in equipment design. Stent dislodgement and embolization are serious complications of PCI and these complications are sometimes life-threatening and require emergency surgical treatment. Finally, physicians should keep in mind that stent loss occurred more frequently in lesions with calcification and/or significant proximal angulation and despite everything, unexpected complications may occur every time.

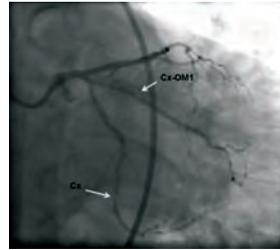


Figure 1. Coronary angiogram showing a critical stenosis of the highly angulated circumflex (Cx) and first obtuse marginal branch of the circumflex (Cx-OM1) coronary artery.



Figure 2. Coronary angiogram showed a 2.5x25-mm sirolimus-eluting stent entrapment in left main and left circumflex arteries (arrow).

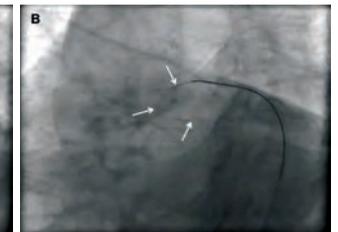
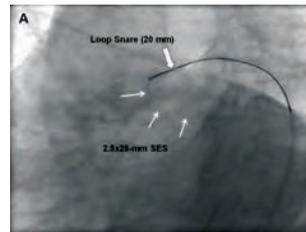


Figure 3. (A) Retrieval was not accomplished by a loop snare technique (20 mm snare). (B) Retrieval was not accomplished by a loop snare technique (20 mm snare).

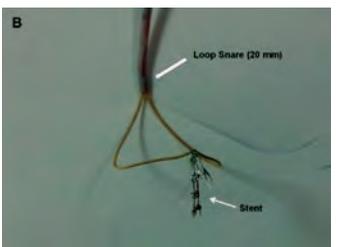


Figure 4. (A) Disruption of the sirolimus-eluting stent. (B) Disruption of the sirolimus-eluting stent.

OS-34

### Successful percutaneous mitral valve repair with the MitraClip system of acute mitral regurgitation due to papillary muscle rupture as complication of acute myocardial infarction

**Akut miyokard enfarktüsünün komplikasyonu olarak papiller kas rüptürüne bağlı akut mitral yetersizliğinin MitraClip sistemi ile başarılı perkütan mitral kapak tamiri**

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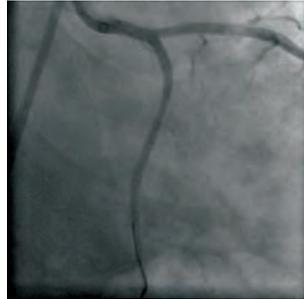
**Introduction:** Myocardial infarction (MI) is a well-recognized cause of papillary muscle rupture (PMR). For patients with PMR, the standard therapy for MR is open surgical repair or replacement. Percutaneous mitral valve repair with the MitraClip is a new promising therapeutic option for symptomatic severe MR. Here we present, to the best of our knowledge, the first case of percutaneous mitral valve repair with the MitraClip system of acute MR due to PMR as complication of acute MI.

**Case:** A 60 year-old woman was transferred to our hospital with acute MI, pulmonary edema and cardiogenic shock that required endotracheal intubation. Her medical history included hypertension, diabetes mellitus and colon cancer which was treated with surgery, chemotherapy and radiotherapy and recently complicated by metastases. An electrocardiography showed an acute posterolateral MI. Coronary angiography showed a thrombotic total occlusion in the ostium of circumflex artery and a critical lesion in high obtuse marginal branch with relatively small size (Figure 1). The lesion in the ostium of circumflex artery was stented successfully without protection of obtuse marginal branch (Figure 2). After the primary percutaneous coronary intervention, physical examination revealed a 3/6 holosystolic murmur at the apex and significant bilateral rales. A transesophageal echocardiography (TEE) demonstrated the ruptured anterior papillary muscle and very severe MR from the lateral scallops of both mitral leaflets (Figure 3). Since the cardiac surgery refused the patient due to metastatic colon cancer, the patient and her family were offered percutaneous repair of the acute MR and informed consent was obtained. With the patient under general anaesthesia and using fluoroscopic and TEE guidance, the MitraClip device was directed towards the origin of the regurgitant jet mainly between P1 and A1 scallops and advanced into the left ventricle. Only one attempt at grasping was necessary to achieve the optimal result without any technical issues. In the end of the procedure, TEE demonstrated a significant reduction of MR grade from IV to trace residual MR (Figure 4). At 5 months follow up, she was clinically stable and transthoracic echocardiography revealed a mild degree of MR.

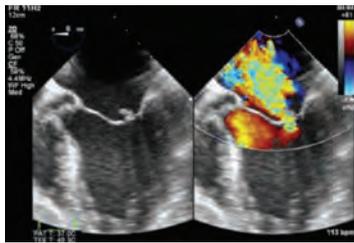
**Discussion:** If PMR is untreated, the prognosis is poor and the mortality could be as high as 80% during the first week of post MI. In our case, mitral valve repair with the MitraClip was chosen technique because of the metastatic colon cancer. This report shows that MitraClip could be safe, feasible and effective treatment in the treatment of acute MR due to PMR as complication of acute MI. Treatment of acute MR due to PMR by MitraClip may be off-label but a feasible treatment in selected no-option patients.



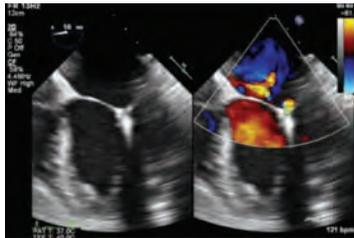
**Figure 1.** Coronary angiography shows thrombotic total occlusion in the ostium of circumflex artery and a critical lesion in high obtuse marginal branch.



**Figure 2.** Angiogram after successful primary percutaneous coronary intervention.



**Figure 3.** Transesophageal echocardiography demonstrates the ruptured anterior papillary muscle connected to chordae tendineae and severe mitral regurgitation from the lateral segments of both leaflets (A1 and P1 segments) in superior five-chamber view.



**Figure 4.** Postprocedure transesophageal echocardiography demonstrates resolution of the severe mitral regurgitation in superior five-chamber view.

OS-35

### Spontaneous spinal epidural hematoma following percutaneous coronary intervention: early detection, early intervention ang good result

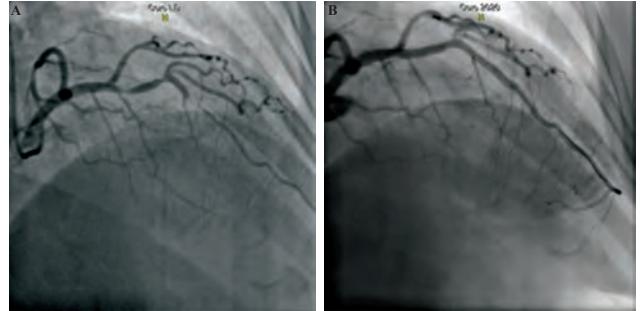
**Perkütan koroner girişimi takiben spontan spinal epidural hematom: Erken tanı erken girişim ve iyi sonuçlar**

Ferhat Özyurtlu<sup>1</sup>, Nihat Pekel<sup>1</sup>, Mehmet Emre Özpeli<sup>1</sup>, Nurullah Çetin<sup>2</sup>

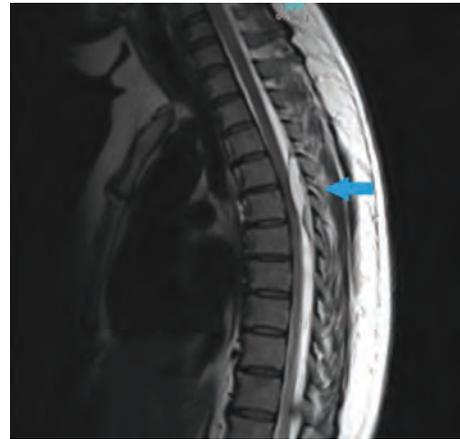
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<sup>2</sup>Celal Bayar University, Faculty of Medicine, Department of Cardiology, Manisa

A 54-year old female patient entered the emergency department with typical chest pain. She had a history of hypertension. The electrocardiogram showed sinus rhythm and ST segment depression. The troponin value was 0.4 ng/mL. After the patient was admitted to hospital with a diagnosis of non-ST-elevated myocardial infarction, coronary angiography was performed. Consecutive critical lesions were detected in the left anterior descending artery (LAD) (Figure 1a). PTCA+stent application to LAD was successful, and the process was terminated with no complications (Figure 1b). Before the procedure, the patient had been given 300 mg acetylsalicylic acid and 300 mg clopidogrel. During the procedure, 7000 I.U. of unfractionated heparin was given. At the 12th hour after the procedure, the patient developed severe back pain and paraplegia afterwards. A mass of 5x1 cm in size compressing spinal cord prominently and giving the impression of hematoma was observed in magnetic resonance imaging dorsal T5-6 level (Figure 2). The patient underwent urgent surgical operation with laminectomy and decompression attempts. The patient improved progressively after the operation, and on the 6th follow-up day the findings of paraplegia recovered completely. The patient was discharged from the hospital after the arrangements for her therapy. Spontaneous spinal epidural hematoma is a rare clinical condition, and it accounts for less than 1% of the spinal epidural-space occupied lesions. SSEH organic vascular diseases are more common in anticoagulant therapy associated with hemodialysis, coagulation disorders, and stroke. Decompressive treatment should be given urgently if there is neurological deficit. The prognosis of patients who have cardiovascular diseases and who are receiving anticoagulant therapy is poor. Early diagnosis and early treatment is of crucial importance in these patients in terms of mortality and morbidity. And it was because of the early diagnosis and intervention that we achieved a favourable outcome with our patient in a short time. Although a few cases developing SSEH associated with the treatment of acute myocardial infarction have been reported in the literature, no cases have been reported as in our case, where neurological signs disappeared completely in a short time.



**Figure 1**



**Figure 2**

OS-36

### Management of CoreValve embolization to the ascending aorta with a second CoreValve with valve-in-valve approach in the ascending aorta

#### Asendan aortada CoreValve embolizasyonunun kapak içinde kapak yaklaşımı ile ikinci bir CoreValve ile yönetilmesi

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**Introduction:** One of the complications encountered during the procedure of transcatheter aortic valve implantation (TAVI) is embolization of the prosthetic valve. Here, we present a case of CoreValve (CV) embolization to the ascending aorta who was treated with a second CV in the ascending aorta with valve-in-valve approach. In the literature, there has been just one reported case of this kind.

**Case:** Symptomatic severe aortic stenosis was detected in a patient who underwent coronary bypass surgery and aortic valvotomy procedures in 2004. The patient was found to be at high risk for surgical aortic valve replacement and TAVI procedure was planned with CV. After balloon dilatation of aortic valve, a 26 mm CoreValve prosthesis was advanced into the aortic annulus. The CV was positioned with its lower end approximately 4 mm below the annulus towards the left ventricular outflow tract. After making sure that the position of the valve was good in control images, valve implantation was performed. However, the valve separated very quickly from the delivery system and embolization occurred to the ascending aorta (Figure 1). Then a larger CV (29 mm) was passed through the previous valve. The first valve was attempted to pull back to descending aorta with snare catheter but the valve went backward to the mid portion of the ascending aorta each time. As stabilization with snare catheter couldn't be achieved and the patient's ascending aorta was too short, it was decided to implant the second valve in the ascending aorta with valve-in-valve approach by disabling leaflets of the first valve with the second valve's nitinol struts. At this moment, much attention was paid especially to unblock coronary blood flow with the tissue of first valve's skirt part and the second valve was implanted successfully (Figure 2). Control images after the procedure showed there was no restriction of blood flow in the coronary and there was a trace aortic regurgitation.

**Discussion:** The preferred treatment method of prosthetic valve embolization during TAVI procedure is to pull the embolized valve to ascending or descending aorta with a snare catheter to ensure a stable position and, to implant a second valve intra annularly a bit far from the first valve. In our case, as stabilization with snare couldn't be achieved and the ascending aorta of the patient was too short, following the optimal positioning of the first and second valve in the ascending aorta, the second valve was implanted with valve in valve method by disabling leaflets from the first valve with the second valve's nitinol struts. This procedure was not performed at intraannular level, but was performed at the higher position in the ascending aorta. As a result, embolization of the CV prosthesis to ascending aorta can be treated with a second valve by valve-in-valve method in the ascending aorta.



Figure 1. The image shows the embolization of CoreValve to the ascending aorta.

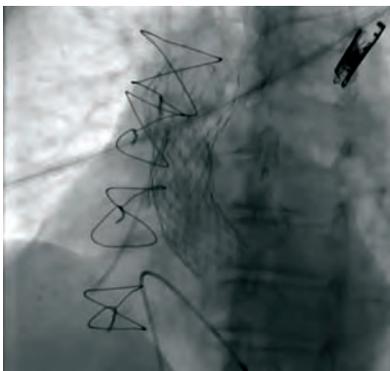


Figure 2. The image shows successfully implantation of second valve by approach of valve-in-valve.

OS-37

### An adult patient with ALCAPA syndrome and his 8 years follow up

#### ALCAPA sendromu olan erişkin bir hasta ve sekiz yıllık takibi

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**Case report:** 36 years old male patient was admitted to Sanko Hospital Cardiology Clinic with a complaint of left sided atypical chest pain in 2005. He was an active sportsman without any known disease and drug use. Electrocardiography showed sinus rhythm without any pathologic finding. Transthoracic echocardiography was suboptimal due to poor echogenicity and revealed normal systolic functions with normal sized heart chambers, mild mitral and tricuspid regurgitation and pulmonary artery pressure of 40 mmHg. In parasternal short axis view, an indefinite turbulent flow was detected in pulmonary artery by color and continuous wave Doppler. There was not any ischemic ST-T changes in exercise stress test. Soon after cardiac catheterization was performed. Since the left coronary artery (LCA) could not be seated by left Judkins and Amplatz catheters in the first step, the operator shifted to the right coronary angiography. In right coronary angiography, right coronary artery (RCA) was dilated and there were well developed coronary collaterals filling LCA and leading pulmonary artery washing by a reverse flow. In coronary computed tomography, LCA was shown to arise from pulmonary artery and perfused by collaterals directly from aorta and RCA. A surgical correction was planned but the patient refused the therapy. He has been under follow up since 2005 and still asymptomatic.

**Discussion:** Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) or Bland-White-Garland (BWG) syndrome, is a rare congenital anomaly. If it is left untreated, ALCAPA has a 90% mortality in the first year of life due to mostly myocardial ischemia and heart failure. ALCAPA is a rare congenital malformation with a prevalence of 1/300,000 live births and constitutes about 0.24-0.46 % of all congenital heart defects. Other congenital cardiac malformations like aortic coarctation, atrial septal defect and ventricular septal defect can accompany in 5% of patients. Adult patients can be completely asymptomatic or present with angina, dyspnea, syncope, myocardial infarction, arrhythmia or sudden cardiac death. Sudden cardiac death secondary to malign ventricular arrhythmias is the most common presentation in adult patients. Herein, we presented a case of ALCAPA syndrome in a patient who was 36 years old and an active sportsman with atypical chest pain having only an indefinite turbulent flow in his pulmonary artery during echocardiography due to poor echogenicity. In our case, we detected collaterals to LCA directly arising from aorta which was not reported previously in the literature. As a result, ALCAPA syndrome is a rarely seen clinical entity in which early diagnosis and treatment can prevent myocardial ischemia and related symptoms.

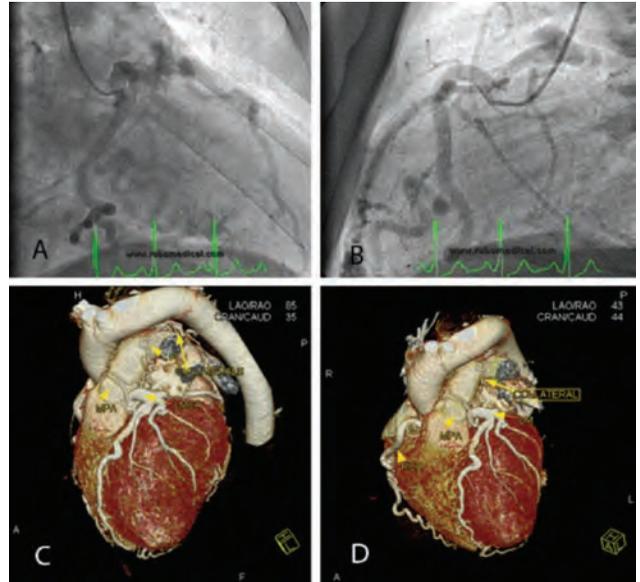


Figure 1. A-B view of right coronary artery during angiography, C-D view of collaterals in CT angiography.

OS-38

### Woven coronary anomaly of right coronary presenting with hypokinesia in posterobasal segment of the heart

#### Sağ koroner arterin kalbin posterobasal segmentinde hipokinezi ile kendini gösteren seyrek anomalisi

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**Introduction:** Woven coronary artery is an extremely rare and is still not a clearly defined coronary anomaly in which epicardial coronary artery is divided into multiple thin channels at any segment of the coronary artery, and subsequently, these multiple channels merge again in a normal conduit. A few cases have been reported till now.

**Case Presentation:** A 55 year-old Caucasian man presented to our outpatient clinic with chest pain. It was the first time he had such pain. It is a pressure like non radiating chest pain on the left side of thorax which takes 5-10 minutes. Physical examination and vital signs were within normal limits. Biochemistry and cardiac biomarker results were also within normal limits. Q wave was found in D2,3,aVF leads in the ECG. Segmenter wall motion disorder in posterobasal segment was found in the echocardiographic examination. Patient was admitted for coronary angiography. We found critical long lesion composed of thrombus in the mid part of right coronary artery. We decided to continue to procedure with PTCA. After careful examination we decided it to be a woven coronary anomaly. We also found a hypokinesia in posterobasal segment after ventriculography.

**Conclusion:** In this case we describe a patient in which we expected to find coronary artery disease in right coronary artery prior to coronary angiography. But after careful examination we decided it to be a woven coronary anomaly. A woven coronary artery is a rare coronary anomaly but an expertise must always show a careful attention especially in conditions in which severe coronary artery disease is expected like acute coronary syndrome, segmenter wall motion disorder etc. Otherwise a misdiagnosis of lesion with a thrombi is inevitable.

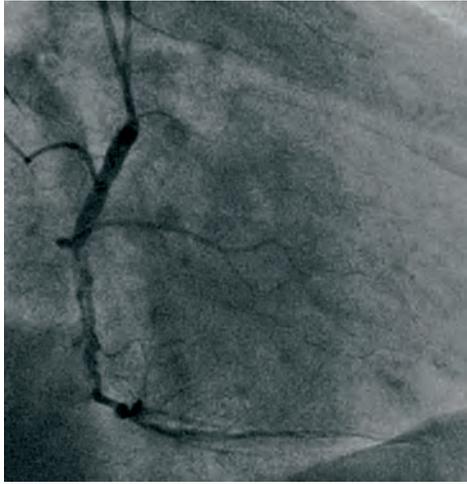


Figure 1. Woven coronary artery.

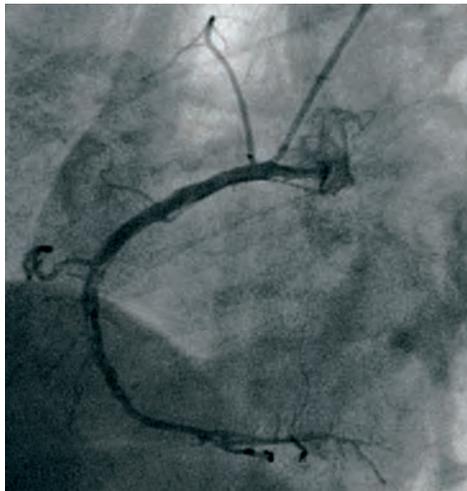


Figure 2. Woven coronary artery in RCA.

OS-39

### Intra-arterial tPA application with help of ultrasonic waves in critical lower limb ischemia

#### Kritik alt ekstremité iskemisi için ultrason dalgaları yardımıyla intraarteriyel tPA uygulaması

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**Introduction:** Thrombolytic therapy has been established as an important therapeutic tool in the treatment of acute ischemic events. Patients with peripheral arterial diseases (PAD) are at high risk for thrombotic occlusions. In patients with acute lower extremity ischemia (ALEI), thrombus is present in the majority of vessels. Endovascular ultrasonic treatment is a new therapeutic procedure for arterial recanalization. EKOS system is the first system that allows the application of endovascular ultrasound-lysis, using a catheter for intra-arterial or intra-venous administration of thrombolytics terminated with the emitter of ultrasonic waves. It emits ultrasound waves with the frequency between 1.7 and 2.35 MHz and with the emitted intensity of 400 mW/cm<sup>2</sup> into the thrombus. Herein, we describe a case of PAD complicated with acute left femoral artery thromboembolism.

**Case Report:** A 77 year old male, applied to our emergency department with a sudden left lower extremity pain and pallor, had coronary and peripheral artery diseases. His blood pressure was 135/88 mmHg. Electrocardiography of him showed prior inferior myocardial infarction and heart rate was 88/min. Laboratory studies revealed a creatinine level of 1.18 mg/dl, urea 43 mg/dl and potassium 4.6 mEq/l. CRP level was increased. In the physical examination, we didn't sense his left tibialis posterior and dorsalis pedis pulses. Doppler ultrasonography showed occluded left lower extremity arteries. The patient was taken back to the angiography laboratory immediately. Peripheral angiography showed total occlusion of the superficial femoral artery (Figure 1). At the beginning of the procedure, heparin was administered intravenous (50 IU/kg). We inserted a long sheath into the right common femoral artery and turned from iliac bifurcation into the left iliac artery. We passed through the lesion with a hydrophilic wire. The lesions were dilated with a peripheric balloon. (Figure 2). There were huge thrombus particles in the superficial femoral artery after balloon dilatations (Figure 3-4). We discussed the patient's treatment and decided to use endovascular ultrasound system. In this case, a dose of 15 mg bolus and 20 mg/24h of tPA was delivered by EKOS system (Figure 5). The next day, after 24 hours of ultrasonic tPA application had expired, the control peripheral angiography was performed. Right superficial femoral artery flow was found to be good (Figure 6). It was seen that proximal thrombus completely disappeared but they were present in the vessel lumen as small particles while progressing through distal (Figure 7). We decided to follow-up the patient with the medical treatment. The patient was discharged with the therapy of warfarin, clopidogrel, silositazol, atorvastatin and diltiazem.

**Conclusion:** Endovascular ultrasonic thrombolysis is a relatively safe treatment with a high efficacy in the acceleration of lower extremity arteries recanalization.



Figure 1. Left lower extremity before intervention.

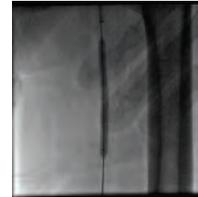


Figure 2. Balloon dilatation.



Figure 3. After balloon dilatations. There were huge thrombus particles in the superficial femoral artery after balloon dilatations.



Figure 4. After balloon dilatations. There were huge thrombus particles in the superficial femoral artery after balloon dilatations.



Figure 5. Endovascular ultrasound thrombolysis system.



Figure 6. Control Angiography. The next day, after 24 hours of ultrasonic tPA application had expired, the control peripheral angiography was performed.



Figure 7. Control Angiography. It was seen that proximal thrombus completely disappeared but they were present in the vessel lumen as small particles while progressing through distal.

## OS-40

## Recurrent pheochromocytoma and the recurrence of cardiomyopathy

## Rekürren feokromasitoma ve kardiyomiyopati rekürrensi

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Pheochromocytoma is a rare neuroendocrine tumor and its most frequent cardiovascular complications are hypertension, myocardial ischemia, arrhythmia and heart failure (HF). In this case report, a patient with a recurrence of cardiomyopathy due to recurrent pheochromocytoma is presented. A 38-year old female patient presented with decompensated heart failure due to dilated cardiomyopathy as a complication of pheochromocytoma. The patient, who had a history of hypertension, was diagnosed with dilated cardiomyopathy six years ago with a left ventricular ejection fraction (LVEF) of 20% and normal coronary arteries on cardiac catheterization. An implantable cardioverter-defibrillator was implanted 4 years ago with an indication of persistent left ventricular dysfunction. Patient was hospitalized with decompensated heart failure 3 years ago and 1 month duration of positive inotropic therapy failed to improve clinical status. Emergency heart transplantation was planned and a left adrenal mass of 55x95 mm was discovered during pre-implantation work-up. The patient was diagnosed with pheochromocytoma after the discovery of very high levels of VMA, normetanephrine and metanephrine in 24-hr urine analysis. 3 months after the surgical excision of pheochromocytoma, patient's functional capacity was improved and LVEF was 45%. With the recurrence of heart failure symptoms 1 year after surgery, LVEF was reevaluated and was found to be 28%. Abdominal computed tomography showed left adrenal recurrent mass of 2 cm and activity compatible with pheochromocytoma on I-123 MIBG scan was demonstrated. A second surgical excision for left adrenal mass was performed and pheochromocytoma was confirmed with immunohistochemistry. Patient's symptoms improved promptly, and 3 months after the second surgery LVEF was increased up to 40% and urinary catecholamine levels were demonstrated to be normal. Cardiomyopathies due to pheochromocytoma improve generally after tumor resection. In this case, symptoms and cardiomyopathy which were improved after tumor resection, reproduced with tumor recurrence. Excision of the tumor with a second surgery resulted in the improvement of cardiomyopathy. This is the first case report to demonstrate recurrent cardiomyopathy with the recurrence of pheochromocytoma.

## OS-41

## Hemolysis as a first sign of thrombo-embolic events in patients with continuous-flow left ventricular assist devices

## Devamlı akım sol ventrikül destek cihazı olan hastalarda tromboembolik olayların ilk işareti olarak hemoliz

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**Objectives:** Continuous flow left ventricular assist devices (cf-LVAD) has become a cornerstone in the treatment of end stage heart failure. Despite advances in pump technology, thrombus formation and subsequently thrombo-embolic events (TE) remains a potential life-threatening complication of LVADs. The aim of our study was to determine the early signs and symptoms of TE in patients with cf-LVAD which could lead to earlier intervention.

**Methods:** We retrospectively analyzed all patients (n=40) in which a Heart-Mate II device was implanted as bridge-to-transplant between 2007 and February 2013 in our tertiary referral hospital. TE was defined as transient ischemic attack (TIA), cerebro-vascular ischemic attack (CVA) or acute pump thrombosis.

**Results:** during median LVAD support of 336 days [IQR 172-839] there were 8 (20%) patients with TE (six patients with TIA/CVA, two with acute pump thrombosis). Of these two patients with acute pump thrombosis, one was treated with urgent pump change and other with thrombolysis. There were no significant difference in the baseline characteristics between the groups with and without TE (age (53 ± 9 vs. 46 ± 13 years, p=0.13; sex: male 75% vs 63% p=0.51; etiology: ischemic 4/8 vs. non-ischemic 13/32, p=0.63; basic LVAD settings: mean pump rate 9375 ± 225 rpm vs. 9312 ± 568 rpm, flow 4.8 ± 1.0 vs. 5.0 ± 1.2, pulse index (PI) 4.9 ± 0.7 vs. 4.9 ± 0.9 or pump power (PP) 6.1 ± 1.0 vs. 6.0 ± 1.3). At the time of TE (median INR 2.4 [2.1-4.0]), the TE patients had significant higher PP (8.2 ± 3.0 vs. 6.4 ± 1.4, p=0.05), a trend to lower PI (4.2 ± 1.5 vs. 5.1 ± 1.1 p=0.07) but normal flow (5.7 ± 1 vs. 5.0 ± 0.9 p=0.73). In TE group, there was a significant higher lactate dehydrogenase (median LDH: 1548 [748-2565] vs. 350 [320-439] U/L, p=0, 0001) as sign of severe hemolysis. In the half of these patients, an infection preceded the TE.

**Conclusions:** Thrombo-embolic events in cf-LVADs were accompanied by symptoms and signs of hemolysis, in particular with high levels of LDH and elevated pump power. Infections could be a trigger for these events. This could probably aid clinicians in making an early diagnosis and intensify timely the anticoagulation and antiplatelet therapy in an attempt to prevent acute pump thrombosis/thromboembolic events.

## OS-42

## Massive pericardial effusion due to intrapericardial mixed germ cell tumor in a premature baby: a case report

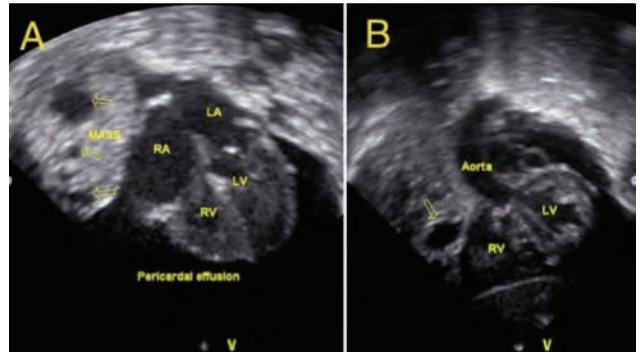
## Prematür bebekte intraperikardiyel germ hücreli tümöre bağlı masif perikardiyel efüzyon: Olgu sunumu

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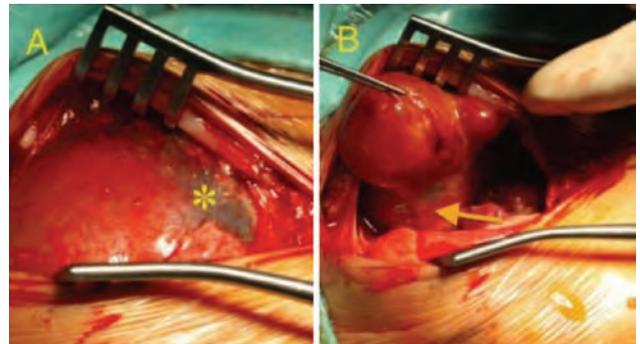
**Introduction:** Having unique location and clinical presentation, germ cell tumors are rare tumors of childhood representing only 1% to 3% of childhood tumors. Twenty percent of germ cell tumors are malignant, related with age and location. Extragonadal sites account in nearly half of the patients where as adult form only in %10. Yolk sac tumor in which alpha fetoprotein (AFP) is the marker of the disease is the most seen malignant histology. Striking localization of malignant germ cell tumors is anterior mediastinum but rare involvement of pericardium and aortic adventitia have been reported. Here we present a premature baby of twins with hydrops fetalis having intrapericardially located mixed germ cell tumor.

**Case:** A male appropriate for gestational age premature twin baby was born after 33 weeks of gestation with hydrops fetalis that was detected during routine ultrasound follow-up. The telecardiogram revealed a wall to wall cardiac silhouette. Echocardiography showed massive pericardial effusion with heterogeneous solid and multicystic 40x35 mm intrapericardial mass. Although it was adherent to ascending aorta, didn't cause any obstruction to caval system (Figure 1). Other-wise cardiac structures and functions were normal except patent foramen ovale. Diagnostic and therapeutic pericardiocentesis was performed and 55 ml of serohemorrhagic pericardial fluid was taken. The fluid was consistent with exudates nature. Although cytopathological examination revealed numerous red blood cells no malign cells were detected. Alfa fetoprotein level was 62576 ng/mL (N: 0.0-7.0) and beta human chorionic gonadotropin (beta hCG) level was 1.1 mIU/mL (N: 0.0-5.0). With median sternotomy an encapsulated 6x5 cm sized, extracardiac tumor was removed (Figure 2). Post surgical recovery was uneventful except minimal pericardial effusion. Alfa fetoprotein level fell to 14048 ng/mL, two days after the operation. The specimen was lobulated and 6x4.5x2.5 cm in size. Pathological macroscopic examination revealed solid gray-white areas with small cystic formations with mucoid liquid inside were seen. Microscopically; although mature teratoma was seen in most of the microscopic sections; immature fields were detected in some areas. Yolk sac component was seen in only one section among numerous samples and sections, it was stained strongly positive by AFP and alfa-1 antitrypsine and weakly positive with pancreatine and beta hCG. Finally, the diagnosis was mixed germ cell tumor. The first cure of chemotherapy which contain etoposid, cisplatinium and ifosfamide is given. In follow-up there were no pericardial effusion and any residual mass.

**Conclusion:** Primary cardiac tumors are uncommon in childhood, with an incidence of 0.06% to 0.32% fortunately intrapericardial teratoma represents an exceptional rarity among these entities. Early fetal imaging with ultrasound and echocardiography are reliable tools in identifying these cardiac teratomas.



**Figure 1.** (A) Echocardiography showed massive pericardial effusion, solid and multicystic (arrows) anterior placement intrapericardial mass; adjacent to the right atrium and vena cava superior. (B) Mass is adherent to ascending aorta.



**Figure 2.** (A) After sternotomy, anteriorly located mass and pericardial effusion (\*) are shown. (B) Mass, adherent to ascending aorta (arrow) is shown.

OS-43

**A case of left ventricular noncompaction with genital and skeletal anomalies and mental retardation****Genital ve iskelet anormallikleri ve mental retardasyon ile birlikte sol ventriküler non-kompaksiyon olgusu**

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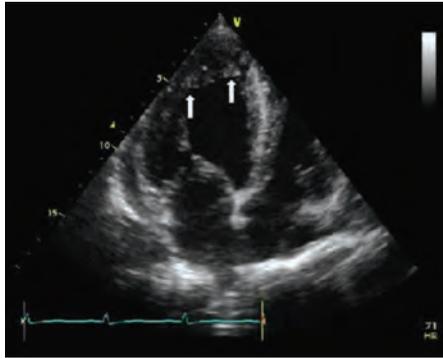
**Introduction:** Here we report a 48-year-old female with left ventricular noncompaction, ovarian dysgenesis, uterine aplasia, skeletal anomalies and mental retardation.

**Case description:** 48-year-old female was admitted to the intensive care unit with acute pulmonary edema. Her past medical and surgical history were unremarkable except for primary amenorrhea. There was consanguineous marriage (fourth degree) between parents of the patient. The patient had healthy three brothers and a sister, but two sisters and a brother had died in childhood from unknown reasons. On admission vital signs were as follows: blood pressure 90/55mmHg, heart rate 124 bpm, respiratory rate 30 per min. On inspection macrocephaly, acromegaloïd face appearance, arachnodactyly, pectus carinatum, bilateral amazia and mild scoliosis were remarkable (Figure 1). The ECG at admission showed sinus tachycardia with rate of 120/min, left ventricular hypertrophy with secondary ST segment changes. Transthoracic echocardiography revealed dilatation of all heart chambers, globally impaired systolic function (EF Simpsons biplane = 25%) and mild mitral and tricuspid regurgitation. There were prominent trabeculations and intertrabecular recesses in lateral wall, septum and apex of the left ventricle. The noncompacted to compacted ratio was 3.5 at the thickest part of the lateral wall on the parasternal short-axis view (Figure 2). Coronary angiography showed normal coronary arteries. Cardiac MRI confirmed the diagnosis of left ventricular noncompaction cardiomyopathy (LVNC) (Figure 3). The patient was treated medically with carvedilol, perindopril, spironolactone and furosemide. In respect to past medical history, gynecology consultation was done. Laboratory investigations revealed hypergonadotropic hypogonadism and pelvic MRI demonstrated absence of ovaries, uterus or prostate. Cytogenetic analysis showed 46, XX karyotype without any major chromosomal abnormalities. Due to non-adherence to the medical treatment, there were recurrent hospitalizations with heart failure decompensation.

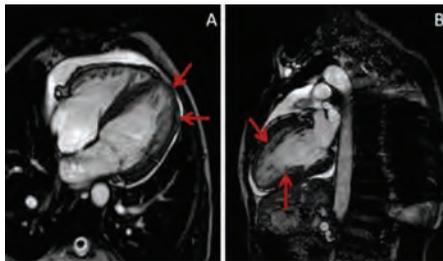
**Discussion:** Coexistence of LVNC with genital anomalies is very rare and we could find only two recently reported cases. Our case had genital and skeletal anomalies in addition to LVNC. First case of coexistence of hypergonadotropic hypogonadism with dilated cardiomyopathy described in 1973 by Najjar et al. and since that time only 15-20 similar cases have been reported in English literature (4). The common features of these cases are dilated cardiomyopathy, hypoplastic genitalia and hypergonadotropic hypogonadism and. Although cardiac involvement in our case is distinct from these reports but extracardiac manifestations are very similar, especially to the case reported by Narahara (5). To the best of our knowledge, this is the first case with LVNC and female genital anomalies. Although our case is isolated, but unexplained death of the patient's siblings may support the inheritance.



**Figure 1.** Acromegaloïd face appearance, pectus carinatum, arachnodactyly and bilateral amazia.



**Figure 2.** Apical four chamber view with a dilated left ventricle (LV) and a non-compacted layer at the apical septal segment and the anterolateral wall (arrows).



**Figure 3.** (A) Four chamber plane showing the non-compacted left ventricle (LV) myocardium at the apical segment and lateral wall (arrows); the maximum ratio non-compacted/compacted layer is 3.5 (B) Two chamber plane, depicting the non-compacted myocardium at the apical and the mid anterior and inferior segments (arrows).

OS-44

**The rare cause of hypertension in turner syndrome, Midaortic stenosis syndrome****Turner sendromunda nadir görülen hipertansiyon nedeni; Mid aortik darlık sendromu**

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**Introduction:** Midaortic Stenosis Syndrome (MAS) is defined as stenosis seen in the ductal thoracic and abdominal segment of aorta or stenosis that affects the whole length of the aorta. It is a very rare type of coarctation and it makes up 0.5-2% of all coarctation cases. The etiology of MAS consists of congenital causes, acquired diseases, and sometimes some syndromes. Here we are going to discuss a cause of MAS seen together with turner syndrome.

**Case:** In a patient that was being routinely followed up in the pediatric endocrinology outpatient clinic with the diagnosis of turner syndrome was consulted to the pediatric cardiology clinic with discovery of high blood pressure. The following were found in the patient's physical examination; a systolic murmur of 2/6 at the right lower border of the sternum. Most strikingly there was an absent right femoral pulse and a weak left femoral arterial pulse. While blood pressure of the upper extremity was found to be 120/80 mmHg, the blood pressure of the distal extremities could not be measured. The telecardiography indicated a dilated aortic root, normal cardiotorasik ratio and vascularity. The patient's electrocardiography was normal. An echocardiography was also done and it showed a bicuspid aorta with mild stenosis. Due to the discovery of the tortuous appearance of the descending aorta with a non-diastolic flow gradient pattern of 20 mmHg, the clinical presentation of the hypertension in the patient and non palpable femoral pulses, a decision to do catheter-angiography was made. The results of the catheter-angiography showed a slight tortuous descending aorta but there was no significant pressure difference between the upper and lower segments of the descending aorta. In the opaque contrast investigation of the full aorta, downward stenosis started from the level of midaorta was seen. This stenosis was seen to be more apparent in the right femoral artery when compared to the left. The decision was taken as routinely follow-up by pediatric cardiology and cardiovascular surgery department.

**Conclusion:** There are about 200 diagnosed cases of MAS that have been reported in the literature but this is the second case of MAS associated with turner syndrome. In turner syndrome patients with hypertension whose echocardiography shows no classical evidence of coarctation, it is necessary to perform further radiological evaluation of the whole thoracoabdominal aorta an even femoral arteries by either performing a catheter angiography or a cardiac computerized tomography.

**Giriş:** Mid aortik darlık sendromu (MAS) distal torakal ve abdominal aortanın segmenter yada tüm aorta boyunca olan daralması olarak tanımlanan nadir rastlanılan bir koarktasyon tipidir. Tüm koarktasyonların %0.5-2'sini oluşturur. Etiyolojide konjenital nedenlerin yanı sıra, edinsel nedenler ve bazı sendromlar rol oynamaktadır. Burada Turner sendromuna eşlik eden MAS'lı bir olgu sunulmuştur.

**Olgu:** Çocuk endokrin polikliniğinde Turner Sendromu tanısı ile takip edilirken tansiyon yüksekliği saptanması üzerine çocuk kardiyoloji polikliniğine yönlendirilen hastanın fizik muayenesinde sternum sağ alt kenarda 2/6 sistolik üfürüm duyuldu. Özellikle sağ femoral arter pulsasyonu alınmayan, sol femoral arter nabızı zayıf alınan hastanın alınan üst extremite tansiyon değerleri 120/80 mmHg iken alt extremite tansiyonları ölçülemedi. Telekardiyoğrafik incelemede aort kökü genişlemiş, vaskularite ve kardiyotorasik oran normaldi. Elektrokardiyoğrafisi normal idi. Çekilen ekokardiyoğrafide biküspit aorta, hafif derecede aort stenozu izlendi. Desendan aortanın tortuöz görünümde olması, buradan ölçülen diastole uzanmayan akım gradientinin 20 mmHg olması, hastada hipertansiyon olması ve femoral arter nabızlarının alınmaması nedeni ile kateter-angiografi yapmaya karar verdik. Yapılan kateter anjiyografide desendan aortada hafif tortuöz görünüm izlendi, ancak bu bölge ile üst ve alt segmentler arasında belirgin basınç farkı saptanmadı. Tüm aortanın opak madde ile injeksiyonunda özellikle midaortik seviyeden itibaren aortanın giderek daraldığı, bu daralmanın sağ femoral arterde sola göre daha belirgin olduğu izlendi. Kalp damar cerrahi konseyinde takibe karar verildi.

**Sonuç:** Literatürde yaklaşık 200 kadar MAS tanılı olgu vardır, ancak turner sendromunun eşlik ettiği vakamız literatürdeki ikinci vakadır. Hipertansiyonu olan Turner Sendromlu hastalarda ekokardiyoğrafisi ile klasik koarktasyon saptanmayan vakalarda gerek kateter-angiografi gerekse de kardiyak bilgisayarlı tomografi ile torakal-abdominal aortanın ve femoral arterlerin görüntülenmesi gereklidir. Vakamız nadir görülmesi ve Turner Sendromlu hastalardaki hipertansiyonun değerlendirilmesinde yeni bir boyut kazandırması nedeni ile sunulmuştur.

### Transjugular approach to device closure of atrial septal defect in a child with interrupted inferior vena cava

#### Kesintili vena kava inferiorlu olguda juguler ven yolu kullanılarak perkütan atrial septal defektin kapatılması

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**Introduction:** Interrupted vena cava is a rare congenital anomaly; associated with other congenital heart defects and left isomerism.

**Case:** Six years old girl referred to our clinic for heart murmur. Second degree systolic ejection heart murmur was heard on the left sternal border. Incomplete right branch block was seen in electrocardiography and cardiomegaly was found in telecardiography. 16 mm sized atrial septal defect was shown in echocardiography. Firstly we planned to close ASD percutaneously but interrupted vena cava with azygos continuation to the superior vena cava was revealed. Therefore ASD closure was not performed through femoral venous route. We planned to use jugular venous route. From right jugular vein vena cava superior, right atrium and left atrium was accessed. Defect was closed 18mm sized Amplatzer Septal Occluder in the guidance of TTE and TEE. After the release of the device no residual shunt was seen. Position of the device in relative to valves and aorta.

**Conclusion:** Transjugular ASD closure is an alternative when the conventional approach is not possible.

**Giriş:** Azygos ven devamlılığı ile birlikte olan kesintili vena kava inferior nadir bir konjenital anomalidir. Diğer konjenital kalp hastalıkları ve sol izomerizm bu duruma eşlik edebilmektedir. Kesintili vena kava inferiorlu bir olguda juguler ven yolu kullanılarak perkütan yolla ASD kapatılma işlemi tartışılmıştır.

**Olgu:** Üfürüm nedeniyle kliniğimize refere edilen 6 yaşında kız hastanın, fizik muayenesinde sol üst sternal kenarda 2/6 sistolik ejeksiyon üfürüm, elektrokardiografisinde inkomplet sağ dal bloğu, telekardiografide hafif kardiyomegali saptandı. Ekokardiyografik incelemede 16 mm genişliğinde sekondum ASD belirlendi. Perkütan yolla ASD' si kapatılması planlanan hastada; femoral vene kontrast madde verildiğinde geniş azygos ven devamlılığı gösterildi. Femoral ven yolu ile perkütan ASD kapama işleminde başarı sağlanamadı. ASD kapama işleminin juguler ven yolu ile yapılması planlanarak Sağ juguler venden vena kava superior yolu ile sağ atriya, ASD yolu ile sol atriya girildi. Kılavuz telin pulmoner venlere sabitleştirilememesi üzerine taşıma sistemi sol ventriküle ilerletildi. TEE, TTE ve floroskopi rehberliğinde 18 mm Amplatzer Septal Oklüderin sol diski mitral kapaktan uzakta olacak şekilde sol atriyumda açıldı. Sağ disk açılmak istendiğinde cihazın kobra hareketi nedeniyle, işlem tekrarlandı. İkinci denemede cihazın septuma paralel konumlandırılması sağlandı. İşlem sonrasında serbest bırakılan cihaz üzerinde şant saptanmadı, cihazın kapaklar ve aorta ile olan konumu normaldi.

**Sonuç:** Azygos ven devamlılığı ile birlikte olan Kesintili vena kava inferior olgularında, perkütan ASD kapama işleminin, juguler ven yolu kullanılarak yapılabileceği rapor edildi.

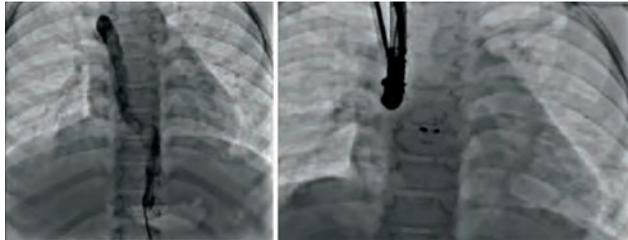


Figure 1

### A case of bicuspid aortic valve with aneurysm of sinus of valsalva

#### Sinüs valsalva anevrizmasının eşlik ettiği enfektif endokardit ile başvuran biküspid aort kapağı olgusu

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**Introduction:** Sinus of valsalva aneurysm is a rarely observed lesion; course of with includes localized thinning or enlargement of the aortic wall distinct from the diffuse dilatation observed in bicuspid aortic valve or Marfan syndrome. It can lead to severe heart failure cases commensurate with the shunt size by rupturing into all most of the heart chambers. While it may present with infective endocarditis, which one is the cause or the outcome often cannot be identified. The case represented presented at our clinic due to syncope, and was diagnosed with sinus of valsalva aneurysm while being investigated for infective endocarditis developing on the basis of bicuspid aortic valve.

**Case:** A 17-year old female patient presented at our clinic with complaints of fatigue for a year and four short-term fainting incidents over the past week. The patient, who had swelling in the right leg for a month, accompanied by severe pain and inability to step on it with no erythema, and occasionally a fever up to 38°C, was identified with 2 nodules with a diameter of 1 cm between the 3rd and the 4th distal phalanges of her right hand via physical examination. Also, 4/6 systolic ejection murmurs accompanied by thrills were heard on the aorta and the mesocardiac focus. The liver was palpable by 1.5 cm. The laboratory results yielded a low hemoglobin (10.6 g/dL), and high a sedimentation (120 mm/hour), c-reactive protein (CRP) (32 mg/dL) and rheumatoid factor (RF) values. Echocardiography showed bicuspid aortic valve, moderate aortic stenosis (gradient: 41 mmHg), mild aortic failure, and sinus of valsalva aneurysm. A three-dimensional transesophageal echocardiography was performed as the patient had inadequate transthoracic images, which revealed 5.5x5 cm of sinus of valsalva aneurysm and vegetation on the aortic valve. Antibiotic treatment was initiated for infective endocarditis. The acute phase reactants were normal again during the 6th week of treatment. As a result of the discussions with the Department of Cardiovascular Surgery, the patient received aortic valve replacement (bioprosthesis), sinus of valsalva aneurysm with an autologous pericardial patch repair, and reconstruction of the ascending aorta. The patient was discharged after the operation with proposed warfarin therapy for 6 months. We concluded that sinus of the valsalva aneurysm may have developed on the basis of endocarditis in this patient. Development of the aneurysm may have also been facilitated by the pathological changes in the aortic valve media presenting with bicuspid aortic valve. Surgical intervention is necessary to prevent sudden ruptures and complications.

Sinüs valsalva anevrizması nadir görülen biküspid aort kapağı ya da Marfan sendromundaki diffüz dilatasyondan farklı olarak aort duvarında ilokal incelleme ve genişleme ile seyreden bir lezyondur. Hemen tüm kalp odacıklarına rüptüre olarak şantın büyüklüğü ile orantılı ciddi kalp yetersizliği bulgularına neden olabilir. Enfektif endokardit ile birlikte görülebilmekle birlikte hangisinin neden hangisinin sonuç olduğu çoğu kez belirlenemez. Kliniğimize, senkop nedeni ile başvuran, biküspid aort kapağı zemininde gelişen enfektif endokardit araştırılırken sinüs valsalva anevrizması saptanan bir olgumuzu sunuyoruz.

**Olgu:** On yedi yaşında kız hasta, bir yıldır çabuk yorulma ve son bir haftada dört kez kısa süreli bayılıma yakınması ile başvurdu. Bir aydır sağ bacağına şişlik ve kızarıklık eşlik etmediği, şiddetli ağrı, basamama ve ara sıra 38°C'ye varan ateşi olduğu da öğrenilen hastanın, fizik incelemesinde, sağ elinin 3-4. parmak distal falanklarında 1 cm'lik 2 tane nodül vardı, aort ve mezokardiyak odakta, trilin eşlik ettiği, 4/6 sistolik ejeksiyon üfürümü duyuluyordu. Karaciğer 1,5 cm ele geliyordu. Laboratuvar incelemesinde; Hb:10,6 g/dl düşük, sedimentasyon (120 mm/saat), CRP (32 mg/dl) ve RF değerleri yüksek bulundu. Ekokardiografisinde (EKO), biküspid aort kapağı, orta derecede aort stenozu (gradient: 41 mmHg), hafif derecede aort yetmezliği ve Sinüs Valsalva anevrizması görüldü. Transtorasik görüntüleri yetersiz olan hastaya üç boyutlu transözofageal EKO yapıldı, sinüs valsalva anevrizmasının boyutları 5,5x5 cm ölçüldü ve aort kapağı üzerinde vegetasyonları görüldü. Enfektif endokardite yönelik antibiyotik tedavisi başlandı. Tedavinin 6. haftasında akut faz reaktanları normale döndü. Kalp Damar Cerrahisi ile yapılan konsey sonucunda, hastaya aort kapak replasmanı (bioprotez), otolog perikard yama ile sinüs valsalva anevrizma onarımı ve asendan aorta rekonstrüksiyonu uygulandı. Hastamız operasyon sonrası 6 ay süreyle kumadin tedavisi kullanması önerilerek taburcu edildi. Sonuç olarak; sinüs valsalva anevrizmasının bu hasta da endokardit zemininde gelişmiş olabileceğini düşünüyoruz. Ayrıca biküspid aort kapağı ile birlikte giden aort duvarı mediasının patolojik değişiklikleri de anevrizma gelişimini kolaylaştırmış olabilir. Ani rüptür ve komplikasyonların önlenmesi açısından cerrahi tedavi gereklidir.

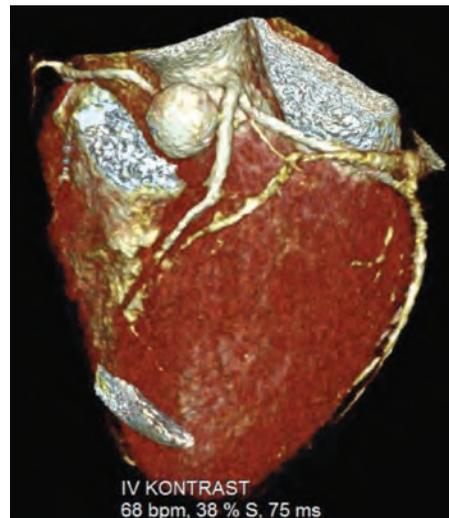


Figure 1. Aneurysm of sinus of valsalva.

OS-47

### Application of extracorporeal membrane oxygenation during SynCardia total artificial heart implantation

#### SynCardia total yapay kalp nakli sırasında ekstrakorporeal membran oksijenasyonu kullanılması

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**Background:** The SynCardia Total Artificial Heart (SynCardia Systems Inc, Tuscon, Arizona) TAH-t has been implanted worldwide as a bridge to transplantation in many centers. The SynCardia TAH-t is apneumatic pulsatile blood pump implanted to the pericardial cavity, replacing both ventricles and four heart valves. Especially 6-kg Freedom driver and the Remote Monitoring that permits hospital discharge.

**Methods:** 61-year-old gentleman had myocardial infarction and PTCA, stent implantation at another hospital mid-June 2011. He admitted to our hospital with severe biventricular failure with INTERMACS level I-II. His cardiac catheterization was reported as; PAP was 74/30 mmHg, PCWP was 30 mmHg, CI was 1.3L/min/m<sup>2</sup>, and his EF was 15%. He had TAH implantation at March 2011. While weaning from cardiopulmonary bypass, the TAH-t was allowed to take over patient's circulation. After single strokes, a significant amount of plasma like fluid was noted in his endotracheal tube. Despite the addition of positive end-expiratory pressure and maximization of ventilator support. The patient continued to have large amount of fluid in his endotracheal tube with PaCO<sub>2</sub> of > 80 and PaO<sub>2</sub> < 40 mmHg. The patient was re-heparinized, extracorporeal membrane oxygenation circuitry was connected to cannulas via right and left femoral veins. The ECMO circuit consisted of Centrimag Blood Pump (Levitronix, Waltham, MA). ECMO support was run 2.5 to 4.0 L/min with target TAH-t support 1.5 to 2.5 L/min (Fig-1). The patient's arterial blood gases and hemodynamic parameters progressed. His lungs improved and ECMO support was weaned at post-op 7th day (Fig-2,3). He was supported with TAH-t for more than 30 days and died from multi-organ failure.

**Conclusion:** The preoperative presence of pulmonary edema combined with cardiopulmonary bypass may have contributed to lung injury at the alveolar level. The use of ECMO during implantation of a TAH-t may improve patient outcomes, provide oxygenation and a safe circulatory support in emergent cases of hemodynamic instability and respiratory failure.

OS-48

### Heart assist 5 left ventricular assist device experince in Turkey Türkiye'de Heart assist 5 sol ventrikül destek cihazı deneyimi

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**Background:** Left ventricular assist device (LVAD) implantation had been reported as an effective therapy in supporting cardiovascular circulation and end-organ function for weeks to years. Increased used of LVAD therapy for patients with advanced heart failure has shown us a new paradigm for this new physiology.

**Methods:** From December 2011 to February 2013, Heart Assist 5 (Micromed DeBakey, Houston, Texas) LVADs were implanted in six patients at our hospital (Fig-1,2). All of them had ischemic cardiomyopathy. The mean age of the patients was 53±13 (34-64) years. All the patients were followed up with Remote Monitoring System from smart phones or computers (Fig-3) Through December 2011, 43 serial two-dimensional TTE had been performed on these patients, with Vivid 3 (General Electric, Fairfield, Connecticut), 15 serial three-dimensional TTE have been done with Philips IE33 xMATRIX (Royal Philips Electronics, Amsterdam, Netherlands), 6 TEE echocardiograms were done in the operating room under general anesthesia during LVAD implantation to evaluate inflow cannula position, septum position and also for de-airing process while weaning from cardiopulmonary bypass. The specific protocol used for these echocardiographic studies included standart TTE parasternal, apical, subcostal and suprasternal notch views (Fig4,5,6). In-hospital during pre-implant period; to evaluate intracardiac structure PFO, ASD, chamber dimensions, LV and RV function, valvular structure and function, pericardial disease, volume status, and abnormalities of aorta, in-hospital during post-implant LVAD period.

**Results:** The pre-implant echocardiographic data of the 6 patients; the mean EF, 23±5 (18-28)%, the mean LVEDD was 6.9±0.6 (6.3-7.7)cm, the mean LVESD was 5.8±0.5 (5.1-6.4)cm, the mean IVS was 0.9±0.1 (0.9-1.1)cm, the mean RVFAC was 43±9 (35-55)%. The post-implant 3rd month echocardiographic measurement of the 6 patients were; the mean EF was 19±6 (10-25)%, the mean LVEDD was 6.4±0.4 (6.1-7.0)cm, the mean LVESD was 5.6±0.3 (5.2-6.0)cm, the mean RVFAC was 35±11 (21-43), the mean TAPSE was 13±2 (11-16)mm, the mean rpm 9800±600 (9500-10400)rpm, the mean pulsatility index (PI) was 2.79±1.7 (1.9-4.9)m/sn, outflow cannula velocity 0.9±0.2 (0.8-1.2)m/sn, the mean cardiac out-put was 4.6±0.6 (4.1-5.5) L/min, the mean power was 7.2±2.1 (6.8-9.2) watts, aortic valve opening was observed every 1to1 cycle or 1to3cycle. All six Heart Assist 5 LVAD patients are still on LVAD support. Four patient is status post-LVAD more than 12nd months, 2 patients are status post-LVAD 8th month, and the other 1 patient is status post - LVAD 4th months with a good quality of life and waiting for a donor heart.

**Conclusion:** This study is an observational analysis of a new center with a new generation pump. The remote monitoring of this pump from smart phones and home computers, decreases hospital admission of the patients and helps the physicians to follow-up their patients.

OS-49

### Diagnosis of subvalvular membran by different imaging modalities

#### Farklı tanı yöntemleri ile subvalvuler membran tanısı

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Subaortic stenosis is likely an acquired cardiac disorder which requires anatomic precursors at level of LVOT and a genetic background. It is a cause of LVOT narrowing. It poses a serious threat not only because of the obstruction, but also because of its associated risks of infective endocarditis, aortic insufficiency and severe left ventricular hypertrophy. A 40-year old woman was admitted to our hospital with a 4 month history of dyspnea (NYHA class III). The patient had no cardiovascular disease risk factors. On cardiac auscultation, heart sounds were soft and 1-2/6 early diastolic murmur and 3/6 systolic ejection murmur were heard in the mezocardiac area. The respiratory sounds on auscultation were normal. The electrocardiogram showed sinus rhythm. Chest x-ray was normal. The transthoracic echocardiogram (TTE) showed severe aortic insufficiency, bicuspid aortic valve, normal ejection fraction. Besides, TTE and Real-time three-dimensional echocardiography demonstrated an increase in thickness of interventricular septum 10 mm below the aortic valve (Figure 1, 2). Continuous-wave Doppler echocardiography showed a 31 mmHg peak left ventricular outflow tract (LVOT) gradient. Transesophageal echocardiography was performed for a detailed examination of LVOT. The exam revealed a subaortic membrane measured 15×10 mm, extending from interventricular septum to LVOT (Figure 3). Cardiac magnetic resonance imaging (CMR) also showed a sickle-shaped, subaortic membrane, similar to that observed by echocardiography (Figure 4). In the present case, surgical treatment was considered but the patient refused. We believe this case illustrates the complimentary roles that can be played by echocardiography and CMR in the care of patients with heart disease.



Figure 1. TTE showed an increase in thickness of interventricular septum (Ao: aorta, LA: left atrium, LV: left ventricle).

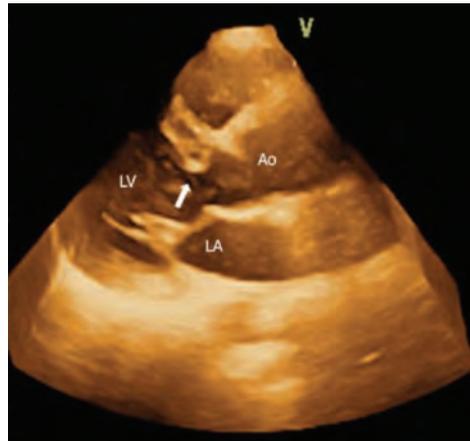


Figure 2. 3D echocardiography showed an increase in thickness of interventricular septum (Ao: aorta, LA: left atrium, LV: left ventricle).



Figure 3. TEE showed a subaortic membrane measured 15×10 mm. (Ao: aorta, AOV: aortic valve, LA: left atrium, LV: left ventricle).

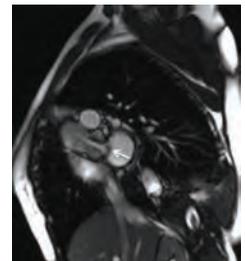


Figure 4. CMR showed a sickle-shaped subaortic membrane.

OS-50

### Complex coronary artery fistula between distal branches of the left anterior descending coronary artery and a branch of celiac trunk *Sol ön inen koroner arter distal dalları ile çöliak trunkusun dalları arasında kompleks koroner arter fistülü*

Lale Dinç Asarcıklı<sup>1</sup>, Melih Ereren<sup>2</sup>, Habibe Kafes<sup>1</sup>, İbrahim Onur Alici<sup>3</sup>, Sarper Ökten<sup>2</sup>, Orhan Maden<sup>1</sup>, Omaç Tüfekçioğlu<sup>1</sup>

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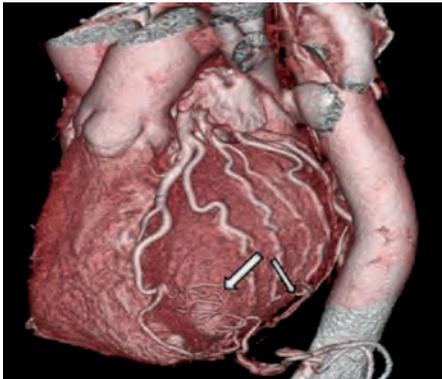
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The patient is a 42 year-old-male with severe chronic obstructive pulmonary disease. He admitted to our lung transplantation center for preoperative evaluation and during the course, the routine computed tomography-angiography of the coronary arterial system revealed an arterial anastomosis between the distal branches of the left anterior descending artery and the celiac trunk. The conventional coronary angiography and bilateral ventriculography were unremarkable. He denied any complaints related to the condition and a past medical history for any cardiac disease. Although the condition doesn't have a clinical importance, we have reported the case because of the rarity of such a condition. During lung transplant surgery, surgeon also shall be aware of the anastomosis and take care of the arterial integrity.



**Figure 1.** Arterioarterial anastomose was shown (arrow) at anteroposterior position between distal branches of left anterior descending artery and a branch of celiac trunk.



**Figure 2.** Arterioarterial anastomose was shown (arrow) at left anterior caudal position between distal branches of left anterior descending artery and a branch of celiac trunk.



**Figure 3.** Arterioarterial anastomose was shown (arrow) at left posterior caudal position between distal branches of left anterior descending artery and a branch of celiac trunk.

OS-51

### Congenital coronary artery fistula on intercoronary communication between left main and diagonal branch of the left anterior descending coronary artery: an interesting case report

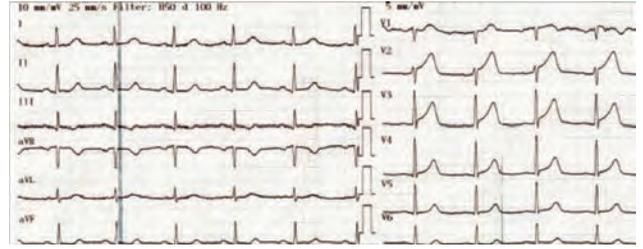
*Sol ana koroner arter ve sol ön inen koroner arterin diyagonal dalları arasında interkoroner ilişki ile konjenital koroner arter fistülü: İlginç bir olgu sunumu*

Yasin Türker, Yusuf Aslantas, Hakan Tibilli

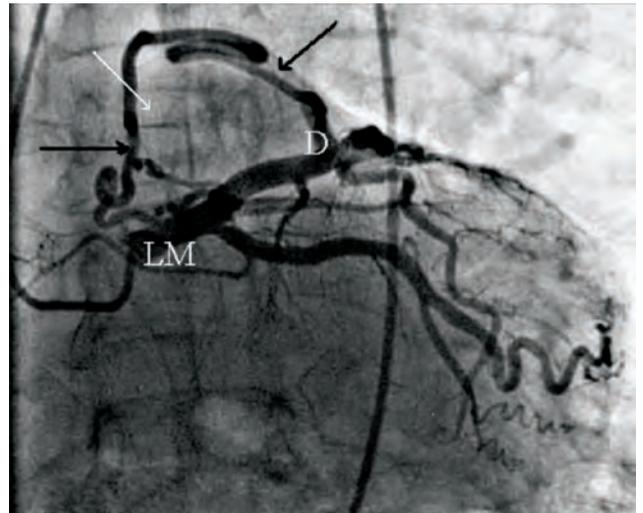
Department of Cardiology, Duzce University, Duzce

**Background:** Intercoronary communication is a very rare coronary artery anomaly. It is defined as an open-ended circulation with bidirectional blood flow between two coronary arteries. Coronary artery fistulas are abnormal communications between a coronary artery and a cardiac chamber or major vessel.

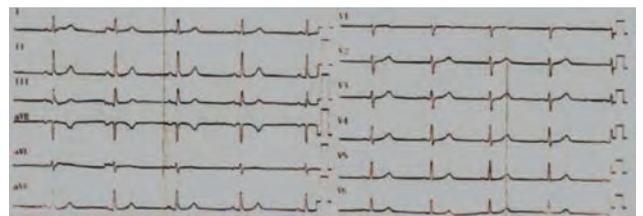
**Case report:** A 62-year-old man was admitted to our hospital with sudden development of general weakness, dizziness and a sense of compression on his chest. He had previous history of diabetes mellitus. At presentation blood pressure 80/40 mmHg and heart rate was 65 beats/min. The ECG revealed sinus rhythm and 1-2 mm ST elevation at anterior leads (Figure 1). Patient was taken to the catheterization laboratory for percutaneous coronary intervention. Left main, left circumflex coronary arteries were normal. Coronary angiography showed a communication between the left main and diagonal branch of LAD and fistula between intercoronary connection and left atrium (Figure 2). The other coronary arteries were normal. The laboratory test results, including cardiac troponin I and creatine kinase-MB levels were normal. The angina symptoms were gone and ST elevation resolved within 4 hours (Figure 3). To the best of our knowledge, we report the first case in the literature involving a congenital coronary artery fistula on intercoronary communication. We believe that intercoronary communication between left main and diagonal branch of the left anterior descending coronary artery and congenital coronary artery fistula may be the cause of ischemia in this case.



**Figure 1.** The electrocardiography revealed 1-2 mm ST elevation at anterior leads.



**Figure 2.** Injection of the left coronary artery showed a large connection between the LM and diagonal branch of LAD (black arrow) and fistula between intercoronary connection and left atrium (white arrow).



**Figure 3.** The electrocardiography demonstrated normal sinus rhythm.

## Giant left atrial mass stemming from pulmonary vein in a patient with lung adenocarcinoma and significant improvement after chemotherapy-radiotherapy combination

**Akciğer adenokarsinomu olan bir hastada pulmoner venden köken alan dev sol atriyal kitle ve kemoterapi-radyoterapi kombinasyonu sonrası belirgin iyileşme**

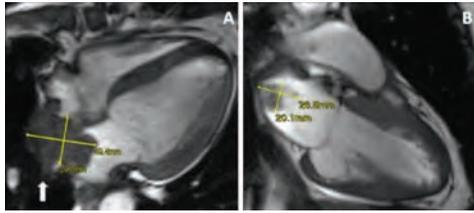
Fatih Besiroğlu, Fuad Samadov, Halil Atas, İbrahim Sarı

Marmara University School of Medicine, Department of Cardiology, Istanbul

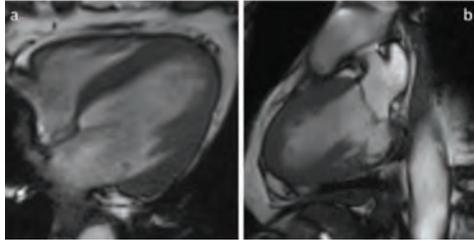
**Introduction:** Primary tumors of heart are very rare and most of them are metastatic. We present a very rare case with left atrial extension of lung adenocarcinoma stemming from the right upper pulmonary vein and subsequent marked regression of the mass with combined administration of chemotherapy and radiotherapy.

**Case Presentation:** A 66 years-old male presented with the complaints of fatigue, weakness, hemoptysis for 2 months and shortness of breath for the last 2 weeks. His physical examination and ECG was unremarkable. Transthoracic and transesophageal echocardiographic evaluation revealed moderate depression of left ventricular function with ejection fraction of %35-40 and a left atrial lobulated mass 30x25mm in diameter, compression of the mass to the superior vena cava and mild mitral regurgitation (Figure 5, Figure 6). Thoracic computer tomography demonstrated 52x36mm of mass in the right lung hilum, atelectasis distal to the mass and same sided pleural effusion which were consistent with lung carcinoma and a suspicious extension of a mass to the left atrium via right upper pulmonary vein. Bronchoscopic evaluation and pathologic investigation confirmed the diagnosis of lung adenocarcinoma. PET-CT scan with F-18 FDG revealed increased uptake in mediastinum consistent with pulmonary arterial, venous and left atrial involvement (Figure 3). Cardiac MRI confirmed the findings in echocardiography (Figure 1). The patient was considered inoperable and cisplatin and taxotere chemotherapy in combination with radiotherapy was initiated. Five months later significant decrease in size of the mass was observed (Figure 2, Figure 4).

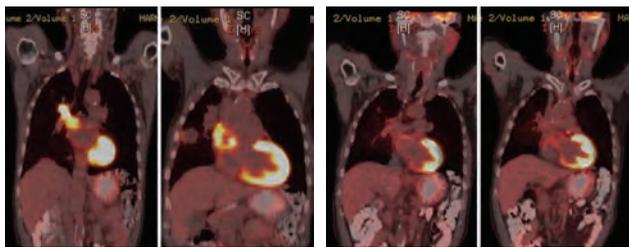
**Discussion:** To date, very few patients were reported with lung adenocarcinoma, its transvenous metastasis to the heart chamber and significant regression of the metastatic mass with combination of chemotherapy and radiotherapy. This case is a nice example of integrated multimodality imaging in order for timely diagnosis and appropriate management of a clinical scenario.



**Figure 1.** Four-chamber (a) and two-chamber (b) steady-state free precession images showing the tumour mass with maximal dimensions of 49x29mm originating from the right upper pulmonary vein.



**Figure 2.** Repeat cardiac MRI images showing nearly complete regression of the tumour mass following chemoradiotherapy.



**Figure 3.** PET-CT images showing cardiac involvement before chemoradiotherapy.

**Figure 4.** PET-CT images showing regression in cardiac involvement after chemoradiotherapy.



**Figure 5.** Subcostal echocardiographic image showing the pulmonary origin of the mass.



**Figure 6.** Transesophageal image of the mass before chemoradiotherapy.

## A rare combination: biventricular myocardial noncompaction and atrial septal defect

**Nadir bir kombinasyon: Biventriküler miyokardiyal "noncompaction" ve atriyal septal defekt**

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**Introduction:** Myocardial non-compaction (MNC) is recently described a rare type of congenital cardiomyopathy. The appearance of spungy myocardium due to the excessive trabeculation is the diagnostic finding. The apical and middle segments occupied more often. It is tend to occupy left ventricular but combination or isolated right ventricular involvement is also reported. Also cases with complex congenital cyanotic heart defects has been reported. Our aim is to report a case with biventricular MNC with secundum atrial septal defect (ASD) and discuss the subject briefly.

**Case:** A 31 year-old female patient who has shortness of breath with exercise for three months has referred to our hospital. On her physical examination, the blood pressure was 90/60 mmHg, pulse rate was rhythmic with 96 bpm; on her respiratory examination her sinuses were opened, she has bilateral rales on basal levels of the lungs; on her cardiovascular examination there was a 1/6 systolic murmur on mitral area. Other systemic examinations were unremarkable except for +1 pitting bilateral pretibial oedema. On her ECG sinus rhythm, normal axis, pulse rate of 71 bpm, pathological q wave on her III and aVF leads; T wave inversion on I, aVL, V4-6; quadrigemine VPC was observed. On her telecardiography cardiac index was increased in favor of the heart. Her transthoracic echocardiography showed left ventricular (LV) lateral wall, mid and apical inferior, apicoseptal and mid posterior segmental spungy trabecular increase which suggests MNC. (Figure 1). LV was moderate global hypokinetic (EF: % 35). On her transesophageal echocardiography; her right ventricular trabeculation was also found higher than expected, biventricular MNC and atrial septal defect (ASD) was confirmed (Figure 2-3). Left and right heart catheterization was performed. Left ventriculography was consistent with MNC (Figure 4). Qp/Qs was calculated 1,7. To confirm the diagnosis cardiac MRI was performed. LV appearance was evaluated in favor of MNC. And 5 mm defect with left to right shunt in interatrial septum was also found (Figure 5-6). Due to the sudden death probability an intracardiac defibrillator (ICD) was implanted. Then surgical repair of the ASD was agreed.

**Discussion:** MNC occurs due to the myocardial evolutionary standstill during the embryogenesis. Occupies mostly LV, but biventricular and rarely only right ventricular involvement had been reported. However the normal right ventricle also shows hipertrabeculation, thus the diagnosis of right ventricular MNC must be considered carefully. MNC can be seen with complex congenital cyanotic heart defects. There is no literature about the combination with ASD. Thus this has been recognized a case worth to publish.

**Giriş:** Miyokardiyal "noncompaction"(MNC), son yıllarda tanımlanmış, nadir görülen doğumsal bir kardiyomyopati türüdür. Miyokarda süngerimsi bir görünüm veren aşırı trabekülasyon tanı koydurucu bulgusudur. Daha çok apikal ve kısmen de orta segmentleri tutmaktadır. Daha çok sol ventrikül tutma eğilimindedir; ancak kombine veya izole sağ ventrikül tutulumu da bildirilmektedir. Kompleks doğumsal siyanotik kalp defektleri ile birlikte bulunan olgular bildirimiştir. Amacımız, her iki ventrikülü tutan ve birlikte sekundum tipi atriyal septal defekt (ASD) bulunan bir MNC olgusunu sunmak ve bu olgu nedeniyle konuyu kısaca tartışmaktır.

**Olgu:** 31 yaşında kadın hasta üç aydır eforla olan nefes darlığı şikayeti ile başvurdu. Son 1 haftadır günlük işlerini yaparken bile nefes darlığı olmaktadır. Fizik muayenesinde, kan basıncı 90/60 mmHg, nabız dakikada 72 ve ritmik bulundu. Solunum sistemi muayenesinde, sinüsler açık; her iki akciğer bazalinde yaş raller mevcuttu. Kardiyovasküler sistem muayenesinde mitral odakta 1/6 şiddetinde sistolik üfürüm duyuldu. Batın muayenesinde özellik yoktu. İki tarafı, I(+), pretibial ödem saptandı. EKG'de, sinüs ritmi, aks normal, hız 71, III ve aVF'de patolojik Q; I, aVL, V4-6da negatif T, quadrigemine ventriküler erken vuru saptandı. Teleröntgenogramda kardiyotoraksik indeks kalp lehine artmış bulundu. Transtoraksik ekokardiyografide, sol ventrikül (SV) lateral duvarında, mid ve apikal inferior, apikal septal ve mid posterior segmentlerde MNC düşündürden süngerimsi trabekülasyon artışı saptandı. (Şekil 1). SV global olarak orta derecede hipokinetik; SV EF % 35 bulundu. Transözofajiyal ekokardiyografide, sağ ventrikül trabekülasyonunda da beklenden fazla olduğu görüldü, biventriküler MNC ve atriyal septal defekt tanıları teyid edildi (Şekil 2 ve 3). Sağ ve sol kalp kateterizasyonu yapıldı. LV grafi MNC ile uyumlu idi (Şekil 4). Qp/Qs 1.7 bulundu. Tanıyı doğrulamak için kardiyak MR yapıldı. SV görüntüsü MNC lehine değerlendirildi. Ayrıca interatriyal septumda 5 mm defekt ve soldan sağa akım saptandı (Şekil 5 ve 6). Ani ölüm olasılığı nedeniyle ICD takıldı. ASD'nin daha sonra cerrahi yolla kapatılması kararlaştırıldı.

**Tartışma:** MNC, embriyogenez sırasında miyokard gelişiminin duraklaması sonucu oluşur. Çoğunlukla sol ventrikülü tutar; ancak, her iki ventrikülü ve daha nadir olarak yalnızca sağ ventrikülü tutulduğu olgular bildirilmiştir. Ancak sağ ventrikül normalde de hipertrabekülasyon gösterdiği için sağ ventriküler MNC tanısının daha dikkatli konulması gerekir. MNC kompleks siyanotik kalp defektleri ile birlikte görülebilmektedir. Atriyal septal defekt ile kombinasyonuna literatürde rastlanmamıştır. Bu nedenle olgu yayınlanmaya değer bulunmuştur.



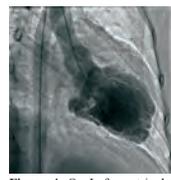
**Figure 1.** Transthoracic echocardiography showed left ventricular spungy trabecular increase.



**Figure 2.** Transesophageal echocardiography; her right ventricular trabeculation was also found higher than expected, biventricular MNC and atrial septal defect.



**Figure 3.** Transesophageal echocardiography; her right ventricular trabeculation was also found higher than expected, biventricular MNC and atrial septal defect.



**Figure 4.** On Left ventriculography, myocardial noncompaction view.



**Figure 5.** On Cardiac MRI, left ventricular MNC view and 5 mm defect at interatrial septum.



**Figure 6.** On Cardiac MRI, left ventricular MNC view and 5 mm defect at interatrial septum.

OS-54

### Right ventricular metastasis of lung cancer: Persistent ST-segment elevation and constrictive physiology

#### Akciğer kanserinin sağ ventriküler metastazı: Israrıcı ST segment elevasyonu ve konstriktif fizyoloji

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**Introduction:** Myocardial metastasis of lung cancer is rare. Although secondary cardiac tumors have been found in 10.7%–25% of autopsy cases with malignant tumors, antemortem diagnosis is not easily. We present a patient with squamous cell carcinoma of the lung with heart metastasis and constrictive pericarditis-like physiology that was identified by cardiac magnetic resonance imaging (MRI), fluorodeoxyglucose positron emission tomography (FDG-PET), and right heart catheterization (RHC).

**Case Presentation:** A 65-year-old man was admitted to our clinic because of progressively worsening dyspnea. His had a history of non-small cell lung cancer which had been diagnosed 2 years ago. Physical examination was compatible with right heart failure. Electrocardiogram revealed ST elevation in leads V1–V4 (the most prominent in V2 with 3 mm elevation), inverted T waves in the inferolateral leads, and low QRS voltage in the limb leads (Fig. 1). Echocardiography demonstrated right ventricular enlargement, increased pericardial thickness and increased echogenicity of the right ventricular free wall (Fig. 2). The FDG-PET scan showed increased uptake in the right ventricular free wall and surrounding pericardium (Fig. 3). Cardiac MRI revealed tumor involvement of the right ventricular free wall and right ventricular outflow tract with increased pericardial thickness over the right ventricle and the apex (Fig. 4). The patient underwent cardiac catheterization with a preliminary diagnosis of constrictive pericarditis that demonstrated elevated and equalized end diastolic pressures in all cardiac chambers (end diastolic pressure in both ventricles, diastolic pulmonary pressure, and pulmonary capillary wedge pressure were 20 mmHg, right atrial mean pressure was 23 mmHg) without evidence of ventricular interdependence (Fig. 5). Because absence of ventricular interdependence excludes the diagnosis of constrictive pericarditis, we described the patient's clinical situation as constrictive pericarditis-like physiology. Failure of complete resection of the myocardial metastases, the high risk of postoperative mortality, and the decreased life expectancy of our patient (because of the metastases) forced us to offer palliative medical treatment with chemotherapy and heart failure treatment.

**Conclusion:** Important features in our case are the myocardial metastasis of lung cancer leading to right heart failure, its antemortem diagnosis using multimodal diagnostic tools, as well as the persistent ST-segment elevation and constrictive physiology caused by myocardial involvement.

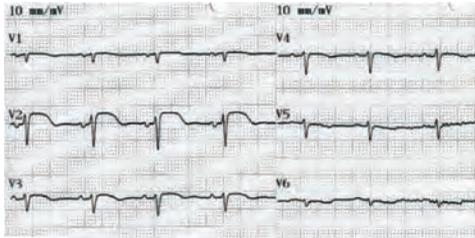


Figure 1



Figure 2

Figure 3

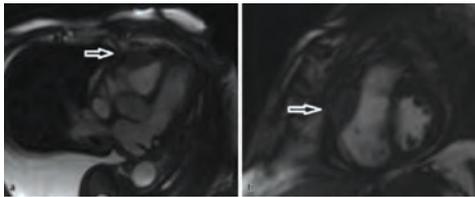


Figure 4



Figure 5

OS-55

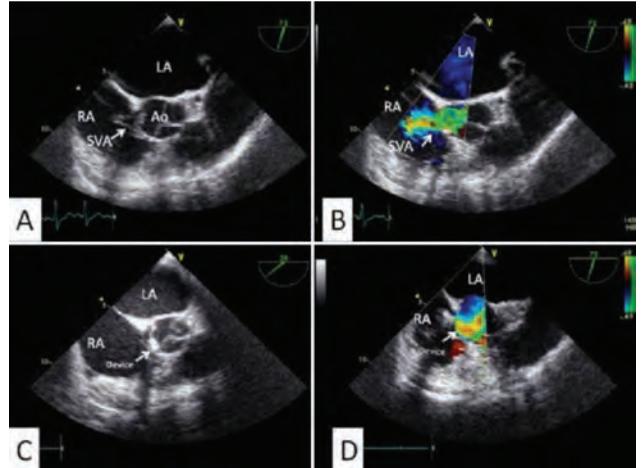
### Transcatheter closure of ruptured of sinus of valsalva aneurysm: a case from Turkey

#### Sinüs Valsalva anevrizma rüptürünün transkateter kapatılması: Türkiye'den bir olgu

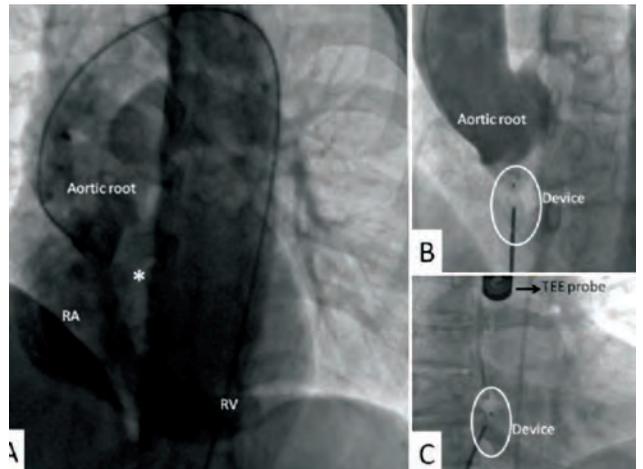
Lale Dinç Asarcıklı, Habibe Kafes, Halil Kisacik, Orhan Maden, Yesim Guray, Umit Guray, Omac Tufekcioglu

Turkey Yüksek İhtisas Hospital, Ankara

Ruptured aortic sinus of valsalva aneurysm (SOVA) is usually congenital, occurs in adolescence or in early adulthood. It can also be observed following infective endocarditis or valve surgery. Although SOVA may involve all three sinuses, the right and noncoronary sinuses are more frequently involved. The unruptured SOVA is usually asymptomatic; however, when it ruptures into one of the cardiac chambers, variable clinical conditions can happen according to the involved chamber of the heart. The conventional treatment of these aneurysms has been surgical repair with patch closure. A 44 year old woman was admitted to our hospital with the complaints of palpitation and peripheral edema. Her symptoms were worsening gradually in preceding month. Her physical examination was notable for a harsh pansystolic murmur and pretibial edema. Transthoracic echocardiography and transesophageal echocardiography (TEE), revealed a ruptured of SOVA of the non-coronary cusp, protruding into the right atrium (figure 1, panel A) with a severe shunt (figure 1, panel B), associated with enlargement of the right chambers of the heart. The patient underwent cardiac catheterization. Routine right and left cardiac catheterization were performed to obtain pressure data and to assess the magnitude of shunt and oxygen step up. Right heart catheterisation documented the oxygen step-up at the mid-level of the right atrium. Aortic root cineangiogram was performed using a pigtail catheter in various orthogonal views and showed most of the contrast material was filling the right atrium and right ventricle through the ruptured SOVA (figure 2, panel A). The defect was occluded by using a 10x8 mm PDA occluder device (figure 1, panel C-D, figure 2, panel B-C). A minimal residual shunt (figure 1, panel D) remained, with significant improvement in patient's symptoms. This case illustrates that transcatheter closure of ruptured SOVA with the help of TEE can be an effective treatment in selected cases.



**Figure 1.** Panel A and B was preprocedural, panel C and D was postprocedural aortic short axis view by transesophageal echocardiography. Ruptured of sinus of valsalva aneurysm protruding the right atrium was shown with arrow (Panel A), by colored Doppler imaging the shunt from aorta to right atrium was also seen (Panel B). After implanting the device to sinus valsalva of the noncoronary cusp (Panel C), regurgitant flow was diminished as showed by Doppler imaging (Panel D).



**Figure 2.** Panel A: Aortic root cineangiogram showed most of the contrast material filling the right atrium and right ventricle by the way of ruptured sinus of valsalva aneurysm (\*). Device was implanted and was checked by transesophageal echocardiography and anjiogram, minimally regurgitation from the defect was seen (Panel B), and the device was left in place (Panel C).

OS-56

### Multi-modality imaging of an unusual interventricular septal pouch related with left anterior descending coronary artery Sol ön inen koroner arter ile ilişkili nadir interventriküler septal kesenin çok-yöntemli görüntülenmesi

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**Introduction:** The unusual chamber at interventricular septum is a rare congenital anomaly, and has usually a fistulous communication with a coronary artery. We called this rare challenging congenital anomaly as interventricular septal pouch. In this case report, we want to emphasize the importance of multi-modality imaging in such challenging cases, especially echocardiography.

**Case report:** A 64-year-old male patient with ascending aortic aneurysm was admitted our institute for the evaluation for surgery. Interestingly, we observed a large pouch located in the apical segment of the interventricular septum. Also, we noticed an abnormal Doppler color jet flow with a predominant flow in diastole, suggestive of coronary artery blood flow in this interventricular septal pouch. We performed a CT scan of the thorax and confirmed our diagnosis. Subsequently, a coronary angiography was performed and revealed that left anterior descending (LAD) coronary artery was dilated and this rare interventricular septal pouch was related with LAD. The patient was underwent a surgical repair of ascending aorta aneurysm with a graft and pouch removal surgery. He was discharged in a well condition at the 10th hospital day.

**Discussion:** Our case emphasized the role of multi-modality imaging in the diagnosis of such rare cardiac anomalies.

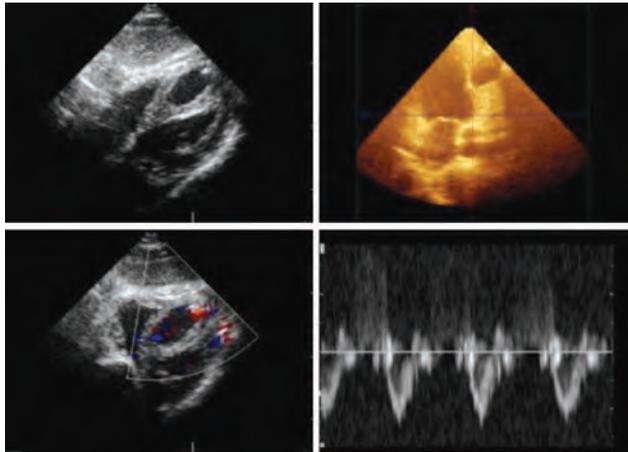


Figure 1. 2D and 3D transthoracic echocardiogram.

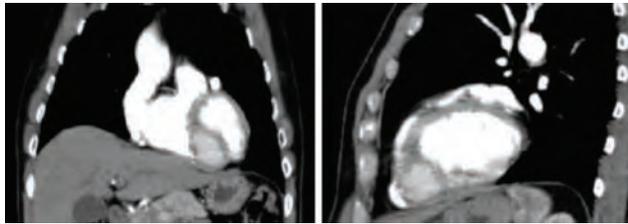


Figure 2. Computed tomography of the thorax.



Figure 3. Coronary angiography.

OS-57

### Acute pulmonary edema and pregnancy: a challenging case

#### Akut akciğer ödemi ve gebelik: İlginc bir olgu

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**Case:** A -29 year- old and 17 weeks pregnant woman was admitted to the emergency room with acute pulmonary edema. In her medical history, It was realized that she had been admitted to the emergency room with dyspnea and fever two days ago. She had been discharged with antibiotics for a diagnosis of upper respiratory tract infection. When she was consulted in the emergency room, she was tachycardic, confused, ortopneic, hypoxic and almost near to respiratory arrest. In her medical history, she had no cardiovascular disease and she had had no problem in her previous two pregnancies. Her physical examination revealed rales and rhonchi all over the chest and a diastolic murmur over the mesocardiac area. Transthoracic echocardiogram which was performed by hand-held echocardiography device (Vscan GE) showed a rheumatic mitral valve with severe stenosis (figure 1). The patient immediately was taken to coronary care unit. Upon arrival to the coronary care unit, endotracheal intubation was performed due to respiratory arrest.

What is your diagnosis?

1. Pulmonary edema due to rheumatic mitral stenosis
2. Pulmonary embolism
3. Preeclampsia followed by heart disease
4. H1N1 influenza-related pneumonia
5. Overfilling

The correct answer is #4 (H1N1 influenza-related pneumonia)

Within two hours of endotracheal intubation, the patient was in deep hypoxia despite mechanical ventilation. Approximately 600-700 cc tracheal aspirates was drained during few hours after intubation. A chest X-ray showed diffuse reticulonodular opacities in both lungs and pulmonary edema (figure 2). A chest CT scan showed a diffuse consolidations and ground-glass opacities in both lungs (figure 3). The most important finding is mitral stenosis that is disproportionate to physical examination, not to be recovered with diuretics and laboratory data findings. Real-time reverse-transcriptase polymerase chain reaction testing yielded a positive result for influenza A (H1N1). Antiflu drug oseltamivir was started immediately and patient was followed with conservative treatment. The patient was followed 6-7 days with endotracheal intubation. The patient's clinical situation and x-ray findings were gradually improved (figure 4) and she was extubated. The detailed transthoracic echocardiography revealed severe mitral valve stenosis. The patient was discharged on day 28 with total cure. Percutaneous mitral balloon valvuloplasty has been planned for severe mitral stenosis.



Figure 1



Figure 2

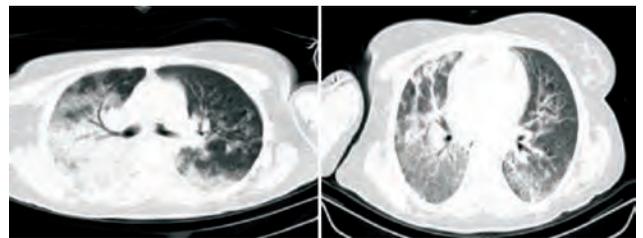


Figure 3



Figure 4

OS-58

**A case of multiple brain abscesses treated by cardiac valve surgery**  
**Kardiyak kapak cerraahisi ile tedavi edilmiş çoklu beyin absesi olgusu**

Mustafa Oylumlu, Abdulkadir Yıldız, Mehmet Ata Akil, Murat Yüksel, Nihat Polat, Mehmet Zihni Bilik, Mesut Aydın

Dicle University Faculty of Medicine, Department of Cardiology, Diyarbakir

We still don't know whether there is an optimal timing for surgery in infective endocarditis with cerebrovascular complications, as no randomized study data exist. A 33-year-old male patient referred to our clinic with the preliminary diagnosis of infective endocarditis due to one-week history of fever, vomiting, fatigue and right hemiparesis after noncardiac diagnoses were ruled out. His heart rate was 113 beats per minute, blood pressure was 100/60 mmHg and body temperature was 38.9 C. On auscultation a grade 3/6 systolic murmur was heard over the apical region. Transthoracic echocardiography (TTE) revealed vegetations on both mitral valve leaflets and severe mitral regurgitation (MR). Transesophageal echocardiography (TEE) confirmed severe MR and showed 17x10 mm and 16x11 mm vegetations on the atrial sides of the anterior and posterior mitral leaflets, respectively (Figure 1). Empirical ampicillin and gentamicin treatment was initiated. Cranial computed tomography showed 3.0 and 3.5 cm hypodense lesions on left parafalsin and left occipital lobes, respectively. For characterization of the lesions magnetic resonance imaging (MRI) was suggested by consultant neurologist. It revealed multiple cortical and subcortical lesions on the left parafalsin and bilateral occipitoparietal areas, which were surrounded by hyperintense edema zones (Figure 2). And there were small cortical lesions on bilateral frontoparietal cortices compatible with embolic infarctions showing diffusion limitation and no contrast enhancement. Staphylococcus aureus was detected on blood cultures and ampicillin was replaced with vancomycin. Because of the uncontrolled fever, vegetations and intracranial abscesses despite the medical treatment for 10 days, the valve surgery was decided by cardiology, cardiovascular surgery and neurosurgery departments. The patient underwent successful mitral repair (Figure 3) procedure and continued antibiotics for 4 weeks postoperatively. Including right hemiparesis all symptoms resolved and control cranial MRI revealed apparent regression of the lesions (Figure 4). Appropriate antibiotic therapy and early surgical treatment may reduce morbidity and mortality owing to the neurological complications of infective endocarditis.

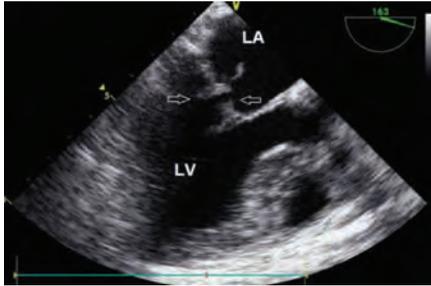


Figure 1

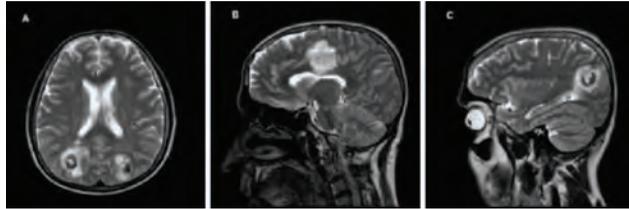


Figure 2

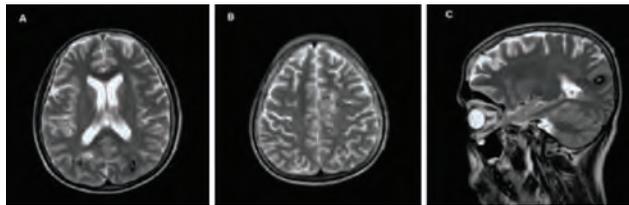


Figure 3



Figure 4

OS-59

**Transcatheter aortic valve implantation with the CoreValve for the treatment of rheumatic aortic stenosis: the first cases in the literature**  
**Romatizmal aort darlığı tedavisi için CoreValve ile transkateter aort kapak implantasyonu: Literatürdeki ilk vaka**

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**Introduction:** Rheumatic aortic stenosis (AS) is considered a contraindication to transcatheter aortic valve implantation (TAVI) because of its specific anatomic characteristics, and there is no information about TAVI in this specific population. This form of AS may increase the risks of misplacement and migration of the prosthesis. To the best of our knowledge, it is the first time the TAVI was used for rheumatic AS in the literature. Here we present two cases with severe symptomatic rheumatic AS who underwent successful TAVI with the CoreValve (CV) system because of contraindication to conventional valve replacement.

**Case:** The first case was a 72-year-old woman and admitted to our hospital for severe rheumatic AS. Her medical history included rheumatic AS and mitral valvular disease, chronic renal insufficiency, chronic pulmonary disease and operated colon cancer. The aortic valve was thickened and little calcified and there was commissural fusion (Figure 1-2). She had also 2+ aortic insufficiency with a normal left ventricular ejection fraction of 50%. In addition, she had concomitant involvement of the mitral valve, which was also moderately stenotic with a mean valve gradient of 5 mmHg an estimated valve area of 1.7 cm<sup>2</sup> (Figure 1). Echocardiogram showed an aortic valve area of 0.8 cm<sup>2</sup> and a mean aortic pressure gradient of 45 mm Hg. Systolic pulmonary artery pressure was 60 mm Hg. During deployment, to prevent pop-out, embolization and migration of the prosthesis we performed accelerated right ventricular pacing with 180 bpm. In addition, because the CV prosthesis had to anchor solidly, an oversized 29 mm CV prosthesis was selected. Implantation depth was approximately 6 mm. But, pop out of the valve occurred. After reloading and repositioning of the CV, the prosthesis could be implanted a little bit deeper in the second attempt (Figure 3). The second case was a 62 year-old woman and admitted to our hospital for severe rheumatic AS and moderate aortic regurgitation with an echocardiographic aortic valve area of 0.7 cm<sup>2</sup>, with a depressed left ventricular function of 20%. Her medical history included rheumatic heart disease, atrial fibrillation and mitral valve replacement for mitral stenosis in 1998. AS was of rheumatic cause, with commissural fusion, little calcification, and was associated with 2+/3+ aortic regurgitation. The technique was similar to that described by the previous case. An oversized (29 mm), CV were used. Implantation depth from aortic annulus to left ventricular outflow tract was approximately 6 mm (Figure 4).

**Conclusion:** This report shows that TAVI could be safe, feasible and effective treatment in patients with rheumatic AS. Pop out and embolization of the valve may become an issue, and could be a drawback to this approach. Treatment of rheumatic AS by TAVI may be off-label but a feasible treatment in selected no-option patients.



Figure 1. Transesophageal echocardiography demonstrates thickening and commissural fusion of the aortic valve with little calcification in basal short axis view in case 1.



Figure 2. Transesophageal echocardiography demonstrates concomitant rheumatic involvement of the mitral valve in long-axis view of the left ventricle in case 1.

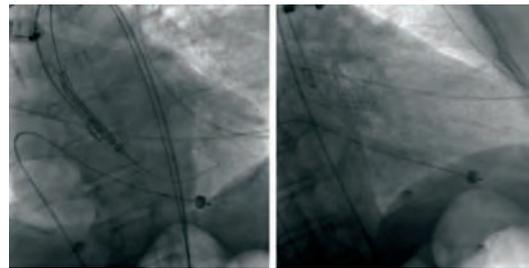


Figure 3. Positioning and deployment of the CoreValve in case 1.

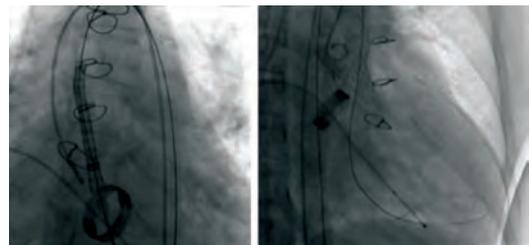


Figure 4. Positioning and deployment of the CoreValve in case 2.

OS-60

### The first experiences of transcatheter aortic valve implantation (TAVI) in Şifa University Faculty of Medicine

#### Şifa Üniversitesi Tıp Fakültesi'nden transkateter aort kapak implantasyonu için ilk deneyimler

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Transcatheter Aortic Valve Implantation (TAVI) is a new technic for treatment of high risky or inoperable patients with severe aortic valve stenosis. Herein we presented first two TAVI cases in our center. BC was 79 years old man and AK was 72 years old female with severe symptomatic aortic stenosis presented with dyspnea and angina despite medical treatment. The patients had a high risk for valvular surgery operations. STS mortality score was %16 for BC and % 14 for AK. Logistic Euroscore was %39 for BC and %23 for AK. BC underwent CABG operation after acute coronary syndrome in 2001. His aortic valve area was 0.67 cm<sup>2</sup> and gradients were 92/53 mmHg at transthoracic echocardiography (TTE). The annulus diameter was measured 24.8 mm at transesophageal echocardiography (TEE). There were moderate aortic and mitral regurgitation and moderate pulmonary hypertension (sPAP:48 mmHg). Coronary angiography revealed normal and open by-pass grafts. Iliofemoral angiography showed large calibre iliofemoral arteries (right;9.8 mm, Left 8.9 mm) The annulus to LMCA distance was measured 13.5 mm and RCA distance was 15.5 mm on aortography. AK had moderate chronic obstructive pulmonary disease (COPD) and insulin dependent diabetes. Her aortic valve area was 0.66 cm<sup>2</sup> and gradients were 72/40 mmHg. The annulus diameter was measured as 23.5 mm at TEE. There were mild aortic and moderate mitral regurgitation and moderate pulmonary hypertension (sPAP:55 mmHg). Coronary angiography revealed normal. Iliofemoral angiography showed moderate calibre iliofemoral arteries (right; 7.8 mm, left 7.2 mm). The annulus to LMCA distance was measured 11.5 mm and RCA distance was 16 mm on aortography. The conclusion from the Heart Team (HT) discussion was that, patients were found to be at high risk and HT decided transfemoral TAVI with 26 mm Edwards Sapien (ES) XT valves. Temporary pacemakers were implanted on each patient's right ventricular apex. Pigtail catheters were introduced to the ascending aorta over a regular 0.38 guide wire from right common femoral artery. Guide wires were exchanged with 0.035 Amplatz extra stiff guide wires. They had serial dilatations with different size dilators. Finally, 18F E-sheaths were placed into the right femoral arteries. Amplatz left 2 (AL2) catheters were introduced to ascending aorta over a 0.38 guide wires. The wires were exchanged with soft straight tipped wires. The catheters were pushed forward to the left ventricles. The straight tipped wires were exchanged with amplatz extra stiff wires (ESW). We used 80 and 95 ml contrast agents and two brief rapid pacing (180/min) trains to deploy a 26 mm ES XT valve under general anesthesia for each patient (Figure 1-4). We didn't use TEE. There was a mild paravalvular aortic regurgitation near BC's ES XT valve. AK hadn't any problems with her ES XT valve. They were discharged well on the fourth day after TAVI implantation with acetylsalicylic acid and clopidogrel once a daily treatment.

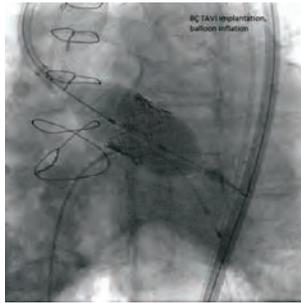


Figure 1. BC aortic valve implantation.

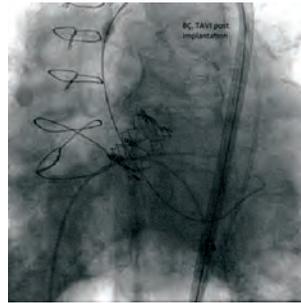


Figure 2. BC Edwards Sapien XT valve implantation.

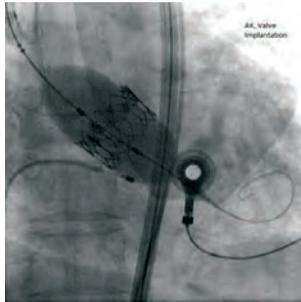


Figure 3. AK aortic valve implantation.



Figure 4. AK Edwards Sapien XT valve implantation.

OS-61

### Tricuspid valve repair for functional tricuspid regurgitation

#### Fonksiyonel triküspid yetmezliğinde kapak tamiri sonuçlarımız

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Functional tricuspid regurgitation is frequently present in patients with left heart valvular diseases that produces annular dilatation and right ventricular enlargement. Concomitant tricuspid valve repair during left heart valve surgery is recommended. All 26 patients who had tricuspid valve repair for functional tricuspid regurgitation from 2009-2012 were prospectively followed for up to 36 months (mean 20.55 ±8.95, range 6-36 months). Mean age was 61.23±13.78 (37-84). There were 18 female and 8 male patients. All patients had grade 4 tricuspid regurgitation. Clinical, hemodynamic and operative variables were evaluated for their association with operative and follow up mortality and morbidity. There were 10 (38.5%) patients who had previous cardiac surgery. Right heart failure was predominant in 20 patients (76.9%). Isolated tricuspid valve repair was performed in 4 patients (15.4%). All other patients had concomitant left heart valve surgery. Tricuspid leaflet augmentation was performed in 4 patients. Tricuspid ring annuloplasty was performed in all patients. Operative mortality was 15.4% (4 patients). High pulmonary artery pressure, low EF and concomitant aortic valve and coronary artery disease were predictors for operative mortality. Reoperation was not a significant risk factor for operative mortality. During the follow up no mortality was observed. In conclusion operative mortality is increased in patients with concomitant coronary artery disease and multiple valvular pathology. We recommend leaflet augmentation and ring annuloplasty as preferred surgical techniques.

Fonksiyonel Triküspid yetmezliği, sol kalp patolojilerine bağlı olarak triküspid kapakta anüler dilatasyon ve sağ ventrikül geometrisinde değişime neticesi ortaya çıkar. Genellikle sol kalp patolojilerinin cerrahi tedavisi sırasında eşlik eden triküspid kapak patolojisinde düzeltilmesi gerekir. Bu çalışmada 2009-2012 yılları arasında kliniğimizde gerçekleştirilen 26 triküspid kapak tamir vakası incelenmiştir. Hastaların yaş ortalaması 61.23±13.78 (37-84) olup, 18 hasta bayan 8 hasta ise erkektir. Hastaların hepsinde ciddi triküspid yetmezliği (4/4) mevcuttu. Takip oranı %70 dir. Takip süresi 20.55 ±8.95 (6-36) aydır. Hastaların ameliyat öncesi ekokardiyografik ve demografik bulguları ameliyat mortalitesi ve ameliyat sonrası fonksiyonel kapasiteleri ile karşılaştırılmıştır. Daha öncesinde kardiyak operasyon geçirmiş hasta oranı %38.5 (10 hasta), belirgin sağ kalp yetmezliği bulguları olan hasta oranı ise %76.9 (20 hasta) dur. İzole triküspid tamiri oranı %15.4 olup hastaların geri kalanında aynı süreçte ek kardiyak girişim yapılmıştır. Sekiz hastada tamir atan kalpte gerçekleştirilmiştir. Tamir yöntemi olarak 4 hastada ön kapakçık augmentasyonu ve rijid ring, diğerlerinde ise rijid ring ile anüloplasti uygulanmıştır. Ameliyat mortalitesi %15.4 (4 hasta) olup mortaliteyi etkileyen faktörler, düşük ejeksiyon fraksiyonu, yüksek pulmoner arter basıncı ve eşlik eden aort kapak ve koroner arter hastalığı olarak bulunmuştur. Reoperasyon risk faktörü olarak tespit edilmemiştir. Takip edilen hastalarda mortalite tespit edilmemiştir. Fonksiyonel triküspid yetmezliğine eşlik eden koroner arter hastalığı ve birden fazla kapak patolojisi mevcudiyetinde operatif risk artmaktadır. Öte yandan erken dönem tamir sonuçları dayanıklılık açısından iyi sonuç vermekte ve ring anüloplasti ve kapakçık augmentasyonu tercih edilen tamir yöntemleri olmalıdır kanısındayız.

## Endocarditis in mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) syndrome: the first in the literature

### Mitokondriyel nörogastrointestinal ensefalomiyopati (MNGIE) sendromunda endokardit: Literatürde ilk

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**Case:** A 19 years old female patient was admitted to our emergency clinic with the complaints of progressive dyspnea and fever for two days. In her past history, she was known to have MNGIE syndrome for three years. She was tachycardic with S3 and had a 2/6 grade systolic regurgitation murmur at mesocardiac area. In her pulmonary system examination, crepitant rales were detected on basal and mid zones of lungs. In the transthoracic echocardiography, left ventricular functions were normal but a 10x9 mm sized vegetation was detected on the atrial face of anterior mitral leaflet leading severe mitral regurgitation. Blood samples were inoculated into aerobic and anaerobic culture media. The one year old venous port was removed and samples from port, including its tip, were taken for aerobic, anaerobic and fungal cultures. In her follow up, dyspnea had ceased but on the fourth day of hospitalization, she had experienced respiratory arrest and was intubated and connected to a mechanical ventilator. She was unconscious during mechanical ventilation period and had lost her life due to cardiac arrest on the sixth day of intubation.

**Discussion:** Mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) syndromes is a rarely seen multisystem disorder with autosomal recessive inheritance due to thymidine phosphorylase gene mutation. It is characterized by progressive external ophthalmoplegia and/or ptosis, progressive gastrointestinal dysmotility and abdominal pain, postprandial emesis, cachexia, demyelinating peripheral neuropathy, symmetrical and distal weakness especially in lower extremities and diffuse leucoencephalopathy in cranial magnetic resonance. Although the symptoms commonly appear during first and second decades, it can be asymptomatic until fifth decade. In the review of literature, among MNGIE syndrome patients, our case is the first who had endocarditis, so far. In our case, the patient was examined in detail if the vegetation was infective or sterile. The echocardiography of our patient performed two years ago was normal and in the present echocardiographic examination, mitral and aortic valves were structurally normal, also, excluding any valvular etiology. After death of the patient, all of the cultures from urine, sputum and port were fallowed and examined but there was no growth in any of them. Blood cultures were incubated for four weeks but did not show any microbial growth. Although we couldn't exclude the diagnosis of culture negative endocarditis, the findings strongly propose that a sterile vegetation formed due to autoimmunity as in systemic lupus erythematosus, can be seen in MNGIE syndrome.



Figure 1. View of vegetation in the transthoracic echocardiography.



Figure 2. View of vegetation in the transthoracic echocardiography.

## Unusual foreign body in the heart

### Kalpde nadir bir yabancı cisim

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A schizophrenic 42 year old man admitted to emergency service with non specific chest pain. He was brought to the emergency department of our hospital by a relative. Inspection of the chest wall demonstrated no puncture site or no bleeding wound or insertion site and cardiovascular auscultation was unremarkable without murmurs, gallops or friction rubs. Blood pressure and pulse were 124/72 mm Hg and 92 beats/min, respectively. Blood analysis revealed elevated serum troponin I and normal urine analysis. A 12 derivation ECG disclosed no ischemic or arrhythmic finding. A chest radiography showed a linear and needle-shaped metallic density within the cardiac silhouette. Computed tomography (CT) showed the needle as metal density inserted from the left ventricular apex straight to the interventricular septum (Fig. 1). In echocardiography pericardial echogenicity was normal and pericardial fluid was not present. Left and right ventricular systolic functions were normal. Cardiac catheterization and coronary angiography planned in case of acute coronary syndrome and showed non-obstructive coronary artery disease, there was no evidence of intramyocardial hemorrhage but a radio-opaque, linear structure was seen within the heart silhouette on fluoroscopy [fig 2]. The eye of the needle was completely buried under the skin and the tip was directed towards interventricular septum via apex. Surgical removal of the needle was planned. After mid sternotomy, aortic and right atrial cannulations were performed. A sewing needle 6 cm in length was successfully removed from the apex by a clamp. The operation was completed without any complication. A penetrating cardiac injury usually causes cardiovascular compromise by hemorrhage or cardiac tamponade. Iatrogenic complications may result from breakage of a catheter tip, coronary guidewire or metal stylet. The incidence of coronary artery injury with penetrating chest wounds is low, estimated at 4 to 12%. Coronary artery injury frequently results in cardiac tamponade. Involvement of proximal coronary arteries was almost invariably fatal and survivors usually had lacerations of more distal vessels. Late sequelae of coronary artery injury are rare but include coronary arteriovenous fistula, coronary artery aneurysm and, rarely, myocardial infarction. The incidence of ventricular septal defect after penetrating heart injury is 6 to 7% and has been reported in at least 51 cases, although not as a complication of injury by a needle. On serial catheterization, most shunts remained unchanged, some diminished with time and some closed spontaneously. Most patients with a ventricular septal defect remain asymptomatic and their late prognosis is good. Ventricular aneurysm and pseudoaneurysm are also late complications of penetrating heart injury.



Figure 1. Thoracic CT. sewing needle inserted from apex to the interventricular septum.

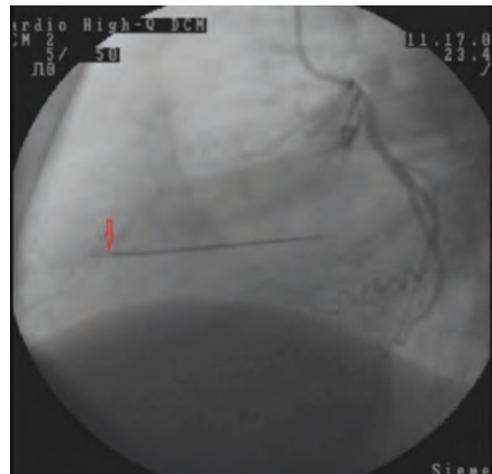


Figure 2. Fluoroscopy. Sewing needle seen on coronary angiography. Positioned from apex to the interventricular septum. Minimal coronary artery disease and no injury to the coronary arteries.

OS-64

### A rare complication after transcatheter aortic valve implantation (TAVI) by transfemoral approach: perimembranous ventricular septal defect

#### Transfemoral yolla transkateter aort kapak implantasyonu sonrası nadir bir komplikasyon: Perimembranöz ventriküler septal defekt

Hüseyin Dursun<sup>1</sup>, Barış Ünal<sup>1</sup>, Zülkif Tanrıverdi<sup>1</sup>, Abidin Cenk Erdal<sup>2</sup>, Dayimi Kaya<sup>1</sup>

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<sup>2</sup>Department of cardiovascular Surgery, Dokuz Eylül University, İzmir

**Introduction:** Transcatheter aortic valve implantation (TAVI) has been a recently established and now widely performed treatment modality for inoperable or high risk patients with severe aortic stenosis (AS). Since the technical expertise and device technology is evolving, unexpected and rare complications are reported with increasing frequency such as ventricular septal defect (VSD). Here we describe a membranous type of VSD occurred after implantation of 26 mm Edwards Sapien XT Prosthesis (Edwards Lifesciences, Irving CA). To our knowledge this is the first report describing such an unusual complication in our country and 6th in the worldwide among this type of device.

**Case:** A 73 year old woman with NYHA functional dyspnea class III-IV was referred to our institution for severe AS. Transthoracic echocardiography (TTE) of the patient showed severe calcific aortic valve with an area of 0.7 cm<sup>2</sup>, and a mean gradient of 45 mmHg. The aortic annulus diameter measured by transesophageal echocardiography was 23 mm. There was no critical stenosis at her coronary and peripheral angiography with a femoral artery diameter of 7 mm. Her logistic EuroScore was 33,8 %. So a multidisciplinary decision was made to perform TAVI from transfemoral approach. After the aortic valve dilatation with a 23 mm Balloon, a 26 mm Edwards Sapien XT Prosthesis was successfully implanted (Figure 1). The patient improved clinically in the care unit and stayed asymptomatic. However, on her fourth day, control TTE showed prominent left to right systolic shunt at membranous interventricular septum with a Qp/Qs ratio of 1.5 (Figure 2). Multislice CT demonstrated a free zone of septum about 3-4 mm further from the prosthesis and membranous type of VSD of 5 mm in diameter which was not present before TAVI (Figure 3-4a,b).

**Discussion:** VSD occurrence is an unusual complication after transfemoral TAVI. In the literature only 5 reports (one in 2009 and the others in 2013) presenting 5 patients with Edwards Sapien XT device and 2 patients with CoreValve ReValving system (Medtronic, Irvine, California) having such a complication. Most of those cases, device post balloon dilatation or oversized prosthesis implantation might be responsible and in the remaining there were no possible explanation other than excessive aortic calcification. In our patient there was only mild calcification rather than intensive calcification in aortic root and valve cusps. A 26 mm Edwards Sapien XT device was implanted and because of the proper outcome there was no need for a post-procedural balloon dilatation. Although the proper implantation of the prosthesis and in the absence of excessive calcification it was surprising to have this unavoidable VSD complication in such a patient. Although it is a serious problem, asymptomatic presentation of this complication in our patient is also intriguing and emphasizes the importance of routine careful TTE examination after TAVI.

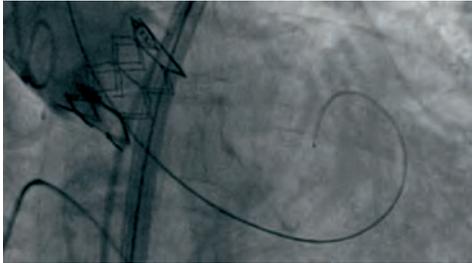


Figure 1. Aortography after the prosthesis implantation.

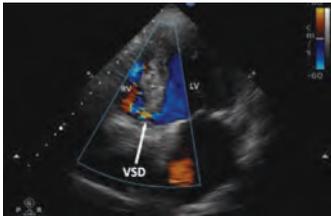


Figure 2. Transthoracic apical 4 chamber view showing left to right shunt at interventricular septum level. VSD=Ventricular Septal Defect, RV= Right Ventricle, LV=Left Ventricle.

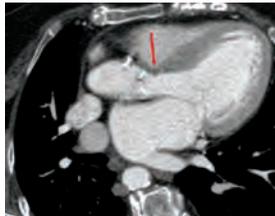


Figure 3. Cardiac CT before TAVI demonstrating intact interventricular septum.

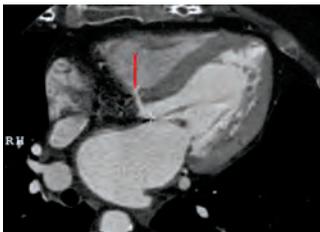


Figure 4. Cardiac CT after TAVI demonstrating ventricular septal defect.



Figure 5. Cardiac CT after TAVI demonstrating the relationship between VSD with prosthesis valve.

OS-65

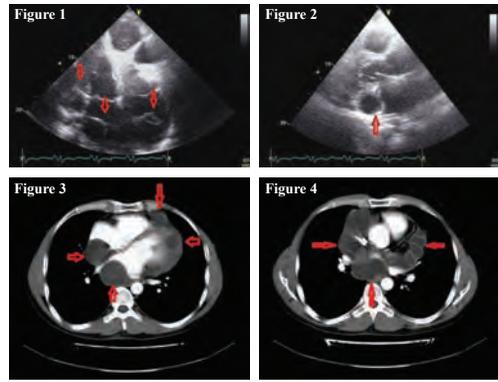
### Pan cardiac hydatid cyst

#### Pan kardiyak kist hidatik

Efial Murat Bakırcı, Kamuran Kalkan, Hakan Duman, Ibrahim Halil Tanboga, Husnu Degirmenci

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Hydatid disease is a human infection caused by the larval stage of *Echinococcus granulosus*, which is still endemic in many cattle-raising areas. Cardiac hydatid cysts are very rare, involving 0.5 to 2% of all cases, but potentially a very serious complication of the hydatid disease. The diagnosis of cardiac cyst hydatid may be difficult due to the nonspecific symptoms and varying clinical presentations. A 46-year old man who had a known previous history of hepatic cyst hydatid was admitted to our clinic because of progressive dyspnea, atypical chest pain and fatigue for two weeks. On physical examination, his respiratory rate was 18/min; heart rate was 112 beats/min and blood pressure was 110/70 mmHg. Electrocardiography showed sinus tachycardia. Transthoracic echocardiography (TTE) revealed multiple intracardiac and pericardial unilocular cystic spheric hyperechogenic masses with well-defined margins (Figure-1 and 2). A contrast-enhanced computed tomography (CT) showed two cyst in the left and right atrium and multiple cyst in pericardium and a cyst in left ventricle posterior wall (Figure-3 and 4). On the basis of these findings, the patients was referred cardiac surgery but he refused and has been followed up medically under albendazole treatment. Herein, we report a case of a pan cardiac hydatid cyst. The diagnosis was established by TTE and CT Scan. This case illustrates the diagnostic value of the non invasive imaging means in hydatid cyst of the heart.



OS-66

### To what extent can a heart chamber dilate? Gigantic right atrium in unrepaired TOF

#### Kalp boşluğu ne kadar genişleyebilir? Düzeltilmemiş TOF'a bağlı dev sağ atriyum

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A 43-year-old man with unrepaired Tetralogy of Fallot (ToF) was admitted to our institution with dyspnea on exertion and cyanosis. The diagnosis of ToF had been established in late childhood, at time when he was considered as inoperable due to advanced heart failure and pulmonary hypertension. Since then he has been followed on medical therapy on outpatient basis and finally referred to our institution for consideration for heart-lung transplantation. Transthoracic echocardiography showed a giant right atrium (GRA) (Figure 1, Panel A and B) along with the four anatomical abnormalities of ToF (Figure 1, Panel C and D). The right atrium measured 19.3x14.2 cm, and severe tricuspid regurgitation was observed in apical 4-chamber view. In this view, the left atrium was completely obscured by the GRA by mass effect which gave false impression as a three chambered heart. This GRA, the largest ever reported to our knowledge, demonstrates the extent to which a heart chamber can dilate.

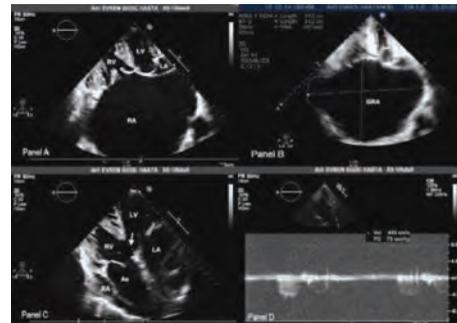


Figure 1. Transthoracic echocardiography of a patient with unrepaired ToF. Panel A: Apical four-chamber view showing a gigantic RA. Note that the left atrium was completely obscured by the GRA by mass effect which gave false impression as a three chambered heart. Panel B: The dimension of RA was measured as 19.3x14.2 cm and the planimetric area was 187 cm<sup>2</sup>. Panel C: Modified apical five-chamber view showing overriding aorta and a 2.5 cm VSD. Note that the RV is hypertrophied. Panel D: CW Doppler interrogation at RVOT revealed a gradient of 79 mmHg. CW: continuous wave, GRA: giant right atrium, LA: left atrium, RA: right atrium, RV: right ventricle, RVOT: right ventricular outflow tract, ToF: tetralogy of Fallot, VSD: ventricular septal defect.

OS-67

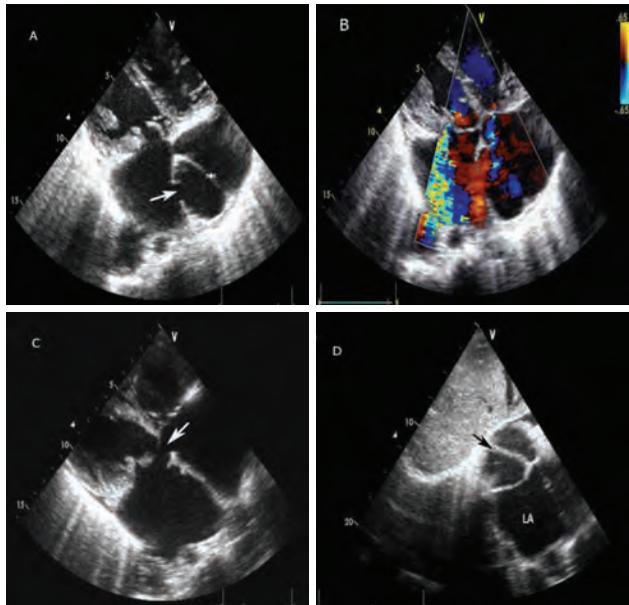
**All in one case;primum and secundum atrial septal defect, cor triatriatum dexter and pulmonary stenosis**

**Hepsi bir vakada; primum ve sekundum atriyal septal defekt, kor triatriyatım dekster ve pulmoner darlık**

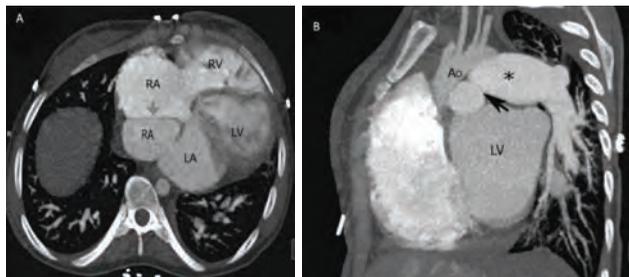
Ziya Simşek, Yavuzer Koza, Muhammed Hakan Taş, Zakir Lazoğlu, Emrah Aksakal

Ataturk University Faculty of Medicine, Department of Cardiology, Erzurum

Cor triatriatum dexter is an extremely rare congenital anomaly in which the right atrium is divided into two chambers by a membrane. It results from persistence of the right valve of the sinus venosus. The estimated incidence of cor triatriatum has been reported as 0.1% of congenital cardiac malformations (1). Cor triatriatum dexter can be diagnosed at any age, especially if it is incidentally discovered. A 22 year old woman, with no previous history of cardiac disease, presented with complaints of dyspnea, palpitation and near syncope for about two years. Physical examination evidenced a harsh systolic murmur in the pulmonary area and a 2/6 grade holosystolic murmur at the left sternal border. Her blood pressure was 100/60 mmHg and the pulse was irregular at 110 beats/min. The jugular venous pressure was increased with prominent v waves of tricuspid regurgitation. Chest X-ray revealed increased cardio-thoracic ratio. A 12-lead electrocardiogram showed incomplete right bundle branch block, atrial fibrillation with about 140 beat per minute and rare ventricular early beats. Transthoracic echocardiogram revealed following findings: primum asd, secundum asd, a membrane that divides right atrium two separate chambers, pulmonary stenosis, prominent tricuspid and mitral regurgitation (Fig. 1 A,B,C,D). Dilatation of the right heart cavities and an eccentric hypertrophy of the right ventricle were noted. The estimated systolic pulmonary artery pressure was 55 mmHg. There was no obstruction of the tricuspid valve or the inferior vena cava. The dimensions and systolic function of the left ventricle were normal To confirm the diagnosis and to exclude other associated congenital abnormalities cardiac computed tomographic imaging (CT) was performed. Cardiac CT imaging showed a membrane that separating the right atrium into an anterior and a posterior chamber with pulmonary stenosis and poststenotic dilatation (Fig. 2 A,B). In cor triatriatum clinical presentation is highly variable and dependent on the degree of septation. The management of cor triatriatum dexter depends on clinic status. Asymptomatic patients do not require treatment unless they are undergoing cardiac surgery for other reasons. Patients with obstructive symptoms need surgical or percutaneous resection of the membrane (2). We suggested surgical treatment to our patient primarily for ASD and other cardiac lesions, she and her family denied operation and she was discharged from the hospital with medical treatment.



**Figure 1.** Transthoracic echocardiography. Panel A: secundum asd (arrow) and the membran (asterisks) that divides right atrium two separate chambers. Panel B: color flow doppler image shows the left to right shunt with prominent tricuspid and mitral regurgitation. Panel C: primum asd (arrow). Panel D: subcostal view shows the membran (arrow). LA- left atrium.



**Figure 2.** Cardiac CT. Panel A: axial maximum intensity projection image shows the right atrium that divided two separate chambers (arrow). Panel B: pulmonary artery stenosis (arrow) and poststenotic dilatation (asterisks). RA- right atrium, RV- right ventricle, LV- left ventricle, Ao- aorta.

OS-68

**An unusual form of Naxos disease and its improvement by low-dose adjuvant colchicine therapy**

**Naxos hastalığının nadir bir formu ve düşük doz adjuvan kolşisin tedavisi ile iyileşme**

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**Aims:** We evaluated a female patient with an unusual form of Naxos disease, who presented with central cyanosis and clubbing, simulating congenital heart disease and whether or not use of low-dose colchicine as adjunct to conventional therapy may be beneficial in the treatment of this very rare disease.

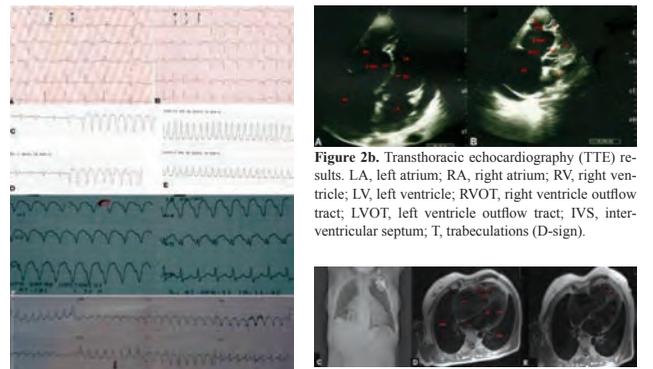
**Method:** A 54-year-old female living in the city of Trabzon in the Black Sea region of Turkey with a history of cyanosis, clubbing with intermittent exacerbations of congestive heart failure and syncope was referred to our hospital for further evaluation. At presentation, the patient was taking metoprolol 50 mg twice daily. Amiodarone perfusion protocol was started to prevent uncontrolled ventricular tachycardia. During an electrophysiologic study (EPS), the hemodynamic status of the patient deteriorated due to VT, and an ICD was inserted. At discharge, adjuvant low-dose colchicine therapy (0.5 mg once daily) has been added to conventional therapy of the patient and continued for six months.

**Results:** Physical examination revealed central cyanosis, clubbing of digits, increased jugular vein pressure, hepatomegaly, edema and ascites, woolly but no gray hair, alopecia, palmoplantar keratoderma, and squamous lesions on her arms and legs (Figure 1). ECG recordings showed ventricular beats, premature atrio-ventricular nodal reentrant tachycardia (AVNRT), accelerated AVN rhythm, atrial fibrillation (AF), and monomorphic and polymorphic episodes of ventricular tachycardia (VT). Incomplete right bundle branch block (RBBB) and epsilon waves were evident. (Figure 2 a). Echocardiography showed a dilated, hypokinetic right ventricle (RV) with prominent apical trabeculae and dilatation of the RV outflow tract. The right atrium (RA) and left atrium (LA) were also dilated. Estimated RV ejection fraction (EF) was 14%. The interventricular septum (IVS) was D sign and showed paradoxical movement. Her pulmonary artery pressure (PAP) was 30.00 mmHg and her EF was 60% with preserved LV systolic function. (Figure 2 b). Cardiac magnetic resonance imaging (MRI) has been found to accurately assess the structural and functional features of ARVD, including fibro-fatty infiltration, thinning of the RV myocardium, RV aneurysms. Cardiac MRI in our patient showed aneurysmal dilations of the diaphragmatic, apical and infundibular regions of the RV (the so called “triangle of dysplasia”), suggesting ARVD (Figure 2 c). Follow-up of low-dose colchicine therapy (0.5 mg once daily) has been added to conventional therapy showed improvement in the patient’s clinical status, with a dramatic disappearance of the electrical storm and reductions in cyanosis and palmoplantar hyperkeratosis.

**Conclusion:** The study of the present patient disclosed that Naxos disease may mimic cyanotic congenital heart disease with severe hypoxemia. In addition, use of low-dose colchicines as adjunct to conventional treatment may be beneficial in the treatment of this very rare disease.



**Figure 1.** Clinical findings in our patient before (A-F) and after six months (G-J) of treatment with colchicine.



**Figure 2b.** Transthoracic echocardiography (TTE) results. LA, left atrium; RA, right atrium; RV, right ventricle; LV, left ventricle; RVOT, right ventricle outflow tract; LVOT, left ventricle outflow tract; IVS, inter-ventricular septum; T, trabeculations (D-sign).

**Figure 2c.** Cardiac magnetic resonance images (MRI), showing fibrofatty (FF) infiltration at the apex. The arrows indicate aneurysmal dilation of the right ventricular (RV) apex and RV inflow tract; M denotes the aneurysmal dilation of the right ventricular outflow tract (RVOT), the third component of the “Marcus triangle”. RA, right atrium; IVS, inter-ventricular septum; IAS, inter-atrial septum; LVPW, left ventricle posterior wall; RVAW, right ventricle anterior wall; TV, tricuspid valve; LV, left ventricle; ICD, internal cardiac defibrillator.

**Figure 2a.** ECG recordings, showing paroxysmal episodes of ventricular tachycardia (VT) with an LBBB morphology (i.e. right ventricular VT), A-V nodal re-entrant tachycardia, sinus rhythm, and epsilon waves. LBBB, left bundle branch block; M, monomorphic; TdP, Torsades de pointes; AVNRT, A-V nodal re-entrant tachycardia; AVNT, accelerated A-V nodal tachycardia (AVNT); AF, atrial fibrillation.

OS-69

**Long-term survival in double inlet left ventricle with ventriculo-arterial discordance and pulmonary artery banding: a rare case**  
**Ventriülo-atriyal diskordans ve pulmoner arter bandı ile çift girişli sol ventrikülde uzun dönem sağkalım: Nadir bir olgu**

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**Introduction:** Single ventricle is rare and complex congenital heart disease. Its prognosis is poor, without surgical intervention and long term survival into adulthood is unusual. Double inlet left ventricle (DILV) is a form of single ventricle atrioventricular connection. It is a rare congenital cardiac anomaly with an incidence of 0.05 to 0.1 per 1000 live births. DILV exists when the greater part of both atrioventricular junctions is supported by a single ventricular chamber, more commonly the left ventricle. It comprises a heterogenous group of cardiac anomalies that can involve several combinations of morphological and functional variation at the level of atrioventricular valves, ventriculoarterial connection, and systemic or pulmonary outflow obstruction. We report a case with a DILV and transposition of the great arteries (TGA) that was treated with a pulmonary artery banding (PAB) as a child. We describe the clinical course and crucial factors associated with his prolonged survival.

**Case Report:** A 46 year old man was admitted for new onset of symptomatic palpitations associated with fatigue. His history was double inlet left ventricle, transposition of the great vessels, and subpulmonic stenosis. Pulmonary artery banding procedure was performed, when he was 6 months of age. An implantable cardioverter defibrillator (ICD) had been implanted due to history of sudden cardiac death five years ago. On admission ECG detected high ventricular rate atrial fibrillation. Transesophageal Echocardiography (TEE) was performed. Double inlet left ventricle with transposition of great vessels (figure1-2-3), low normal EF (50%), dilated left and right atrial cavity, 1+ tricuspid, mitral and pulmonary regurgitation was detected (figure4). Patent foramen ovale with left-to-right shunt was observed by color doppler imaging (figure5). Both left and right atrial appendages were free of thrombus. DCCV procedure was performed and sinus rhythm was achieved. After anticoagulation therapy was regulated, the patient was discharged.

**Discussion:** Univentricular hearts are rare and complex congenital malformations. Although survival into late adulthood without surgical repair has been previously documented, it is exceedingly rare. PAB is a palliative procedure to reduce the pulmonary flow. Our case is the rare reported case of DILV with transposition of the great arteries that had undergone PAB without further surgical management to survive into late adulthood.



Figure 1. 2D TEE 4 Chamber view. This image shows single ventricle and the absence of inter-ventricular septum.



Figure 2. The biplane function of a real-time three dimensional (RT3D) TEE. This image shows the aorta and pulmonary artery runs parallel to each other (TGA).



Figure 3. RT3D TEE image of the mitral valve and tricuspid valve from atrial perspective. This image shows both atrioventricular valves are connected to the left ventricle(double inlet morphology).

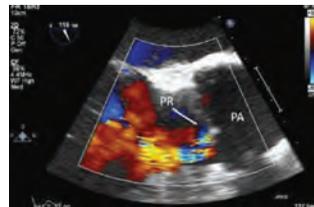


Figure 4. This 2D colored doppler TEE image shows pulmonary regurgitation.

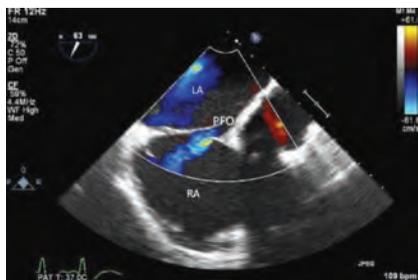


Figure 5. This 2D colored doppler TEE image shows PFO on the interatrial septum.

OS-70

**Atypical form of left ventricular non-compaction resembling mass appearance in papillary muscles**  
**Papiller kaslarda kütle görüntüsünü andran tipik olmayan sol ventrikül non-kompaksiyon formu**

Çağlar Emre Çağlıyan<sup>1</sup>, Rabia Eker Akılılı<sup>1</sup>, Ali Deniz<sup>1</sup>, Onur Sinan Deveci<sup>1</sup>, Şerafettin Demir<sup>2</sup>, Aziz İnan Çelik<sup>1</sup>, Mehmet Kanadaşı<sup>1</sup>

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**Case Report:** A 27 year-old male was referred to our clinic with complaints of unconsciousness and palpitations. His symptoms had started 1 year before his referral, accelerated in the last 6 months; with a frequency of 2-3 attacks in a month. He used to lose his consciousness after an episode of rapid palpitation. His electrocardiogram revealed left ventricular hypertrophy with negative T waves on leads V3-6. Transthoracic echocardiography (TTE) was performed, which showed hypertrabeculation and non-compacted areas in left ventricle with a mass like lesion at the papillary muscles (Figure 1 A). There was blood flow in the intertrabecular space (Figure 1 B). His left ventricular diastolic dimension was slightly increased (60 mm), but his ejection fraction was normal (60 %). Short axis view in the mid-ventricular level showed a lacy spongy appearance (Figure 2A), obstructing most of the ventricular cavity in systole (Figure 2 B). There was also moderate mitral regurgitation. Cardiac magnetic resonance imaging (MRI) showed a hypertrabeculated and two-layered left ventricular structure, with an anomalous muscle shelf dividing the chamber into compacted and non-compacted areas (Figures 3A and B). The entity represented an anomalous persistence and hypertrophy of the trabecular layer. The distal part of the muscle shelf was composed of the trabeculated area, whereas the proximal part was composed of the compact area. Papillary muscles were not well formed and tendinous cords seemed to be attached directly to the anomalous muscle shelf dividing the left ventricle. These findings were compatible with an atypical presentation of non-compaction cardiomyopathy, which looked like a “double chambered left ventricle”. Neurologic evaluation revealed suspicious epileptical waveforms on electroencephalogram. His neurologic examination was normal and he didn't have manifest neurological symptoms. His electro-myography and cerebrospinal MRI were normal. Since the patient was complaining syncopal attacks after palpitations, a single chamber intracardiac cardioverter defibrillator (ICD) had been implanted. Beta-blocker, angiotensinogen converting enzyme (ACE) inhibitor, acetyl salicylic acid and carbamazepine therapy had been initiated to our patient. In the 3-month follow up period, he hasn't experienced any syncopal attack.

**Discussion:** Left ventricular non-compaction (LVNC) is an unclassified cardiomyopathy, which may accompany other congenital heart disease or occur in isolated form. Heart failure (> 50%), genesis of ventricular arrhythmias and thromboembolic events are the most important complications of LVNC. In some patients, LVNC may accompany hypertrophic cardiomyopathy. Another interesting feature of LVNC is its relationship with neuromuscular disorders. ICD implantation is suggested for patients with LVNC accompanied by dilated cardiomyopathy (reduced ejection fraction) and hypertrophic cardiomyopathy, who have documented malignant ventricular arrhythmias.

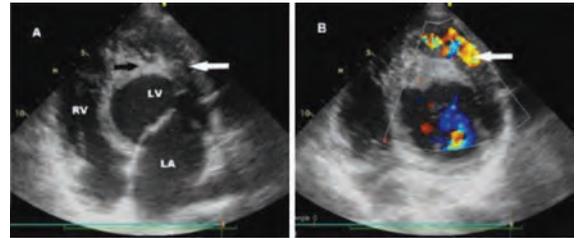


Figure 1. A. Apical 4 chamber images of the patient. Black arrow: Mass appearance in the papillary muscles. White arrow: Two layered structure. B. Color-Doppler appearance of inter-trabecular blood flow (white arrow). LV: Left ventricle, LA: Left atrium, RV: Right ventricle.

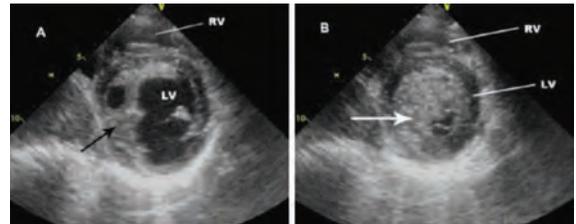


Figure 2. Mid-ventricular short axis plane. A. Spongy, lacy appearance of the trabecular layer (black arrow). B. Obstruction of the ventricular cavity in systole (white arrow). LV: left ventricle, RV: Right ventricle.

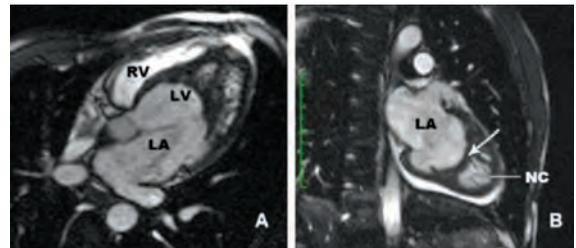


Figure 3. Magnetic resonance imaging. A. Transverse plane B. Sagittal plane. LV: Left ventricle, LA: Left atrium, RV: Right ventricle, White arrow: Anomalous muscle shelf, NC: Non-compacted area.

OS-71

**Pneumomediastinum and subcutaneous emphysema after transesophageal echocardiography: an unusual complication**  
**Transözofageal ekokardiyografi sonrası pnömomediastinum ve subkütan amfizem: Nadir bir komplikasyon**

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**Introduction:** Transesophageal echocardiography (TEE) is a semi-invasive diagnostic technique, and well tolerated by most patients. It is important to bear in mind the TEE-related complications. Difficulty breathing, abnormal heart rhythms, bleeding or damage to the throat or esophagus can be seen during the TEE procedure. Nevertheless, severe, even life-threatening complications have been rare reported in the literature.

**Case report:** A 64-year-old man with aortic valve replacement performed in 1991 was referred to our hospital for shortness of breath. TTE revealed moderate to severe aortic paravalvular leak (PVL). A TEE was performed to delineate the exact localization, the anatomy of the defect and the degree of PVL. Unfortunately, the patient did not tolerate the TEE procedure. One hour after the procedure, the patient complained of sore throat and dysphagia, but he did not mention any dyspnea. Also, he noted crackly skin over both sides of his neck. On examination, he was not in respiratory distress, afebrile and oxygen saturation was 90% on room air. Blood pressure was 120/85 mmHg and heart rate 82 bpm. There was subcutaneous crepitation on palpation of the both sides of his neck up to the mandible. The lungs were clear on auscultation. CT scan of head, neck and thorax demonstrated large amount of air in the subcutaneous tissue of the neck and mediastinum suggesting pneumomediastinum. The patient's oral intake was ceased and high-flow nasal oxygen was given (10 L/min) to facilitate resorption of the mediastinal and subcutaneous air and expedite nitrogen resorption from tissues. Maneuvers increasing intrathoracic pressure such as Valsalva were recommended to be avoided. Also, antibiotic therapy consisting of piperacillin and tazobactam was given to avoid the risk of a possible mediastinitis. Nasal endoscopy was performed and revealed that upper respiratory tract to the vocal cords was intact. Subsequently, an upper gastrointestinal endoscopy was performed and demonstrated that esophagus was intact. Also, he underwent a CT scan of thorax with orally administered non-ionic intravenous contrast media (iopromide) was performed to exclude the possibility of microperforation of esophagus, and demonstrated no contrast extravasation into the mediastinum and/or subcutaneous tissue. Then, oral intake was initiated with liquid. The patient was discharged in a very good condition on the 15th hospital day.

**Discussion:** TEE procedure is usually carried out without an visual control during probe insertion and manipulation in the mouth, upper respiratory tract and/or esophagus. Therefore, TEE procedure may carry the possibility of damage to the throat or esophagus. It is important to be familiar with potential complications of TEE to allow a thorough risk-benefit analysis on an individual basis, especially for practicing echocardiographers. To the best of our knowledge, this complication has been rarely reported after TEE procedure in the literature.

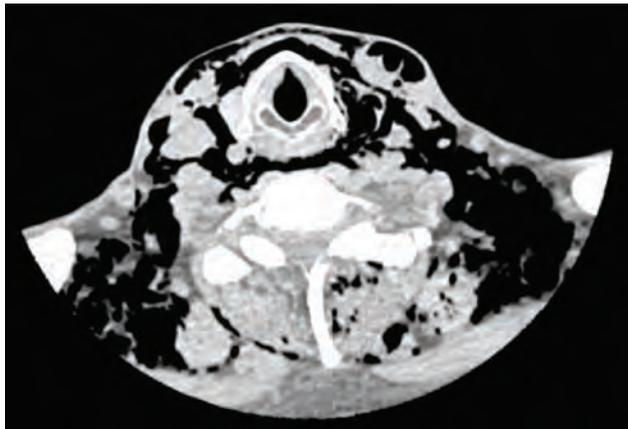


Figure 1. CT scan of the neck shows large amount of air in the subcutaneous tissue of the neck.



Figure 2. CT scan of the thorax shows pneumomediastinum.

OS-72

**Cholesterol forms and traditional lipid profile for projection of atherogenic dyslipidemia: Lipoprotein subfractions and erythrocyte membrane cholesterol**

**Aterojenik dislipidemi projeksiyonu için kolesterol formları ve geleneksel lipid profili: Lipoprotein alt grupları ve eritrosit membran kolesterolü**

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**Aim:** Atherogenic dyslipidemia characterized by abnormal changes in plasma lipid profile such as low HDL and increased TG levels is strongly associated with atherosclerotic diseases. We aimed to evaluate the levels of pro-and anti-atherogenic lipids and erythrocyte membrane cholesterol (EMC) content in normo-and dys-lipidemic subjects to investigate whether EMC content could be a useful marker for clinical presentation of atherogenic dyslipidemia.

**Methods:** LDL, HDL and their subfraction levels and erythrocyte lipid content were determined in 64 normolipidemic (NLs), 42 hypercholesterolemic (HCs) and 42 mixed-type dyslipidemic subjects (MTDs).

**Results:** Plasma atherogenic lipid indices [Small-Dense LDL (sdLDL)/Large-dense HDL (LHDL), TC/HDL-C, TG/HDL-C and Apo B/AI] were higher in MTDs compared to NLs (p < 0.001). The highest sdLDL level was observed in HCs (p < 0.01). Despite a slight increase in EMC level in dyslipidemic subgroups, the difference was not statistically significant. A significant negative correlation, however, was observed between EMC and sdLDL/LHDL in HCs (p < 0.035, r: -0.386). ROC curves to predict sdLDL level showed that TG and EMC levels had higher AUC values compared to other parameters in HCs.

**Conclusions:** We showed that diameters of larger LDL and HDL particles tend to shift toward smaller values in MTDs. Our results suggest that EMC content as well as TG levels may be a useful predictor for sdLDL level in hypercholesterolemic patients.

Table 1. Distribution of baseline characteristics of normolipidemic and dyslipidemic groups.

Variables	Normolipidemic Group (n: 64)	Hypercholesterolemic Group (n: 42)	Mixed-Type Dyslipidemic Group (n: 42)
Age (years), X±SD	58±11	58±13	56±11
BMI (kg/m <sup>2</sup> ), X±SD	29±5	29±5	30±5
Waist circumference (cm), X±SD	101±13	100±11	106±11
Gender, male, % (n)	64(41)	33(14)	67(28)*
Hypertension, % (n)	42(27)	43(18)	43(18)
Smoking, % (n)	16(10)	16(7)	42(18)* a*
Diabetes, % (n)	16(10)	11(5)	29(12)

Comparison of mixed-type dyslipidemic group with normolipidemic (a) and hypercholesterolemic groups (b). \* P < 0.05.

Table 2. Concentrations of plasma and erythrocyte lipids and plasma apolipoproteins

Variable	Normolipidemic Group (n: 64)	Hypercholesterolemic Group (n: 42)	Mixed-Type Dyslipidemic Group (n: 42)
TC (mg/dL)	164±24	239±23***	240±28***
TG (mg/dL)	94 (84-99)	108 (99-120)**	239 (228-272)***
LDL-C (mg/dL)	70±26	116±22***	117±20***
HDL-C (mg/dL)	44 (40-48)	32 (47-40)**	37 (31-41)**
VLDL-C (mg/dL)	41(33-42)	32 (49-41)**	59 (52-63)**
Apo B (mg/dL)	81±20	121±26***	130±29***
Apo AI (mg/dL)	148±27	117±28***	113±24***
LDL (g/L)	18 (15-20)	25 (24-24)	20 (8-42)
Apo B (g/L)	4.7 (3.8-4.8)	4.5 (3.7-4.6)	5.8 (5.1-6.0)***
Broadest LDL-C (mg/dL)	50 (36-49)	39 (37-51)	37 (34-43)
sdLDL-C (mg/dL)	4 (3-10)	7 (10-26)	42 (37-56)***
Mean LDL Size (Å)	268 (257-268)	268 (264-268)	263 (260-272)***
LDL-C (mg/dL)	12 (17-18)	17 (16-22)	10 (9-13)***
sdLDL-C (mg/dL)	5 (3-7)	8 (8-9)**	8 (6-7)*
Eryc. Cholesterol (mg/mg Ph.)	436±111	481±205	490±211
Eryc. Ph (µg/mg Ph.)	703±32	716±34	712±31

Data are presented as mean±SD for normally distributed and as median (Q1) for nonnormally distributed continuous variable. Comparison of normolipidemic with hypercholesterolemic (a), mixed-type dyslipidemic (b), and/or hypercholesterolemic with mixed-type dyslipidemic (c). \* P < 0.05, \*\* P < 0.01, \*\*\* P < 0.001.

Table 3. Prevalence of different forms of atherogenic dyslipidemia in study groups.

Sample Groups	High TC/HDL-C ratio, % (n)	High TG/HDL-C ratio, % (n)	High sdLDL-C ratio, % (n)	Pattern B, % (n)	High Erythrocyte Cholesterol, % (n)
Normolipidemic Group (n: 64)	20 (13)***	16 (10)†	6 (4)†	D (0)†	D (0)†
Hypercholesterolemic Group (n: 42)	64 (27)†***	12 (5)†	26 (11)†*	7 (3)†*	15 (6)†**
Mixed-Type Dyslipidemic Group (n: 42)	98 (41)†***	100 (42)†***	50 (21)†***	17 (7)†**	15 (6)†**

a, b, c indicate significant difference with a column at \* P < 0.05, \*\* P < 0.01 and \*\*\* P < 0.001.

Table 4. Spearman rank correlations between ratios of lipids, lipoproteins, ghost C/P and apoproteins.

	TC/HDL-C (r, p)	TG/HDL-C (r, p)	ApoB/ApoAI (r, p)	sdLDL-C/LHDL-C (r, p)	Mean LDL Size (r, p)	Erythrocyte Cholesterol (r, p)
TC/HDL-C	-	0.664 a*** 0.746 a*** 0.483 c***	0.499 a*** 0.598 b*** 0.534 c***	0.261 a*** 0.494 b*** 0.505 c***	-0.321 b*	NS
TG/HDL-C	-	-	0.579 b***	0.312 a* 0.568 b***	-0.277 a* -0.484 b***	NS
ApoB/ApoAI	-	-	-	0.501 b* 0.389 c*	-0.427 b*	0.440 c*
sdLDL-C/LHDL-C	-	-	-	-	-0.861 a*** -0.951 b*** -0.802 c***	-0.386 b*
Mean LDL Size	-	-	-	-	-	0.257 a* 0.415 b***

a Normolipidemic group b Hypercholesterolemic group c Mixed-type dyslipidemic group \* P < 0.05, \*\* P < 0.01 and \*\*\* P < 0.001.

## Unusual cases with crooked descending thoracic aorta compressing the left atrium

### Eğri torasik aortanın sol atriyuma bası yaptığı nadir vakalar

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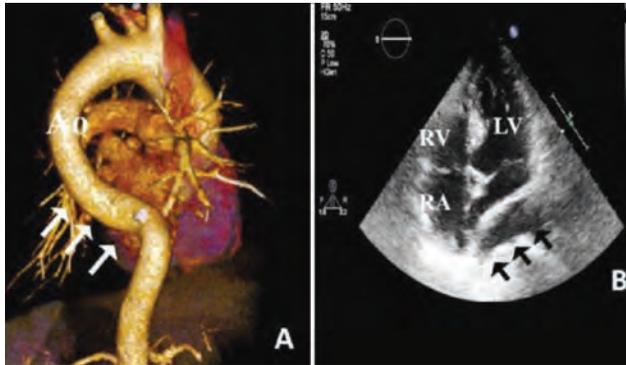
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**Introduction:** Characteristics such as central position within the thorax, low intraluminal pressure and relatively thin wall, make left atrium vulnerable to compression caused by various mediastinal masses. Left atrial encroachment caused by a thoracic aorta is an extremely rare malformation of the cardiovascular system. Echocardiographic manifestations may mimic left atrial space-occupying lesions. Despite publications on aortic anomalies, left atrial compression has rarely been reported. Herein, we present two cases with crooked descending aorta mimicking primary cardiac tumors or metastatic neoplasms by compressing left atrium on echocardiography.

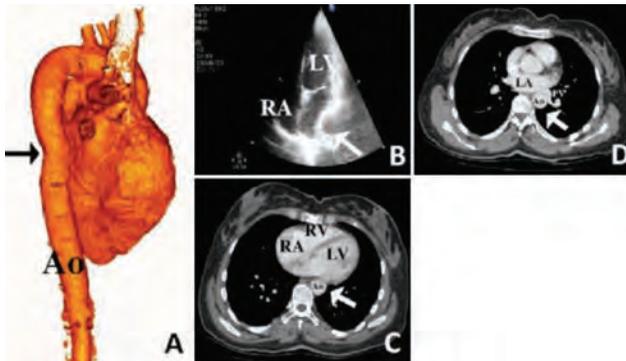
**Case 1:** A 65-year-old Caucasian female presented to the cardiology clinic with symptoms of dyspnea and retrosternal chest pain. She had no known medical history. Physical examination, electrocardiogram and blood biochemistry values were in normal range. Her blood pressure on admission was 120/70 mmHg. Two-dimensional echocardiography was performed which revealed moderate aortic insufficiency and compression of left atrium by a luminal structure (Figure 1B). And left and right ventricle systolic functions were preserved. The three-dimensional computed tomography evaluation shows an extrinsic compression of left atrium by displaced descending aorta (Figure 1A). These findings can explain symptoms by decreasing left atrial compliance.

**Case 2:** A 47-year-old Caucasian female admitted to our cardiology clinic with symptoms of dyspnea and retrosternal chest pain. She did not have prior cardiovascular disease. Her past medical history displays hypothyroidism, and she is on thyroid hormone supplementation. Physical examination and blood biochemistry values were normal. The electrocardiogram showed normal findings. Echocardiography was performed for further evaluation. Transthoracic echocardiography revealed normal systolic function and almost partial compression of the left atrium (Figure 2B). A subsequent thoracic computed tomography imaging showed thoracic aorta with a low-angle twist through left atrium was caused external compression to atrium and left pulmonary vein (Figure 2A, C, D). We recommended medical management and follow up. The patients remain stable on medical follow-up.

**Conclusions:** In patients who do not respond to medical therapy for heart failure or recurrent heart failure events, compression of the heart by aorta must be kept in mind and should be further evaluated by echocardiography.



**Figure 1.** Left atrial compression by aorta. (A) The image of contrast enhanced 3-Dimensional thoracic computed tomography shows elongated and curving (tortuous) descending aorta leans to the left atrium wall (white arrows). (B) Apical four chamber echocardiographic examination demonstrates left atrium compression by descending thoracic aorta (black arrows). Descending aorta causes nearly complete obliteration of the left atrial cavity. Ao= Descending aorta; LV= Left ventricle; RA= Right atrium; RV= Right ventricle.



**Figure 2.** Left atrial compression by aorta. (A) Lateral image of 3-Dimensional computed tomography shows descending aorta angulation (black arrow) causes left atrial compression. (B) Apical four chamber transthoracic echocardiographic view depicting a prominent mass encroaching to left atrium (white arrow). (C) Axial contrast enhanced computed tomography imaging shows descending thoracic aorta with a left atrial course (white arrow). (D) Another computed tomography view clearly demonstrated that the left pulmonary vein was critically squeezed by descending aorta (white arrow). Ao= Descending aorta; LA= Left atrium; LV= Left ventricle; PV= Pulmonary vein; RA= Right atrium; RV= Right ventricle.

## Bicuspid aortic valve with type A interrupted aortic arch in a 34-year-old man

### Tip A interrup aortik arkus ile biküspit aort kapak olan 34 yaş erkek

Murat Akkoyun<sup>1</sup>, Bulent Mese<sup>2</sup>, Gurkan Acar<sup>1</sup>, Idris Ardic<sup>1</sup>

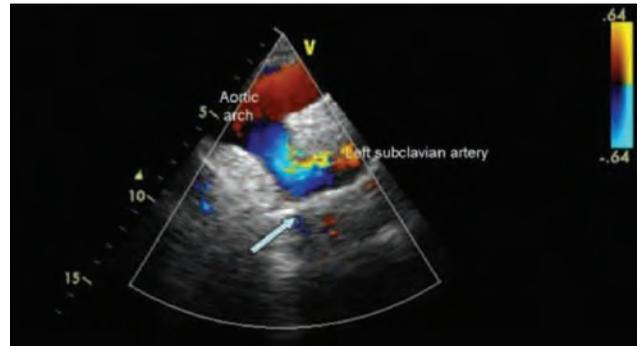
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A 34-year-old man was referred to our outpatient cardiology department complaining of chest and back pain. He had no remarkable medical history. Regular medical examination revealed elevated arterial blood pressure of 150/85 mmHg at the upper arm and weak femoral arterial pulse. On cardiac auscultation, mild diastolic murmur was heard at the left parasternal border. A 12-lead electrocardiogram showed normal sinus rhythm. Chest radiographs revealed bilateral notching of the ribs (Fig. 1). Two-dimensional echocardiography showed a bicuspid aortic valve, mild aortic regurgitation and left ventricular hypertrophy with normal left ventricular systolic function. Interrupted aorta just after the left sub-clavian artery was determined by suprasternal notch echocardiography (Fig. 2). Cardiac angiograms showed a type A aortic interruption and a hypoplastic distal arch (Fig. 3). Also contrast-enhanced computed tomography demonstrated type A interrupted aorta just after left sub-clavian artery (Fig. 4). Surgical repairment of interrupted aortic arch was recommended. Bicuspid aortic valve with interrupted aortic arch is very rarely reported in adults.



**Figure 1.** Chest radiograph shows bilateral notching of the ribs.



**Figure 2.** A type A interrupted aortic arch (arrows) just after the left subclavian artery was determined by suprasternal notch two-dimensional echocardiography.



**Figure 3.** Aortograms after radial arterial puncture show the site of aortic interruption (arrows).



**Figure 4.** Multislice computed tomographic angiograms in oblique sagittal view show a type A interrupted aortic arch (arrows).



OS-77

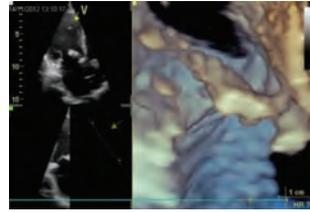
### A pre- and postoperative assessment of mitral valve defect in a child using real-time three-dimensional transthoracic echocardiography Çocukta mitral kapak defektinin pre- ve postoperatif olarak gerçek zamanlı üç boyutlu transtorask ekokardiyografi ile değerlendirilmesi

Arda Saygılı<sup>1</sup>, Yasemin Türkel<sup>2</sup>, Ahmet Armaz<sup>2</sup>, Yusuf Yalçınbaş<sup>2</sup>, Tayyar Sarıoğlu<sup>3</sup><sup>1</sup>Acibadem Hospital, Pediatric Cardiology, İstanbul<sup>2</sup>Acibadem Bakırköy Hospital Cardiac Surgery, İstanbul<sup>3</sup>Acibadem University, Cardiac Surgery, İstanbul

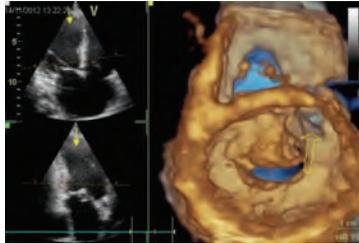
A 14 year-old boy presented with severe mitral regurgitation and a dilated left ventricle. He had a history of partial atrioventricular septal defect repair in 2011. An echocardiographic examination with real-time 3D image reconstruction revealed severe mitral regurgitation due to a 0.5 cm defect at the annular junction of the anterior leaflet. In addition, intraoperative exploration of the left atrioventricular valve verified a 7 mm defect close to the annular junction of the anterior leaflet that had been identified via the 3D echocardiography. Repair of the left atrioventricular valve was completed with a fresh autologous pericardial patch, and a postoperative echocardiographic examination demonstrated competent left atrioventricular valve function.



**Figure 1.** Two-dimensional echocardiographic images and color Doppler showing a mitral defect regurgitation.



**Figure 2.** A mitral valve defect in the modified four chamber view.



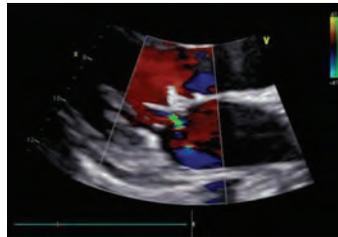
**Figure 3.** A real-time 3D image taken below the mitral valve showing the mitral valve defect near the annular junction of the anterior leaflet in more detail from the surgical point of view.



**Figure 4.** Surgical image demonstrates same details of the mitral defect before valve repair.



**Figure 5.** Surgical image demonstrates same details of the mitral defect after valve repair.



**Figure 6.** Color Doppler show minimal mitral regurgitation after repair.

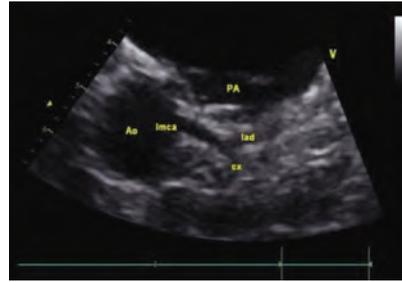
OS-78

### Does very high takeoff of the left main coronary artery in children result in a high risk for sudden death? The importance of echocardiography in routine practice

#### Çocuklarda sol ana koroner arterin çok yüksek çıkışı ani ölüm için yüksek risk midir? Rutin uygulamada ekokardiyografinin önemi

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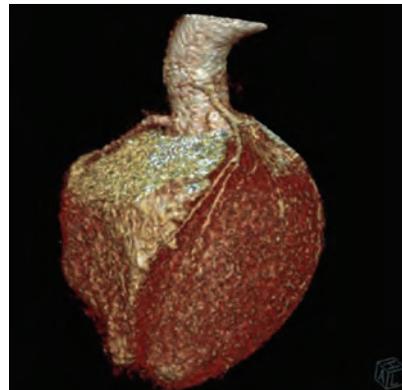
A 10-year-old boy was referred to the emergency services of our hospital for an evaluation of precordial chest pain that occurred during basketball training. The patient had no family history of sudden death, syncope, or palpitation. He was asthmatic and had been followed up regularly by a pediatric pneumologist. On examination, his blood pressure was 90/60 mmHg, and his pulse rate was 84/sec. In addition, a grade 1/6 systolic ejection murmur was audible at the left upper sternal border. The chest radiography and electrocardiography results were unremarkable, and the cardiac troponin levels were normal. However, echocardiography (Figure 1) revealed that the left main coronary artery (LMCA) was originating abnormally from the high position of the right coronary artery (RCA) above the sinotubular junction. Furthermore, the left coronary ostium could not be seen. It is normal to find that the cardiac structure has minimal tricuspid regurgitation, and patients with this condition are usually given a treadmill stress test. In our case, the patient exercised for 10 minutes and achieved 95% of the predicted targeted heart rate without electrocardiographic changes during the examination and the recovery phase. Dual-source computed tomography coronary angiography (CTCA) showed an anomalous implantation in the LMCA that originated from approximately 2 cm above the coronary sinus and passed between the aorta and the right ventricle outflow tract (RVOT) where compression often occurs (Figure 2 and 3). Surgical treatment was then proposed for the patient.



**Figure 1.** Parasternal modified short-axis view of the right ventricular outflow tract in two-dimensional echocardiography showing the left main coronary artery originating abnormally from a high position above the sinotubular junction.



**Figure 2.** Coronary computed tomography angiograms in multiplanar reformatted views show a very high takeoff of the left main coronary artery from above the left coronary sinus. (arrows).



**Figure 3.** Coronary computed tomography angiograms in three-dimensional show a very high takeoff of the left main coronary artery from above the left coronary sinus.

## Percutaneous lead extraction by femoral approach, case report

### Perkütan femoral yolla yapılan lead ekstraksiyonu, olgu sunumu

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**Case:** Our aim in this case report is to discuss a patient who has undergone lead extraction procedure by femoral approach. The patient is 72 years old female who had a VVI pacemaker with passive fixation lead in 1997 for atrial fibrillation with low ventricular rate. In 2009 the lead and battery are replaced because of EOL and lead dysfunction in former lead and this time a new active fixation lead is used. The lead with dysfunction is cut and capped and left in the same PM pocket. At April 2013 patient presented to our clinic with erosion and serous type discharge from PM pocket. Interrogation of device confirmed that she was pace dependent. She is admitted to hospital, pocket discharge and blood cultures are taken, transthoracic and transesophageal echocardiography is performed and revealed no sign of infective endocarditis. We decided to remove all hardware and insert a new battery and lead on contralateral side. However the first passive lead seemed to be in deeper side of subclavian region (Figure 1) and if it is not going to be reached by subclavian approach femoral approach for the extraction of lead is planned. The patient is taken to the catheterisation laboratory and invasive hemodynamic monitoring during procedure is undertaken. When the active fixation lead is unscrewed by inserting stylet in it, it quickly released from myocardium and is extracted by simple traction. However the passive lead could not be reached by subclavian approach. Then we proceeded to the femoral Bryd Workstation 12F™ and Needle's eye Snare® to extract lead femorally. (Figure 2) The lead is snared at subclavian end and after its release from superior vena cava it is extracted by applying simple traction at inf vena cava and then it released from right ventricular myocardium and taken into the femoral Bryd Workstation 12F™ as shown in Figures 3, 4 and 5. Because of higher inner diameter of femoral Bryd Workstation™ the femoral vein is sutured by vascular surgeons at the end of the procedure. Control echocardiography was normal except minimal pericardial effusion that occurred after procedure. Patient didnt have a septic clinic and the blood cultures were negative so we implanted a VVI PM at contralateral side the next day after procedure. The patient's follow-up was uneventful after procedures and she is discharged in a few days.

**Conclusion:** Percutaneous femoral approach for lead extraction has been reported as a useful alternative for leads that cannot be reached by subclavian approach. It does have the same risks with subclavian approach but not more because heart is held by ligaments mostly from upper parts. It must be emphasized that one should not cut leads too short or damage inner coils during first procedure because it complicates later revision attempts. If reaching leads by superior approach is not possible an inferior approach can also be thought as a treatment option in experienced hands.

**Amaç:** Bu vaka bildirisinde perkütan femoral yolla lead ekstraksiyonu yapılan bir olguyu tartışmayı amaçladık.

**Olgu:** 72 yaşında kadın hasta 1997'de düşük ventrikül hızlı atrial fibrilasyon nedeni ile pasif fiksasyon lead kullanılarak VVI pacemaker implante edilmiş. 2009 yılında ise leade kırık olması ve End of Life nedeniyle aktif fiksasyon lead kullanılarak batarya değişimi yapılmış ve ilk lead kesilerek keplenmiş ve cepte bırakılmış. Hasta Nisan 2013'te PM bölgesinde erozyon ve akıntı nedeniyle kliniğimize başvuruyor. Hasta yatırılarak tetkik edildi; yara kültürü ve kan kültürleri alınmasını takiben antibiyotik tedavisi başlandı. Yapılan transtorakik ve transözofajial ekolarında vejetasyon izlenmedi. Cook® cihazı kullanılarak lead ekstraksiyonu yapılmasına karar verildi ancak ilk takılan pasif leadin kesilen kısmından sonra kalan proksimal kısmının subklavian bölgede derinde olduğu izlendi. (Şekil 1) Bu nedenle gerekirse ilk leadin femoral yolla çıkartılması planlanarak hasta kateter laboratuvarına alındı. İşlem sürekli invaziv hemodinamik monitörizasyon altında bilinci anestezi ile uygulandı. Subklavian bölgenin explorasyonunu takiben aktif fiksasyon lead uc kısmından stile ile uyestleştirildikten sonra basit traksiyonla kolayca çıkarıldı. Ancak pasif leadin proksimal ucu subklavian venin içinde olduğundan femoral bölgeden Bryd Workstation™ 12F ve Needle's eye snare® (Şekil 2) kullanılarak pasif leadin çıkartılmasına karar verildi. Lead önce subklavian bölgede takiben de inferior vena cava'da yakalandı, sürekli traksiyon uygulanarak önce superior vena cava kısmından sonra da sağ ventrikül içindeki yapıyağı kısmından ayrılarak Bryd Workstation™ içine alınarak ekstrakte edildi. (Şekil 3,4,5) Böylece vücutta yabancı madde bırakılmamış oldu. Bryd WorkStation 12 F olması nedeniyle femoral ven kalp damar cerrahisi bölümünce primer olarak sütüre edilerek kapatıldı. İşlem sonrası kontrol ekokardiyografide tamponada sebep olmayan minimal perikardiyal efüzyon izlendi, kapak patolojisi izlenmedi, rezidü koil veya plastik parça saptanmadı. Hastanın ateşinin ve septik kliniğinin olmaması ve kan kültürlerinde üreme olmaması üzerine ertesi gün kalıcı VVI PM karşı subklavian bölgeden implante edildi. Takiplerinde sorun olmayan hasta önerilerle taburcu edildi.

**Sonuç:** Perkütan femoral yolla lead ekstraksiyonu subklavian bölgeden yakalanamayan leadler için kullanışlı bir yöntem olarak literatürde bildirilmiştir. Leade aşağıdan uygulanan traksiyon kalbi sabit tutan ligamentlerin çoğundan yukarıdan tutunması nedeniyle subklavyenden çekmeye nazaran ek risk oluşturmaz. Ancak kullanılmaması hale gelmiş leadlerin eğer yerinde bırakılacak subklavian vena uyuğ mesafede ve leadin iç sarmallarının hasar görmemesine özen gösterilerek keplenmesi ileride yapılacak revizyon işlemleri için kolaylık sağlayacaktır. Yukarıdan yakalamanın mümkün olmadığı durumlarda ise femoral yolla lead ekstraksiyonu tedavi seçeneği olarak düşünülmelidir.

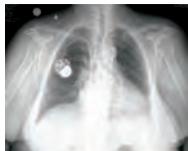


Figure 1. Chest X ray before procedure.



Figure 2. Bryd Workstation™ 12F and Needle's eye Snare®.

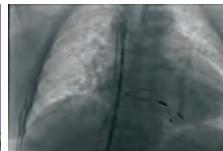


Figure 3. Snaring of lead at subclavian end by femoral approach.

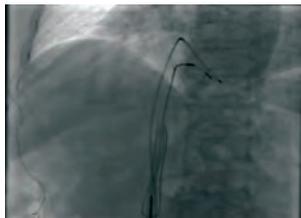


Figure 4. Traction applied to lead by femoral approach.



Figure 5. Extracted lead is taken into the femoral Bryd Workstation™.

## Disappearance of myocardial bridging of the left anterior descending coronary artery after inferior myocardial infarction

### İnferiyer miyokart infarktüsü sonrası sol ön inen koroner arterde kaybolan miyokardiyal köprüleşme

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**Background:** Myocardial bridging (MB) is defined as an intramural segment of a coronary artery that normally courses epicardially. The intramyocardial coronary arterial segment is termed a "tunnelled segment". It is considered a congenital anomaly that most commonly affects the mid portion of the LAD. However, some complications such as ischaemia, acute coronary syndromes, coronary spasm, arrhythmias and sudden death have been reported. Therefore, the diagnosis and treatment of MB are both important. Since MB is congenital, disappearance of a MB is unlikely. We here report a very rare case of disappearance of MB after inferior myocardial infarction (MI).

**Case:** A 54 years old male patient was admitted to our clinic with the complaint of atypical chest pain. The 12 lead ECG was showed sinus rhythm and negative T wave was seen on V3-6 derivations (Figure 1). On the physical examination blood pressure was 130/75 mm Hg, heart rate was 68/min without murmurs. Ejection fraction was found normal in ECHO. He underwent to exercise stress test due to family history, and smoking 2 mm ST depression was seen in D1-aVL and V3-6 derivations. Coronary angiography (CA) was performed. CA showed plaques in RCA and CX, a long MB causes to 99% stenosis in middle of LAD (Figure 2). CABG was recommended to patient but he refused to operation. He was discharged with medical therapy. The patient was came back to our emergency department of the hospital with the complaint of typical chest pain one year later. Cardiac enzymes were elevated and Q waves and negative T waves were seen on D2-3-aVF derivations and negative T waves on V3-6 derivations was disappeared (Figure 3). The patient was admitted to coronary intensive care. Bedside ECHO was performed. Wall motion abnormality was observed in inferior and posterior wall. Ejection fraction was 50%. CA was performed due to ongoing chest pain. CA showed plaque in CX and LAD, 100% stenosis in proximal of RCA. The last CA images were compared with old CA images. Disappearance of RCA in the middle of LAD was seen (Figure 4). Stent deployment following balloon angioplasty was done for RCA. 100% patency of RCA was achieved after (Figure 5). Chest pain was relieved. After 3 months later CA was performed. RCA was open and no MB in the middle of LAD was seen on CA images (Figure 6).

**Conclusion:** The LAD was the most common coronary artery affected by MB. There are two different types of MB: (I) The superficial type, which crosses the coronary artery perpendicularly or at an acute angle toward the apex, and accounts for the majority of cases; and (II) muscle fibres arising from the right ventricular apical trabeculae that cross the LAD transversely, obliquely or helically before terminating in the interventricular septum. We thought that our patient had second type. Because total occlusion of RCA might be caused to necrosis on muscle fibers that cross the LAD and changed motion and direction of muscle fibers. As a result this change might be caused to disappearance of MB of LAD.

**Giriş:** Miyokardiyal köprüleşme (MK) normalde epikardiyal seyreden koroner arterin kas içindeki bölümü olarak tanımlanır. Kas içindeki koroner arteriyel bölümünün tunel bölümü olarak adlandırılır. Bu çoğunlukla sol ön inen arterin (LAD) orta bölümünü etkileyen konjenital bir anomalidir. Ancak iskemi, akut koroner sendrom, koroner spazm, aritmi ve ani ölümler gibi komplikasyonlar bildirilmiştir. Bu yüzden MK tanı ve tedavisi çok önemlidir. MK konjenital olduğundan kaybolması pek beklenmez. Biz ise burada çok nadir görülen inferiyer miyokart enfarktüsü sonrası kaybolan MK vakasını sunacağız.

**Olgu:** 54 yaşında erkek hasta atipik göğüs ağrısı ile kliniğimize başvurdu. 12 derivasyonlu EKG si sinus ritimindeydi ve V3-6 derivasyonlarında T (-) lığı vardı (Şekil 1). Fizik muayenesinde kan basıncı 130/75 mm Hg kalp hızı 68/dak olup üfürümü yoktu. Ejeksiyon fraksiyonu EKO'da normal saptandı. Aile hikayesi ve sigara öyküsü olduğu için egzersiz stres testi yapıldı. D1-aVL ve V3-6 derivasyonlarında 2 mm ST depresyonu izlendi. Koroner anjiyografi yapıldı. Koroner anjiyografide RCA ve CX'te plak, LAD mid bölgede uzun % 99 darlık oluşturan miyokardiyal köprü görüldü (Şekil 2). Hastaya CABG önerildi fakat operasyonu kabul etmedi. Medikal tedavi verilerle taburcu edildi. Hasta 1 yıl sonra hastanemizin acil servisine tipik göğüs ağrısı şikayeti ile geldi. Kardiyak enzimleri yüksek ve D2-3-aVF derivasyonlarda Q dalgası ve T dalga negatifliği olduğu ve V3-6 derivasyonlarında görülen T dalga negatifliğinin kaybolduğu görüldü (Şekil 3). Hasta koroner yoğun bakıma alındı. Yatak başı EKO yapıldı. Inferiyer ve posteriyer duvarda duvar hareket kusuru izlendi. Ejeksiyon fraksiyonu % 50 idi. Devam eden göğüs ağrısı nedeniyle koroner anjiyografi yapıldı. Koroner anjiyografide CX ve LAD'de plak, RCA proksimalinde ise %100 darlık saptandı. Inferiyer ve posteriyer duvarda duvar hareket kusuru izlendi. Ejeksiyon fraksiyonu % 50 idi. Devam eden göğüs ağrısı nedeniyle koroner anjiyografi yapıldı. Koroner anjiyografide RCA ve CX'te plak, LAD mid bölgedeki MK'nin kaybolduğu görüldü (Şekil 4). RCA için balon anjiyoplasti sonrası stent yerleştirme işlemi yapıldı. İşlem sonrası RCA'da %100 açıklık sağlandı (Şekil 5). Göğüs ağrısı geriledi. 3 ay sonra kontrol anjiyografisi yapıldı. Koroner anjiyografide RCA damarının açık ve LAD mid bölgedeki MK'nin olmadığı izlendi (Şekil 6).

**Sonuç:** Sol ön inen arter MK'nin en sık olduğu koroner arterdir. MK'nin iki farklı tipi vardır: (I) Yüzeysel tip, koroner arteri dik olarak çaprazlayan veya apse ani açı veren vakaların çoğunda olan (II) İnterventriküler septumda sonlanmadan önce, sol ön inen arteri yatay, oblik veya helikal olarak çaprazlayan sağ ventrikül apikal trabekülenden kaynaklanan kas lifleri. Biz bu hastanın ikinci tipi uduğunu düşündük. Çünkü sağ koroner arterin total oklüzyonu LAD arteri çaprazlayan kas liflerinin nekroza yol açması olabilir ve bu durum kas liflerinin hareket ve yönünü değiştirmiş olabilir. Sonuç olarak bu da LAD'deki MK'nin kaybolmasına neden olmuştur.



Figure 1. The first incoming ECG of patient.

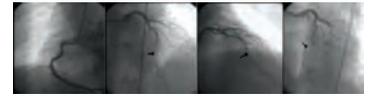


Figure 2. A long myocardial bridge is seen on coronary angiography images of the patient that cause to 99% stenosis in the middle of LAD (black arrow).



Figure 3. Q and negative T waves on D2-3-aVF derivations and loss of negative T waves on V3-6 derivations were seen when compared older ECG.



Figure 4. Coronary angiography images of the patient after myocardial infarction. Disappearance of myocardial bridging can be seen on figure A, B and C (black arrow). Occlusion of proximal RCA and anterog flow in distal part of RCA can be seen on figure D (black arrow).

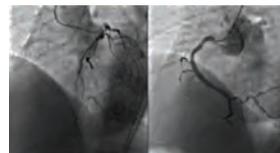


Figure 5. No myocardial bridging in the middle of LAD on LMCA image can be seen during PCI of RCA (left). 100% patency in RCA was achieved after PCI of RCA (right).

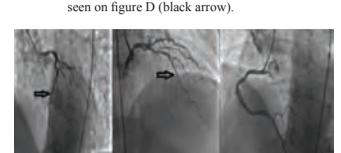


Figure 6. 3 months later coronary angiography images of the patient. RCA is open and no myocardial bridge in the middle of LAD can be seen.

### Implication of serum neutrophil gelatinase-associated lipocalin levels in patients who underwent first-time diagnostic coronary angiography

#### İlk kez tanısal koroner anjiyografi yapılan hastalarda serum nötrofil gelatinase-ilişkili lipocalin seviyelerinin etkisi

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**Purpose:** Atherosclerosis is a chronic inflammatory process and systemic inflammation plays key role in each step from endothelial dysfunction, formation of atheromatous plaques, to clinical presentation as symptomatic coronary artery disease (CAD) due to plaque rupture and formation of occlusive thrombus. Neutrophil gelatinase-associated lipocalin (NGAL) is a protein secreted by activated neutrophils which recently demonstrated as a diagnostic marker of CAD. The aim of this study is to investigate clinical relevance of circulating NGAL levels in patients who underwent coronary angiography with suspected CAD.

**Methods:** One hundred and five consecutive patients (49 females, 56 males; mean age 59.7 years) who underwent first-time diagnostic coronary angiography from March to June 2013 were included in this study. Peripheral blood samples were taken from an antecubital vein for determination of hemogram and serum NGAL concentrations before coronary angiography. Patients with impaired renal function and elevated white blood cell counts were excluded. The severity and extent of CAD was evaluated by calculation of vessel and Gensini scores. Also, SYNTAX score was calculated in patients with  $\geq 50\%$  stenosis. Results were reported as mean  $\pm$  standard deviation and percentages. Continuous variables were analyzed using the Student's t-test. A value of  $p < 0.05$  was considered statistically significant.

**Results:** Demographic and clinical characteristics of the patients were listed in Table 1. Patients with CAD (group 2) were significantly older compared to patients without CAD (group 1) and the number of male patients were significantly higher in group 2 ( $p < 0.05$ ). Although white blood cell and polymorphonuclear neutrophil counts were not different in both groups, neutrophil to lymphocyte ratio (NLR) was significantly elevated in group 2 ( $p = 0.04$ ). Serum NGAL levels in patients with CAD were significantly higher than those in patients without CAD ( $11.9 \pm 3.3$  vs  $6.0 \pm 1.3$  pg/mL;  $p < 0.01$ ). The NGAL levels were correlated with NLR, the number of diseased vessels and Gensini score but not with the SYNTAX score (Figure 1). The diagnostic value for serum NGAL in discriminating patients without CAD from those with CAD was high (AUC=0.975). If we used as cutoff for serum NGAL 7.5 pg/mL, we could predict presence of CAD with sensitivity and specificity, 92.3% and 97.5%, respectively.

**Conclusions:** We demonstrated that serum levels of NGAL as a biomarker of neutrophil activation are higher in patients with CAD than patients with normal coronary angiograms. It could be used to discriminate patients with CAD before diagnostic coronary angiography. Also, it is positively and significantly correlated with severity and extent of CAD.

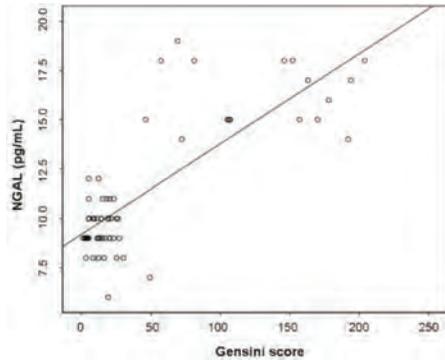


Figure 1. Scatter plot of the serum NGAL levels (pg/mL) against Gensini score ( $r=0.643$ ,  $p<0.01$ ).

Table 1

	Group 1 (n=26)	Group 2 (n=79)	p
Age (years)	54.0 $\pm$ 10.2	61.3 $\pm$ 9.4	<0.01
Male gender (%)	7 (26.9)	49 (62.0)	<0.01
BMI (kg/m <sup>2</sup> )	29.9 $\pm$ 4.0	29.5 $\pm$ 5.2	0.73
Waist circumference (cm)	99.5 $\pm$ 9.5	104.7 $\pm$ 9.9	0.11
Current smoker (%)	6 (23.1)	21 (26.6)	0.72
Hypertension (%)	11 (42.3)	32 (40.5)	0.91
Diabetes mellitus (%)	5 (19.2)	20 (25.3)	0.57
Dyslipidemia (%)	4 (15.4)	15 (19.0)	0.74
WBC (x10 <sup>3</sup> /mL)	7.2 $\pm$ 1.4	7.5 $\pm$ 1.9	0.42
Neutrophil count (x10 <sup>3</sup> /mL)	3.9 $\pm$ 1.0	4.4 $\pm$ 1.2	0.11
Neutrophil/Lymphocyte Ratio	1.7 $\pm$ 0.4	2.0 $\pm$ 0.8	0.04
Vessel score	0	0.8 $\pm$ 1.1 (0-3)	<0.01
Gensini score	0	44.2 $\pm$ 58.5 (1-204)	<0.01
SYNTAX score	0	11.7 $\pm$ 6.6 (2-26)	<0.01
NGAL (pg/mL)	6.0 $\pm$ 1.3	11.9 $\pm$ 3.3	<0.01

Patient demographics and clinical characteristics.

### Simultaneous multivessel stent thrombosis induced by carbon monoxide poisoning

#### Karbon monoksit zehirlenmesinin tetiklediği eş zamanlı çoklu damar stent trombozu

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Carbon monoxide (CO) poisoning is a leading cause of toxicological morbidity and mortality. Brain and heart may be severely affected by CO exposure because these organs are very sensitive to hypoxic injury. Cardiac manifestations of CO exposure have been reported as myocardial ischemia, heart failure and arrhythmias. A 57 year-old Caucasian male patient admitted to the emergency service with the complaints of dizziness, weakness, nausea, vomiting, headache. He had no history of diabetes mellitus and hypertension, he was an ex smoker. He had percutaneous coronary intervention and two bare metal stents were placed in left anterior descending artery (LAD) and first diagonal artery two years ago. He has been receiving medical treatment for coronary artery disease. His vital signs were normal and physical examination revealed no abnormality. The electrocardiogram (ECG) was sinus rhythm with no ischemic changes. Oxygen (O<sub>2</sub>) saturation was 94% by pulse oximetry. Carboxyhemoglobin level was 23% by co-oximetry. The rest of laboratory findings were in normal ranges, including troponin, creatine kinase and creatine kinase-myocardial band (CK-MB). He was diagnosed as acute CO poisoning and received O<sub>2</sub> at a 100% FiO<sub>2</sub> via a non-rebreather mask for treatment of acute CO poisoning. During his follow-up in the emergency room, he started to suffer from accelerating retrosternal squeezing type pain. His control ECG showed sinus rhythm with ST segment depression in lead V4 through V6 (Figure 1). Acute coronary syndrome was considered, 600 mg clopidogrel, and low molecular weight heparin were given to patient. As he had ongoing chest pain and dynamic ECG changes coronary intervention was decided to perform and he was transferred to the cardiac catheterization laboratory. The coronary angiography revealed mobile in-stent thrombi in LAD and first diagonal artery which did not blocking distal flow (Figure 2). Tirofiban infusion was started promptly and continued for 12 hours. He was transferred to coronary care unit where the O<sub>2</sub> therapy using a non-rebreather mask treatment for acute CO poisoning was going on in addition to acute coronary syndrome treatment. Echocardiographic examination showed apical and anterolateral hypokinesis with an ejection fraction 45-50%. The troponin level peaked at 4.8ng/ml and CK-MB peaked at 75 U/L. His chest pain resolved during follow up, and control coronary angiography at the following day revealed both stents in LAD and first diagonal artery were patent, there was no thrombus. He was hemodynamically stable from the cardiac point of view and was discharged in four days. In conclusion, we report a case of simultaneous multivessel stent thrombosis induced by CO poisoning. To our knowledge, this is the first reported case of CO poisoning induced simultaneous multivessel stent thrombosis. In literature there are two cases of stent thrombosis induced by CO poisoning, but both of them involved single vessel.

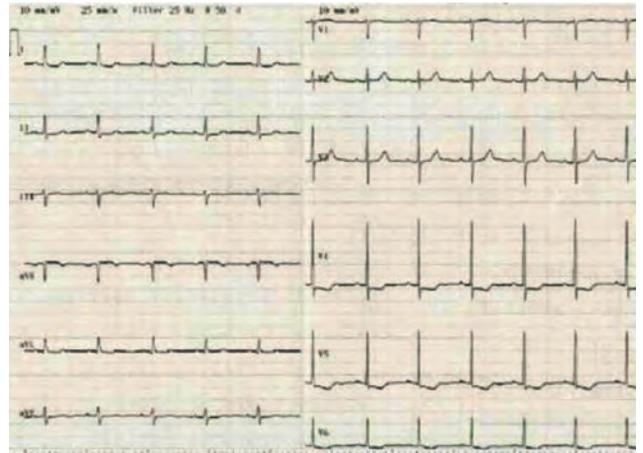


Figure 1. Twelve lead electrocardiogram revealing ST segment depression in leads V4 through V6.

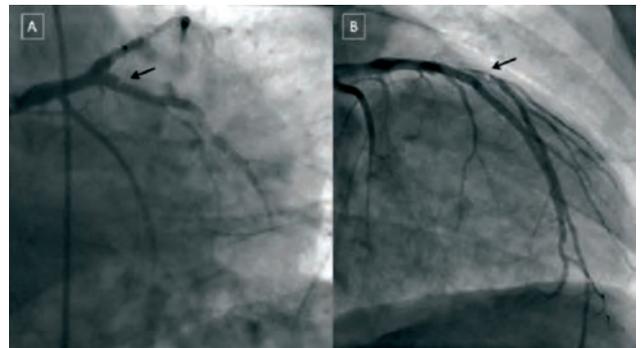


Figure 2. Coronary angiography showing mobile thrombi in the stents in first diagonal artery (A) and left anterior descending artery (B).

### Acute coronary syndrome due to over use of liquid nicotine in a young patient

#### Genç bir hastada sıvı nikotin aşırı kullanımına bağlı akut koroner sendrom

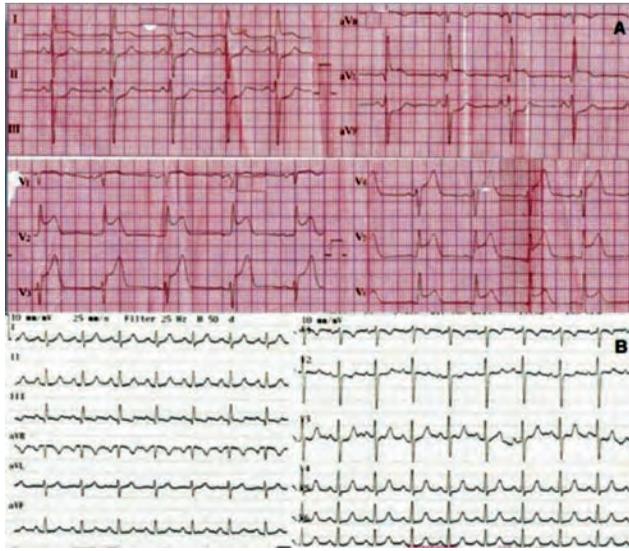
Tarık Kivrak, Murat Sunbul, Ramile Dervisoğlu, İbrahim Sari, Osman Yesildag

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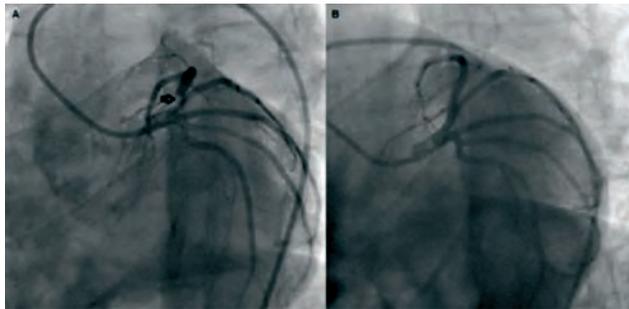
**Objectives:** Myocardial infarction is rare under age of 30. Pathogenesis is known to be different than their older counterparts. Atherosclerotic burden is less and thrombotic burden more prominent in younger patients. Nicotine has toxic effects on endothelium, might alter vascular reactivity, cause vasospasm and activate platelets.

**Case:** A previously healthy 24-years-old male presented to the emergency department with typical chest pain that began 4 hours ago while he was using liquid nicotine (electronic cigarette). Immediate electrocardiogram revealed ST segment elevation on leads DI, aVL and V1-6 with reciprocal ST segment depression and T wave inversion on leads aVF, DII and DIII. He was transferred to cardiac catheterization laboratory which revealed thrombus in the proximal left anterior descending artery. Because patient was young and had no other known risk factors for coronary artery disease, we decided to administer tissue plasminogen activator. After tissue plasminogen activator, chest pain disappeared and electrocardiogram showed more than 70 % of resolution of ST elevation.

**Conclusion:** We present here a rare case of acute myocardial infarction in a 24-years-old male due to liquid nicotine which has not been reported previously. We thought that myocardial infarction was associated with use of high dose liquid nicotine (electronic cigarette).



**Figure 1.** A. Surface ECG showing ST segment elevation on leads DI, aVL and V1-6 with reciprocal ST segment depression and T wave inversion on leads aVF, DII and DIII. B. Surface ECG showing more than 70 % of resolution of ST segment elevation after thrombolytic therapy.



**Figure 2.** A. Coronary angiography showing thrombus in the proximal left anterior descending artery. B. Control coronary angiography showing complete resolution of the thrombus in the proximal left anterior descending artery.

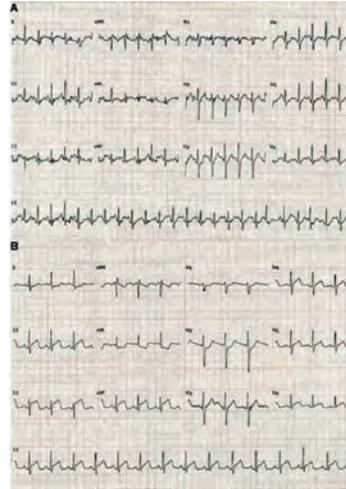
### Asymptomatic ST segment elevation in the recovery phase of exercise stress test due to slow coronary flow

#### Azsemptomatik ST segment yükselmesi egzersiz testinin dinlenme fazında koroner yavaş akıma bağlı

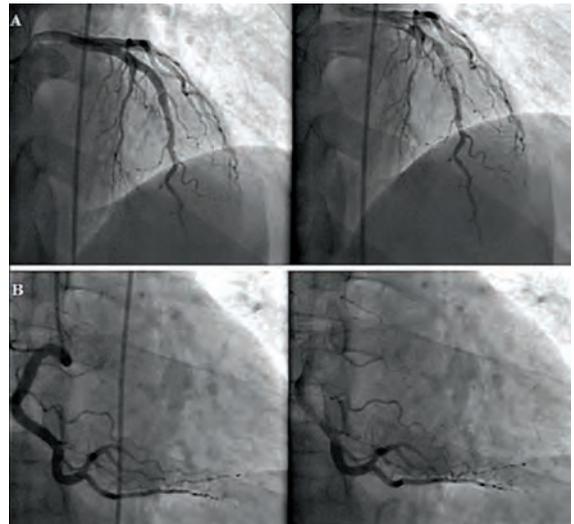
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A 49-year-old man presented with stable angina for more than six months. He had history of smoking and hyperlipidemia. On admission physical examination, resting electrocardiogram and transthoracic echocardiography were normal. He underwent treadmill exercise test with Bruce protocol. The patient tolerated exercise stress test (he was able to exercise 9 minutes and maximal heart rate was 158/min). However, during therecovery period (2 minutes and 57 seconds after starting recovery period) he suddenly developed ST segment elevation in leads D2, D3, aVF and V4-6 associated with reciprocal ST segment depression in leads D1, aVL, V1-3 (Figure 1). The patient was asymptomatic. After sublingual nitroglycerin administration ST segment elevation gradually resolved in about 30 minutes. Coronary angiography (within 45 minutes of diagnosis) revealed normal coronary arteries with no apparent significant stenosis. However, coronary flow was significantly slowed in all coronary arteries (Figure 2). The Thrombolysis In Myocardial Infarction (TIMI) frame count was 68 for left anterior descending artery (normal: 36±1), 52 for circumflex artery (normal: 22.2±4) and 44 for right coronary artery (normal: 20.4±3). The patient was reassured and discharged with acetylsalicylic acid 100 mg q.d, diltiazem 90 mg b.i.d, atorvastatin 20 mg q.d and isosorbidmononitrate 40 mg b.i.d. Exercise induced ST segment elevation without Q wave is rarely observed. Frequent causes are significant coronary stenosis, myocardial bridge and coronary vasospasm. Both exercise induced ST segment elevation due to slow coronary flow and ST segment elevation in the recovery phase of the exercise stress test are very rare. We present a 49-year-old man with asymptomatic inferolateral ST segment elevation in therecovery phase of exercise stress test due to slow coronary flow which has not been reported previously. The learning points of the present paper are; a) although significant coronary stenosis, myocardial bridge and coronary vasospasm are frequent causes of exercise induced ST segment elevation, slow coronary flow might also cause it, b) one should keep in mind that ST segment elevation might also occur in the recovery phase and c) ST segment elevation might be asymptomatic as in the present case.



**Figure 1.** A) Exercise stress test during maximal exercise. B) Exercise stress test at recovery phase with elevated ST segments in leads D2, D3, aVF, V4-6 and depressed ST segment in leads D1, aVL, V1-3.



**Figure 2.** A) Right cranial view of left anterior descending and circumflex arteries during filling (left) and wash-out phase (right). B) Right anterior oblique view of right coronary artery during filling (left) and wash-out phase (right).

OS-85

### Acute anterior myocardial infarction due to stent thrombosis after bee stings

#### Arı sokması sonrası stent trombozuna bağlı akut anterior miyokard enfarktüsü

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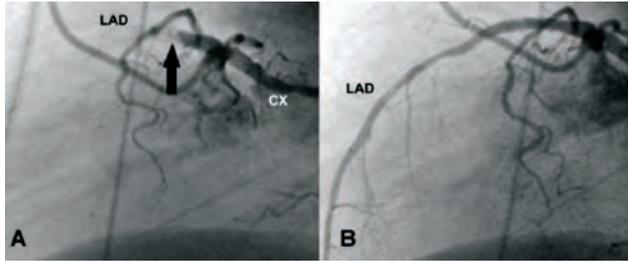
<sup>2</sup>Necip Fazıl City Hospital, Cardiology Department, Kahramanmaraş

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<sup>4</sup>Afsin State Hospital, Internal Medicine Department, Kahramanmaraş

<sup>5</sup>Sutcu Imam University, Faculty of Medicine, Cardiology Department, Kahramanmaraş

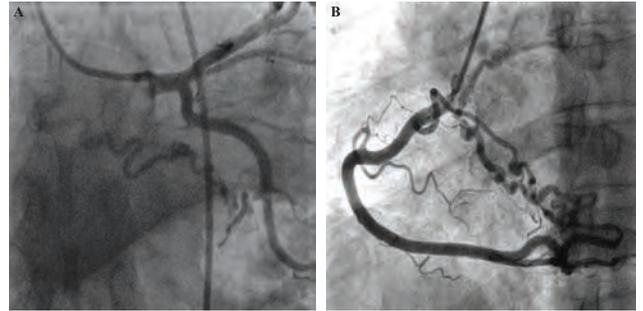
A 60-years-old man admitted to emergency department with chest pain and pruritus. He was diabetic and two months ago his left anterior descending artery was stented. He was exposed to multiple bee stings 4 h prior to presentation. On physical examination vital signs were stable. A 12-lead electrocardiogram showed anterior ST-segment elevation. With the aim of primary percutaneous coronary intervention he was transferred to catheter laboratory. Selective coronary angiography demonstrated that the left anterior descending artery stent was totally occluded with thrombus (Figure 1A). Non-critical lesions were visualized in other coronary arteries. The lesion was passed with wire easily and appropriate distal flow was achieved after balloon inflation (Figure 1B). Follow-up period in coronary care unit was uneventful and he was discharged without any complication. A diagnosis of Kounis syndrome secondary to bee sting was made. In contrast to current literature, this is the first stent thrombosis case described after hymenoptera envenomation. Two types of this syndrome have been defined. In type 1 the coronary arteries are normal and coronary vasospasm secondary to hypersensitivity reaction is responsible mechanism; in type 2 there is underlying coronary artery disease and hypersensitivity reaction leads to plaque erosion and coronary occlusion.



**Figure 1.** Lateral view of left coronary system. A. Black arrow shows thrombus in left anterior descending artery (LAD) stent. Cx: Circumflex artery B. Lateral view after balloon inflation. TIMI-3 coronary flow was achieved.

ischemia. Cardiac enzymes remained at normal levels after the procedure. The patient reported resolution of his symptoms, and he was discharged on the following day. He has been followed up clinically 3 months after the procedure and he has been asymptomatic.

Koroner arter fistülleri, koroner arterlerin en sık görülen doğumsal anomalileridir fakat iki taraflı çok sayıda fistül nadir rastlanılan bir durumdur. Koroner arter fistülüne bağlı semptomları olan hastaların güncel tedavi seçenekleri, perkütan yolla ya da cerrahi olarak defektin kapatılmasıdır. Koil yardımıyla fistüllerin transkateter kapatılması yöntemi, cerrahiye alternatif, etkin ve güvenli bir yöntemdir. Bu olgu bildirisinde, hem sağ hem de sol koroner arterlerden sağ pulmoner artere açılan birden çok fistülün transkateter yöntemle başarılı şekilde tedavi edildiği bir olguyu sunduk. Otuz üç yaşında erkek hasta tipik göğüs ağrısı ile mürcaca etti. Koroner anjiyografide sağ koroner arter (RCA) ve sol sirkümlüks koroner arter (Cx) ile sağ pulmoner arter (PA) arasında çok sayıda fistül izlendi. Tedavi yöntemleri kalp damar cerrahisi ve hasta ile tartışıldı ve hastanın eşlik eden koroner arter hastalığının olmaması ve cerrahi istemesi üzerine perkütan yolla kapatma işlemi planlandı. Lokal anestezi altında sağ femoral artere 6 Fr kılıf yerleştirildi. Sol ana koroner artere 6 Fr EBU 3.75 kılavuz kateter ile oturuldu. İlk olarak hidrofilik bir kılavuz tel ile Cx'den PA'ya olan fistül geçildi. 2.5 mm x 3 cm, 3.0 mm x 4 cm, ve 2.0 mm x 4 cm embolizasyon koilleri (Barricade Coil System, Blockade Medical, Irvine, CA, USA) bir mikrokater (Vasco Plus Braided Microcatheter, Balt Extrusion, Montmorency, France) içerisinde damarın distaline taşındı ve serbestleştirildi. Kontrol anjiyografide fistülün tkandığı izlendi (Fig 2a). Daha sonra 6 Fr JR 4.0 kılavuz kateter ile RCA'ya derin oturuldu. 0.014" hidrofilik bir tel (Asahi Sion, Abbott Laboratories, Abbott Park, IL, USA) yardımıyla posterolateral dal ile PA arasındaki fistül geçildi. 4.0 mm x 6 cm ve 2.0 mm x 4 cm embolizasyon koilleri kullanılarak fistülöz bağlantı kapatıldı (Fig. 2b). Son olarak proksimal RCA ile PA arasındaki fistül, 3.0 mm x 8 cm, 2.0 mm x 4 cm ve 3.0 mm x 4 cm koiller kullanılarak kapatıldı (Fig. 2c). İşlem sonrası koil migrasyonu, koroner arter diseksiyonu, miyokard enfarktüsü, inme gibi majör komplikasyon gelişmedi. İşlem boyunca hastanın hemodinamisi stabildi ve miyokard iskemisi göstergesi elektrokardiyografik değişiklik izlenmedi. Kardiyak enzimlerde artış olmadı. Hasta şikayetlerinin geçtiğini ifade etti ve ertesi gün taburcu edildi. Üç aylık takibinde asemptomatikti.



**Figure 1.** (A) Coronary angiography showing multiple coronary fistulae from the branch of the left circumflex coronary artery and right pulmonary artery. (B) Coronary angiography showing multiple coronary fistulae from the branches of the right coronary artery.

OS-86

### Successful transcatheter closure of bilateral multiple coronary arterial fistulae in one session

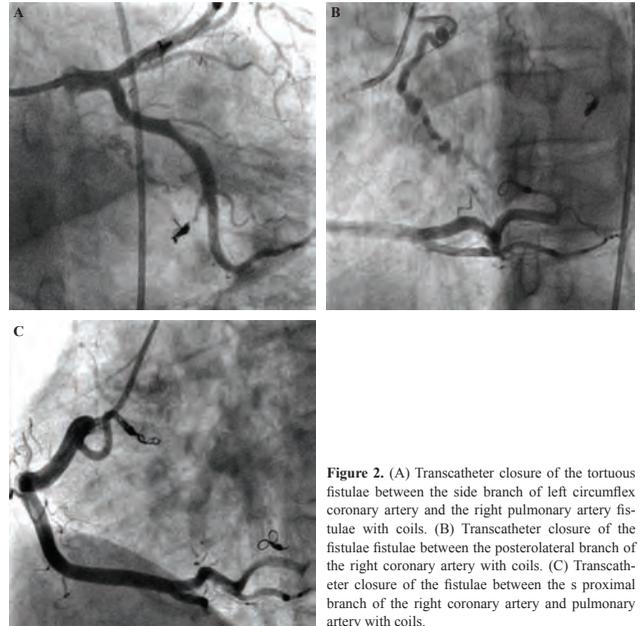
#### Çok sayıda koroner arter fistülü bulunan bir vakanın aynı seansta transkateter yolla kapatılması

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Coronary artery fistulae represent the most frequent congenital anomalies of the coronary arteries, but, multiple bilateral fistulae are a rare condition. Current therapeutic options for symptomatic patients are percutaneous closure and cardiac surgery. Transcatheter closure of fistulae using coils preferred as an effective and safe alternative to surgery. Here we reported the case of a patient with congenital coronary artery fistulae arising from both the left and right coronary arteries draining individually into the right pulmonary artery treated successfully with transcatheter approach.

A 33-year-old man presented with symptoms of anginal chest pain. Coronary angiography revealed patent coronary arteries and multiple fistulae between right coronary artery (RCA), left circumflex coronary artery (Cx) and the right pulmonary artery (PA) (Fig. 1). Treatment strategies were discussed with the patient and cardiac surgeons. Since the patient was young, free of atherosclerotic coronary artery disease, and unwilling to undergo surgical closure, the percutaneous approach was chosen. The procedure was carried out under local anesthesia with sedation and a 6 Fr sheath was inserted in the right femoral artery. The left main coronary artery was cannulated with a 6 Fr extra back-up 3.75 guiding catheter. Initially, a guidewire negotiated from Cx to PA through the fistula. Embolization coils (2.5 mm x 3 cm, 3.0 mm x 4 cm, and 2.0 mm x 4 cm) were delivered to the distal vessel via a micro catheter (Vasco Plus Braided Microcatheter, Balt Extrusion, Montmorency, France). Control angiography demonstrated occlusion of the fistulous communication between Cx and PA (Fig 2a). Then, the right coronary artery was cannulated with a 6 Fr JR 4.0 guiding catheter and deeply engaged to access the distal RCA. A 0.014" hydrophilic guidewire (Asahi Sion, Abbott Laboratories, Abbott Park, IL, USA) was used to pass through the fistula between posterolateral branch and PA. Embolization coils (4.0 mm x 6 cm and 2.0 mm x 4 cm; Barricade Coil System, Blockade Medical, Irvine, CA, USA) were delivered and maintained successful closure of the fistula (Fig. 2b). Last of all, the fistulous connection between proximal RCA and PA was occluded with the use of 3.0 mm x 8 cm, 2.0 mm x 4 cm, and 3.0 mm x 4 cm coils. There were no major complications such as coil migration, dissection of the feeding vessel or of native coronary arteries, myocardial infarction, death, stroke or infection. The patient's hemodynamics remained stable during the procedure, and there were no electrocardiographic changes indicative of myocardial



**Figure 2.** (A) Transcatheter closure of the tortuous fistulae between the side branch of left circumflex coronary artery and the right pulmonary artery fistulae with coils. (B) Transcatheter closure of the fistulae between the posterolateral branch of the right coronary artery with coils. (C) Transcatheter closure of the fistulae between the proximal branch of the right coronary artery and pulmonary artery with coils.

OS-87

## Giant coronary artery aneurysm stent implantation was successful with optical coherence tomography

### Dev koroner arter anevrizmasına optik koherans tomografi eşliğinde başarılı stent implantasyonu

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Behçet's disease is a multisystem autoimmune disease with variable clinical manifestations. Behçet's disease may affect all types of vessels within the arterial and venous system. The most common presentation is the development of venous thrombosis, which mainly occurs in the lower limbs and is significantly more common in males. Cardiac involvement is a rare manifestation in Behçet's Disease and includes pericarditis, endocarditis with valvular lesions, myocarditis, intracardiac thrombosis, endomyocardial fibrosis, coronary vasculitis and myocardial aneurysm formation. Aneurysms and/or thrombosis of the coronary arteries are observed complicated by hemorragia, myocardial infarction and sudden death. We detected the giant coronary artery aneurysm at the patient who typically chest pain had treated for venous thrombosis. Behçet's disease, we decided that at the end of the etiological research. We have successfully covered stent implantation with optic coherans tomography.

Behçet hastalığı çeşitli klinik tablolarla gelebilin multisistem otoimmün bir hastalıktır. Arteriyel ve venöz sistemde bulunan bütün damarları etkileyebilir. En yaygın görünüm şekli venöz trombozdur. Esas olarak alt ekstremitelerde ve erkeklerde meydana gelir. Behçet Hastalığı'nda kardiyak tutulum nadirdir. Perikardit, kapak lezyonlarıyla birlikte endokardit, miyokardit, kalp içi trombüs oluşumu, endomiyokardiyal fibrosis, koroner vaskülit ve miyokardiyal anevrizma şeklinde görülebilir. Koroner arterlerde görülen anevrizma ve/veya trombüs kanama, miyokard infarktüsü ve ani ölüme komplike olabilir. Daha önceden venöz tromboz nedeniyle tedavi edilen ve tipik göğüs ağrısı ile müraacaat eden hastada dev koroner arter anevrizması tespit ettik. Etiyolojik araştırmalar sonrasında Behçet Hastalığı teşhisi koyduk. Optik koherans tomografi eşliğinde başarılı bir şekilde greft kaplı stent implante ettik.

**Vaka Takdimi:** 35 yaşında daha önceden derin venöz tromboz teşhisiyle takip edilen erkek hasta tipik anjina pektoris nedeniyle müraacaat etti. KAG de sağ koroner arterde yaklaşık 2.8x2.3 cm genişliğinde dev anevrizma ve %98'lik darlık tespit edildi. Etiyolojik incelemeler sonrasında oral ve genital afları da olduğu öğrenilen hastaya romatolojik incelemeler sonrasında Behçet hastalığı teşhisi kondu. Hastaya immün supresif tedavi başlandı. Ateroskleroz bulunup bulunmadığını anlamak için optik koherans tomografi(OCT) çekildi. Ateroskleroz bulunmadığı, darlığın da anevrizmanın basısına sekonder olduğu öğrenildi. OCT ile stentin ölçüsü ve lokalizasyonu kararlaştırıldı. Greft kaplı stent implantasyonu sonrasında yine OCT ile kontrol edilen stentin tam açılmadığı tespit edilmesi üzerine post dilatasyon yapıldı. TIMI 3 akım sağlanması ve tam açıklık sağlanması üzerine işleme son verildi.

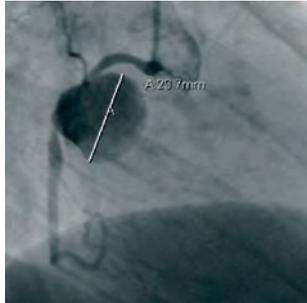


Figure 1. The giant aneurysm of the right coronary artery.

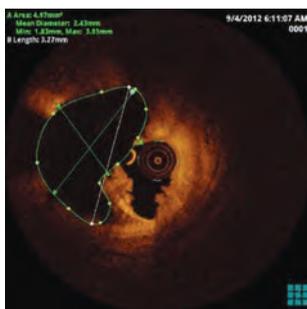


Figure 2. Optic coherans tomography image of the aneurysm.



Figure 3. After the graft covered stent implantation.

OS-88

## Using home-made fenestrated Amplatzer septal occluder for very large PDA and severe pulmonary arterial hypertension in a child

### El yapımı fenestre Amplatzer septal okluder ile kapatılan çok geniş PDA ve ağır pulmoner hipertansiyonu olan vaka

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Nine year old female patient with cachexia, difficulty breathing, with complaints of fatigue was admitted in our clinic. Transthoracic echocardiographic examination showed 15 mm diameter PDA and severe pulmonary hypertension and congestive heart failure symptoms. At the cardiac catheterization, the systemic pressure was 131/72/97 mmHg, the pulmonary artery pressure was 104/62/79 mmHg, pulmonary resistant 6.2 Wood units, pulmonary/systemic resistant ratio was measured as 48%. After the treatment with endothelin receptor antagonist during four months period, cardiac catheterization was repeated. At that time the reactivity test was still unresponsive with pulmonary resistant 4 Wood units. Because of this, we decided closed the defect with home-made fenestrated devices. 19 mm the Amplatzer septal occluder was fenestrated with Seldinger technique on two distinct areas near the waist by 12F regular sheath. Antegrad PDA occlusion device was used in the current path. The patient's clinical condition was observed just before the deployment the device. After the procedure, the patient transferred to the intensive care unit and to take lomedin inhaler using for the pulmonary hypertensive crisis risk. The next day, echocardiography control of the device was stable; left-to-right shunt from the fenestrated device was seen. The patient was discharged with oral bosentan therapy. With partial occlusion in patients with severe pulmonary hypertension is still used with ASD and VSD closure in adulthood with surgical fenestrated patch at the pulmonary hypertensive patients. This is the first time to use home-made fenestration ASD at the very large PDA closure with pulmonary hypertension. And we are planning coil embolization of the fenestrations during the follow-up, if they do not close spontaneously.

Dokuz yaşında kaşektik olan kız hasta nefes almada zorluk, çabuk yorulma şikayeti ile başvurdu. Ekokardiyografisinde 15 mm çapında geniş PDA ve ağır pulmoner hipertansiyon ve konjestif kalp yetmezliği bulguları mevcut idi. Kalp kateterizasyonunda, sistemik basıncı 131/72/97 mmHg, pulmoner arter basıncı 104/62/79 mmHg, pulmoner direnç 6.2 Wood unit, pulmoner/sistemik direnç oranı %48 olarak ölçüldü. Endotelin reseptör antagonistini tedavisi başlanan hastaya 4 ay sonra tekrar kalp kateterizasyonu yapıldı, yapılan reaktivite testinde pulmoner direncin 4 Wood ünitesi olduğu, reaktivite testine çok iyi yanıt alınmadığı görüldü. Bunun üzerine 19 mm Amplatzer septal okluder Seldinger tekniği ile santrale yakın 2 ayrı bölgeden 12 F kılıf ile 2 adet 4 mm'lik fenestrasyon açılarak delindi. Mevcut cihaz antegrad yoldan PDA oklüzyonunda kullanıldı. Oklüzyon sonrası beklenecek hastanın klinik durumu izlendi. Semptom gelişmeyen hasta 1 gün yoğun bakım ünitesinde inhaler lomedin tedavisi verilmek üzere yatırıldı. Ertesi gün eko kontrolünde cihazın stabil olduğu, cihazın 2 ayrı bölgesinden küçük sol-sağ şanlı defekt olduğu görüldü. Hasta oral Bosentan tedavisi ile taburcu edildi. Ağır pulmoner hipertansiyonlu hastalarda parsiyal oklüzyon ile kapatma tekniği erişkin yaş grubunda ASD kapatılmasında ve konjenital kalp cerrahisinde valvüli VSD kapatılmasında halen kullanılmaktadır. Pulmoner hipertansiyon krize karşı önlem olmak üzere çok geniş olan bu PDA'nın el yapımı fenestrasyon ile kapatılması ilk defa uygulanmıştır. Mevcut fenestrasyonlar kendiliğinden kapanmaz ise daha sonra coil embolizasyonu planlanmaktadır.



Figure 1. Very large PDA at the lateral position.



Figure 2. Making a hole to Amplatzer septal occluder with 12F sheath using Seldinger technique.

### Single crossover stenting from the left main to the high obtuse marginal artery in combined distal left main shaft and high obtuse marginal lesions: the first case in the literature

**Kombine distal sol ana gövde ve yüksek obtus marjinal lezyonlarda sol ana koronerden yüksek obtus marjinal artere tek crossover stentleme: Literatürdeki ilk vaka**

Mehmet Bilge<sup>1</sup>, Ayşe Saatci Yasar<sup>2</sup>, Recai Alemdar<sup>2</sup>, Sina Ali<sup>2</sup>, Özgür Kırbas<sup>2</sup>, Turgay Aslan<sup>2</sup>, Cemal Köseoğlu<sup>2</sup>, Özge Kurmuş<sup>2</sup>, Bilge Karaduman Duran<sup>2</sup>, Mehmet Erdoğan<sup>2</sup>, Mustafa Duran<sup>2</sup>, Serkan Sivri<sup>2</sup>, Hakan Sütyün<sup>2</sup>

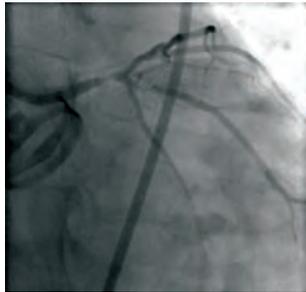
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**Introduction:** Left main (LM) lesions are mainly found in the distal LM bifurcation and isolated distal LM shaft lesion without involving bifurcation is rare. However, frequency of combined significant isolated distal LM shaft and high obtuse marginal artery lesions without significant ostial LAD and CX lesion is strictly unknown. Here we report, to the best of our knowledge, the first case of combined distal LM shaft and high obtuse marginal artery stenosis successfully treated by single crossover stenting, showing the technical feasibility of such procedure.

**Case:** The patient was a 57-year-old man with a chief complaint of chest pain. His medical history included surgical aortic valve replacement in 2007. Coronary angiogram showed 95% stenosis at the distal LM shaft portion not extending to ostia of LAD and LCX and a very critical lesion in the ostium of high obtuse marginal artery (Figure 1). The LAD and high obtuse marginal artery were nearly equal in vessel size. There were no significant lesion in the LAD, CX body and RCA. The calculated Euroscore and Syntax scores were 2.3 and 14, respectively. LM artery was selectively cannulated with a 7F JL 4 catheter. Two 0.014-inch Prowater guide wires were advanced into the distal LAD and the high obtuse marginal artery. Both lesions were dilated sequentially with a Ryujin 2.5 mm x 15 mm balloon at 16 atm. Then, an IVUS examination revealed a distal reference diameter of 3.3 mm in the high obtuse marginal artery and a proximal reference diameter of 4.7 mm at the proximal LM. A Xience 3.5 mm x 23 mm stent was deployed from the mid LM artery to the high obtuse marginal artery at 12 atm followed by post dilatation of the LM stent portion (proximal optimization technique) with a 4.5 mm x 10 mm NC balloon at 24 atm (Figure 2). The postprocedural IVUS study showed an MSA of 5 mm<sup>2</sup> in the high obtuse marginal artery, an MSA of 6.1 mm<sup>2</sup> in the ostium of high obtuse marginal artery, and an MSA of 10 mm<sup>2</sup> in the proximal LM artery with good stent expansion and apposition. The ostia of LAD and CX branches were not compromised after stenting (Figure 3).

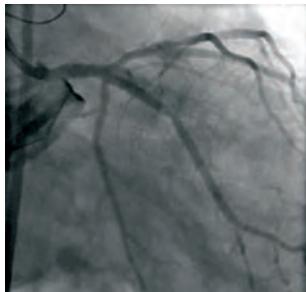
**Discussion:** Isolated distal LM shaft lesions without significant ostial LAD and CX lesion would ideally be treated by stenting from the LM into the LAD. Single crossover stenting from the LM to the CX artery is rarely performed. In our case, we considered that single crossover stenting from the LM to the high obtuse marginal artery had the advantage of providing the opportunity of simultaneous treatment of both lesions. Moreover, because there was lesion in distal LM shaft portion not extending to ostia of LAD and LCX, we selected single crossover stenting technique. This report shows that single crossover stenting could be safe, feasible and effective treatment in the treatment of combined distal LM shaft and high obtuse marginal artery stenosis if there is no significant mismatch between the size of the LM and the high obtuse marginal artery.



**Figure 1.** Left coronary artery angiogram showed 95% stenosis at the distal LM shaft portion not extending to ostia of LAD and LCX and a very critical lesion in the ostium of high obtuse marginal artery.



**Figure 2.** It showing post dilatation of the LM stent portion (proximal optimization technique) with a large NC balloon.



**Figure 3.** Final left coronary artery angiogram showing satisfactory angiographic result.

### Simultaneous stenting of distal cervical internal carotid artery and internal carotid artery bifurcation lesions with proximal embolic protection device (Mo.Ma): the first case in the literature

**Kombine distal sol ana gövde ve yüksek obtus marjinal lezyonlarda sol ana koronerden yüksek obtus marjinal artere tek crossover stentleme: Literatürdeki ilk vaka**

Mehmet Bilge<sup>1</sup>, Sina Ali<sup>2</sup>, Ayşe Saatci Yasar<sup>2</sup>, Recai Alemdar<sup>2</sup>, Özgür Kırbas<sup>2</sup>, Turgay Aslan<sup>2</sup>, Cemal Köseoğlu<sup>2</sup>, Bilge Duran<sup>2</sup>, Özge Kurmuş<sup>2</sup>, Mehmet Erdoğan<sup>2</sup>, Mustafa Duran<sup>2</sup>, Serkan Sivri<sup>2</sup>, Zeynep Seyma Turinay<sup>2</sup>

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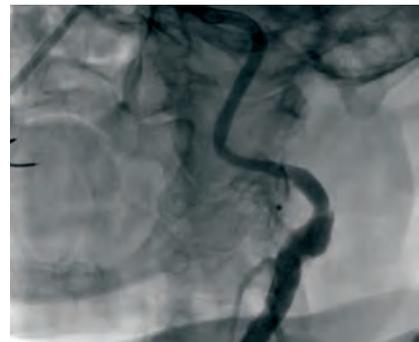
**Introduction:** Distal cervical internal carotid artery (ICA) lesion is a very rare occurrence, representing 0.4 % of patients with carotid disease requiring treatment. But, frequency of combined distal cervical ICA and ICA bifurcation lesion is strictly unknown. The management of this anatomic distribution of disease can be a challenge to plan and perform. A distal embolic protection device (EPD) would face difficulty in the distal ICA lesions because there is a short landing zone for the distal EPD and the distal vessel stretch continues into the skull. Although carotid artery stenting with the Mo.Ma system is one of the theoretically possible technique in the simultaneous treatment of distal cervical ICA and ICA bifurcation lesions, there is no published case available on the use of this technique. The patient is, to the best of our knowledge, the first case of simultaneous stenting of distal cervical ICA and ICA bifurcation lesions with Mo.Ma proximal EPD in the literature.

**Case:** A 70 year old man with asymptomatic left ICA stenosis was referred to our cath lab for carotid angiography. Selective left carotid angiogram showed subtotal left ICA stenosis and a critical lesion in the distal cervical ICA before enters the carotid canal of the petrous bone (Fig.1). We considered that proximal embolic protection had the advantage of providing the opportunity of simultaneous treatment of both lesions. Moreover, because there was a short landing zone for the distal EPD and the ICA bifurcation lesion was subtotal, we selected proximal embolic protection with the Mo.Ma system. The external carotid artery was selectively cannulated with a 5 Fr vertebral catheter over a hydrophilic 0.035-inch wire guidewire. A 300 cm stiff 0.035-inch Supracore wire was then positioned in the distal portion of the artery. Then the Mo.Ma proximal protection device was advanced into the CCA. A 0.014-inch coronary angioplasty guidewire with intermediate stiffness was advanced to the subtotal ICA bifurcation lesion but crossing was unsuccessful. The wire was then exchanged to a Miracle 3 gr stiff coronary angioplasty guidewire. After this, the lesion was crossed, and pre-dilatation of the subtotal lesion was performed. Initially, self-expanding carotid stent was deployed into the distal ICA lesion. Stent postdilatation with a balloon was performed. Then, the second self-expanding stent was successfully deployed into carotid bifurcation that was post-dilated with slightly an undersized balloon (Fig.2).

**Discussion:** Although ipsilateral distal ICA stenosis can be regarded as a relative contraindication to proximal protection device because obscured visualization of the target lesion during stent deployment makes distal ICA stenosis management more difficult, in our case, we did not observe any technical difficulty. The present case shows that CAS with proximal embolic protection could be safe and technically feasible in combined distal ICA and ICA bifurcation stenosis.



**Figure 1.** Left carotid angiogram after inflation of the distal balloon of Mo. Ma embolic protection device in the external carotid artery shows subtotal left internal carotid artery stenosis and a critical lesion in the distal cervical internal carotid artery before enters the carotid canal of the petrous bone.



**Figure 2.** Left carotid angiogram after deployment of stents.

### A Case of carotid stenting via the transbrachial approach with Mo.Ma proximal embolic protection system in the bovine arch: the first case in the literature

**Transbrakial yaklaşımla arkusta Mo.Ma proksimal emboli koruma sistemi ile karotis stentleme olgusu: Literatürdeki ilk vaka**

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**Introduction:** The Mo.Ma is increasingly being used in preference to a distal protection device alternative in patients with carotid artery stenosis. The Mo.Ma seems to be better in cases with symptomatic complex carotid lesion, because allows the whole procedure to be performed under complete cerebral protection. Carotid artery stenting (CAS) with Mo.Ma system is routinely performed by transfemoral approach due to the large size of the available equipment (8Fr, 9Fr). However, in case of the bovine arch anatomy, CAS with transfemoral approach may become impossible. Here, we present a CAS procedure via the transbrachial approach in a patient with anomalous origin of the left common carotid artery, the so-called bovine arch, in which both the right common carotid artery and the left common carotid artery arise from the brachiocephalic artery. The patient is, to the best of our knowledge, the first case of CAS via the transbrachial approach with Mo.Ma proximal protection system in the bovine arch in the literature.

**Case:** A 76 year old man with history of recent stroke was referred to our cath lab for carotid angiography and CAS. A previous Doppler ultrasound scan documented a critical left ICA stenosis. Aortic angiogram showed type II aortic arch with bovine anatomy (Figure 1). Carotid angiogram confirmed the presence of critical stenosis in the proximal portion of the left ICA (Figure 2). Our patient was characterized by a combination of a lesion at high embolic risk by a distal embolic protection device and type II aortic arch with bovine anatomy unfavorable for transfemoral approach. Thus right brachial approach with a Mo.Ma system was planned for CAS procedure (Figure 3). The external carotid artery was selectively cannulated with a 5 Fr headhunter catheter over a hydrophilic 0.035-inch guidewire. A 300 cm stiff 0.035-inch Supracore wire was then positioned in the distal portion of the artery. Then, an 8F Mo.Ma proximal protection device was advanced into the CCA. Once correct positioning and orientation of the device have been confirmed by angiography, the distal and proximal balloons were inflated. A 0.014-inch floppy guidewire was advanced to the left internal carotid artery and a self-expanding carotid stent was deployed into the proximal ICA lesion (7 mm x 30 mm). Then, stent postdilatation with a 5 mm x 20 mm balloon was performed (Figure 4).

**Conclusion:** CAS with Mo.Ma system is routinely performed by transfemoral approach due to the large size of the available equipment (8Fr, 9Fr). But, in our case, brachial approach with the Mo.Ma system was chosen technique because of bovine arch anatomy unfavorable for transfemoral approach and a lesion at high embolic risk for a distal embolic protection system. Carotid stenting via the brachial artery in the presence of bovine arch with Mo.Ma proximal embolic protection device appears feasible as an alternative to standard femoral access.

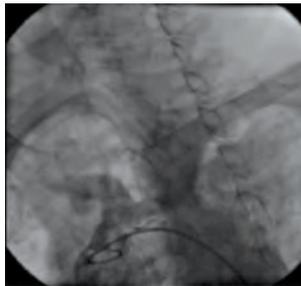


Figure 1. Aortic angiogram shows type II aortic arch with bovine anatomy.

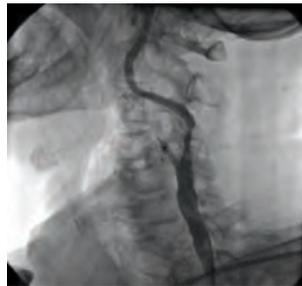


Figure 2. During the ECA balloon occlusion, angiography shows critical ICA lesion.



Figure 3. Advancement of the Mo.Ma system from right brachial approach is seen.



Figure 4. An angiography shows good result in the ICA.

### Occlusion of side branch of recanalised internal mammary artery causing coronary steal syndrome: is it an optimal alternative treatment?

**Koroner çalma sendromuna yol açan rekanalize internal mamarian arterd yan dal tıkanıklığı: Bu optimal tedavi alternatifidir mi?**

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**Case:** 57-year-old female patient was admitted to our clinic with typical anginal symptoms. The patient, who had CABG (Coronary Artery By-pass Graft) operation 10 years ago, underwent coronary angiography because of anginal symptoms four years ago. Unligated large internal mammary artery, caused coronary steal syndrome had been identified and after that treated with occluding with coil. The patient presented with typical anginal complaints was taken coronary angiography. At the IMA side branch, TIMI 3 flow was observed (Figure 1). With left brachial approach, 0,014 wire (Terumo guide wire GT with gold coil) advanced to the lateral internal thoracic artery. Microcatheter (Terumo progreat micro catheter system) was advanced over the wire delivery system. Then four coils, which were 2mm/2cm, were released (coil system MICROPLEX 10, helical soft) and disappearing of flow was observed after 5 minutes (Figure 2). The treatment procedure finished without any complications and the patient was discharged. There are no any symptoms after her 6th month control.

**Discussion and Conclusion:** Unligated large side branches of the LIMA can be potential cause of angina following coronary artery surgery and occlusion of the large branch vessel abolishes angina. Transcatheter coil, gelatin sponge or Amplatzer vascular plug occlusion of IMA side branch is successfully performed to treat coronary steal syndrome. At IMA side branch occlusion, shortening the operation time and reducing the amount of radiation make the risk of recanalization lower. From that point, using vascular plug can be more rational. However, an important disadvantage of this treatment alternative is that devices in use have large diameters. These devices should be 30-50% larger than the vessel diameter and considering that the smallest device's diameter is 4mm in routine practice, the vessel diameter which we will use the device inside, shouldn't be smaller than approximately 2.75 mm diameter. The production of vascular plugs with small diameters would increase the success of IMA side branch occlusion therapy and could be promising to increase the long-term success. Considering IMA side branches diameter which causes the coronary steal phenomenon, the usage of AVP, in most cases, cannot be the appropriate choice nowadays. Under the spot of current data, treatment with coil occlusion, the distance between the coil and the output of the vessel to be occluded, number of coil, diameter and feature (highly structured synthetic-thrombogenic activity) could reduce the possibility of recanalization. To evaluate the selection of the appropriate treatment and the long-term success the method of treatment, long-term and in a large number of follow-up studies are needed.

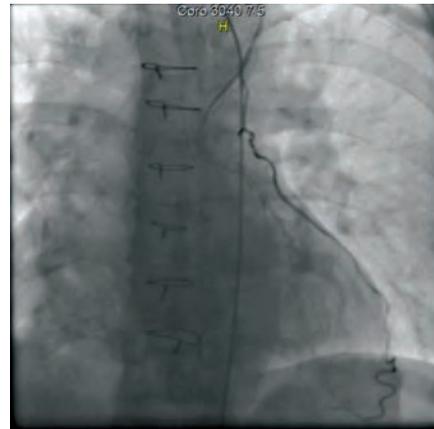


Figure 1



Figure 2

OS-93

### Provisional side branch stenting strategy for renal artery bifurcation lesion

#### Renal arter bifurkasyon lezyonu için provizyonel yan dal stentleme stratejisi

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**Introduction:** Renal artery stenosis is observed ostial and 1/3 proximal renal arteries. Most cases of renal artery stenosis are asymptomatic, and the main problem is high blood pressure that cannot be controlled with medication. Angioplasty with or without stenting is the best option for the treatment of renal artery stenosis due to fibromuscular dysplasia, however stenting is the best technique for atherosclerotic lesions. In the literature, cases with renal artery bifurcation lesions have been treated with implantation of two stents. To the best of our knowledge, this is the first case of renal artery bifurcation lesion which provisional stenting technique was used.

**Case:** 56 years old man with a history of diabetes mellitus and smoking was admitted to our clinic with complaints of chest pain and uncontrolled hypertension. Coronary angiography revealed a 40% stenosis in the left anterior descending artery. Severe stenosis of the right renal artery (90-95%) was seen early bifurcational area by selective renal angiography (Figure 1). We decided to use provisional stenting technique because of the absence of critical stenosis of the side branch ostium. Right renal artery ostium was cannulated with 7F IMA guiding catheter. Then, main branch and side branch were wired with a 0.014 inch Grand Slam wire and a 0.014 floppy wire, respectively. A 7.0x20 mm renal stent was implanted to main vessel so as to protrude 1-2 mm in aorta. Then, stent balloon was pulled back a little bit and stent was postdilated with high atmospheric pressure. After stenting procedure, significant plaque shifting to side branch was seen (Figure 2). Side branch was re-wired with another third wire and the jailed side branch wire was withdrawn. A 4.0x15 mm noncompliant balloon to the side branch and a 5.0x14 mm noncompliant balloon to the main branch were sent, respectively. Both of branch were postdilated with kissing balloon technique (Figure 3). When optimal result was seen, procedure was ended (Figure 4).

**Conclusion:** Provisional stenting is a simple and safe technique for coronary bifurcation lesions. This technique can be applied safely in the presence of suitable anatomy in patients with renal artery bifurcation lesions. However, the long-term results are not known. Larger studies will be needed before its uses become common.



Figure 1. Selective renal angiography shows bifurcation lesion of the right renal artery.



Figure 2. After stenting procedure, an angiography shows significant plaque shifting to side branch.



Figure 3. Both of branch are postdilated with kissing balloon technique.

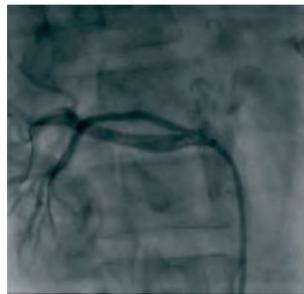


Figure 4. After kissing balloon dilatation, an angiography demonstrates optimal result.

OS-94

### Severe mitral stenosis and acute renal artery occlusion

#### Ciddi mitral darlık ve akut renal arter tıkanıklığı

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**Introduction:** Mitral stenosis and acute renal artery occlusion is a rare but important clinical pathology. Renal arteries embolisms are associated with cardiac diseases and arrhythmias. We describe here a case of atrial fibrillation and severe mitral stenosis complicated with left renal artery thromboembolism and acute renal failure.

**Case Report:** An 58 years old male who applied our emergency department with a sudden left abdominal and back pain. He had atrial fibrillation and severe mitral stenosis (0.9 cm<sup>2</sup> valve area with 18/11 mmHg gradients). Blood pressure was 185/103 mmHg. Electrocardiography showed atrial fibrillation and heart rate was 153/min (Figure 1). Laboratory studies revealed a creatinine level of 1,68 mg/dl, urea 68 mg/dl and potassium 5,6 mEq/l. A contrast enhanced abdominal computed tomography scan demonstrated filling defects in the left main renal artery. The patient was retrieved angiography laboratory. Renal angiography showed total occlusion of the left main renal artery (Figure 2). A 6-Fr right judkins guiding catheter was located in the left main renal artery proximal part. After this, two floppy wires were pushed into the main renal artery and passed into the renal thrombus (Figure 3). We performed serial balloon dilations with a 4.5 x 25 mm coronary balloon (Figure 4). But blood flow didn't improve after this interventions. Therefore a 4-Fr catheter was placed in the renal artery for thrombus aspiration (Figure 5). We performed aspiration procedure for five times. Blood flow was started but it wasn't good enough. We decided to use intraarterial thrombolytic therapy with tissue plasminogen activator (tPA). Firstly 5 mg bolus tPA infused bolus and five minutes later same dose tPA performed again. We repeated angiography. Angiography showed renal blood supply was better than balloon dilatation procedure and left renal artery opened (Figure 6). But a thrombus particle determined in the main renal artery (Figure 7). We started tPA infusion (40 mg/12 hours) and followed the patient in the intensive care unit. The patient's renal functions did not improve immediately after balloon dilatation, thrombus aspiration and thrombolytic therapy. Serum levels of creatinine started to decrease two days after revascularization. Warfarin was started and a mitral valve operation planned with cardiovascular surgeons. Renal scintigraphy showed 50% of the left kidney functions were protected. On the fifth day after admission the patient was transferred to cardiovascular surgery service.

**Conclusion:** 5+5 mg bolus and 40 mg/12 hours infusion of tPA is a safe and efficient protocol to treatment of the acute renal thromboembolism.

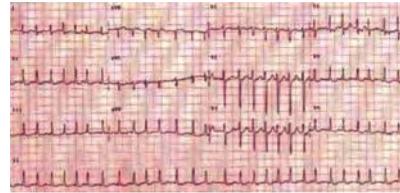


Figure 1. Electrocardiography shows atrial fibrillation.



Figure 2. Renal angiography shows total occlusion of the left main renal artery.

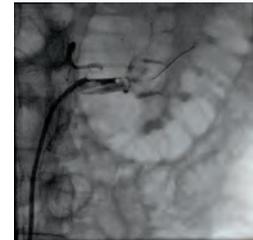


Figure 3. Before renal intervention, a floppy wire located into the renal arteries.



Figure 4. Balloon angioplasty with a coronary balloon.



Figure 5. Thrombus aspiration with a 4-Fr catheter.

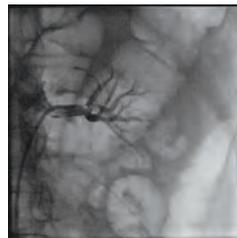


Figure 6. After first bolus dose of tissue plasminogen activator.



Figure 7. After second bolus dose of tissue plasminogen activator.

### Acute myocardial infarction due to simultaneous occlusion of both LAD and RCA complicated with tirofiban induced thrombocytopenia and subacute stent thrombosis

#### *Eş zamanlı LAD ve RCA tıkanıklığına bağlı, subakut stent trombozu ve tirofibanın tetiklediği trombositopeni ile komplike akut miyokard enfarktüsü*

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Acute myocardial infarction due to simultaneous occlusion of more than one epicardial artery is a rare clinical scenario. 57 years old female heavy smoker with no known history of coronary artery disease has admitted to emergency unit with the complaints of chest discomfort, dizziness and weakness. The ECG in has demonstrated ST segment elevation on limb leads (2-3-aVF) as well as precordials (V1-6) and she had significant hypotension (60/40 mmHg) (Figure 1). She directly underwent coronary angiography. On angiography; the occlusion of both RCA and LAD vessels encountered simultaneously (Figure 2,3). Following successful bare metal stent implantation TIMI III blood flow was achieved in both arteries (Figure 4,5). Because of large thrombus burden of both lesions were observed in angiography, tirofiban infusion of (25 mcg/kg IV bolus and maintenance dose of 0.15 mcg/kg per min.) was started immediately. At 8.th hour of tirofiban infusion, control blood counting pointed out severe thrombocytopenia. Platelet count has declined up to 2 K/ml then tirofiban and heparin infusion has been stopped instantly. Platelet count has increased to 47 K/ml with the help of 1 Unit aphaeresis suspension treatment. Platelet count increased in a short time during following days. Patient did not get dual antiplatelet and anticoagulant treatment during only 2 days. Patient was discharged on fifth day of her follow-up without any complaint. One week later after discharge, she has admitted again to emergency unit with acute re-inferior MI. Primary coronary angiography was revealed Stent thrombosis with total occlusion on RCA and some nonobstructive instent thrombus on LAD (Figure 6). Procedure was terminated with additional BMS implantation to RCA after a successful thrombus aspiration. Her follow-up period was uneventful, and she was discharged with intensive antiplatelet therapy as clopidogrel 2 x 75 mg BID and acetylsalicylic acid 300 mg/day. In this case, the most striking points in our interest was simultaneous total occlusion of the two coronary arteries as well as tirofiban-induced thrombocytopenia followed subacute stent thrombosis. It should be noticed that the expected complication of severe thrombocytopenia is not only hemorrhage, but also stent thrombosis we can encounter, probably due to altered medication.



Figure 1. EKG indicating ST elevation in both inferior and anterior localization.



Figure 2. Total occlusion at mid-LAD.



Figure 3. Total occlusion at proximal RCA



Figure 4. Angiogram after 3x24 mm BMS implanted to mid LAD at 16 atm.



Figure 5. Angiogram after 3.5x12 and 3x30 mm BMS to Proximal and mid RCA at 18 atm.



Figure 6. Subacute thrombosis at prior implanted stent.

### Successful treatment of left atrial disk thrombus on a Figulla atrial septal defect occluder with heparin

#### *Figulla atriyal septal defekt tıkaçının sol atriyal diskindeki trombüsün heparin ile başarılı tedavisi*

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A 30-year-old man, with no cardiovascular risk factors, presented with chest pain. He was in sinus rhythm with right bundle branch block on ECG. On transthoracic echocardiographic examination, dilatation of right ventricle was observed. A transesophageal echocardiogram (TEE) demonstrated a 13-mm secundum type atrial septal defect (ASD) with rims that were suitable for percutaneous transcatheter closure. The patient was referred for percutaneous closure. At the start of the procedure, unfractionated heparin (UFH) was administered perioperatively at 7000 IU (100 IU/kg) for anticoagulation. A 18-mm Figulla® ASD Occluder (Occlutech; Jena, Germany) device was implanted. The atrial septal defect was closed successfully. After releasing the device, we observed a 12 x 5 mm mobile mass image in the left atrial face of device immediately after the procedure (Fig 1). Although disc apposition was correct, this mass was resembling like a thrombus. Therefore, we administered an additional 5000 IU UFH after which the procedure was completed. No periprocedural thromboembolic events occurred and treatment was continued with 100 mg acetylsalicylic acid, 75 mg clopidogrel, and an infusion of UFH (aPTT between 50-70 seconds) for two days. A follow-up TEE performed at 48 hours after the procedure and no thrombus formation was observed on the device (Fig 2). TEE revealed correct positioning of the closure device and no signs of residual atrial shunt. Hence, UFH infusion apparently eliminated the thrombus. Moreover, no markers of hypercoagulable state were found positive such as protein C and S deficiency, anticardiolipin antibodies, factor V Leiden mutation. The patient stayed in the hospital for six days, with no complaints. The patient was discharged from the hospital on double anti-aggregation therapy with clopidogrel and aspirin. The further clinical course was uneventful.

**Discussion:** Most recently, transcatheter closure of congenital ASDs has become a less invasive alternative to surgical procedure. Although transcatheter closure is proven to be safe and effective method, there may still be some complications related with this process. One of these complications is the development of device-related thrombus. Due to limited data, there is no consensus on the best management strategy in case of device thrombosis detected during transcatheter ASD closure and there is little information to guide correct management when thrombosis does occur. Different treatment modalities used for treatment in cases of early postprocedure thrombus until now. In this case we observed disappearance of thrombus not only with an 48 hours infusion of heparin, and possibly by physiological fibrinolysis without any sequelae in TEE after 2 days. In conclusion, although Occlutech Figulla Occluder is a safe and effective device to close defects, too early thrombus associated with this device may occur. This case illustrates safety and efficacy of UFH in order to avoid complications in patients with thrombi after ASD closure.

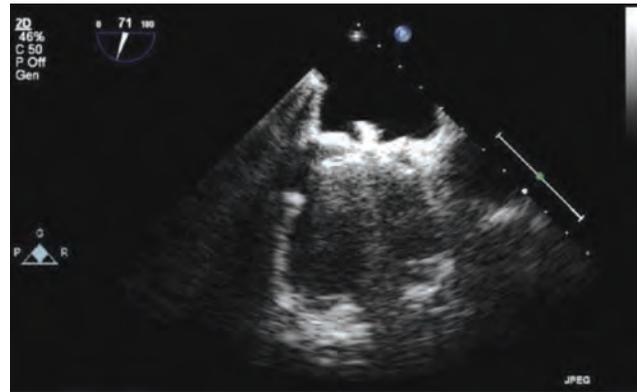


Figure 1. Mass image in atrial face of the device that corresponds to a thrombus.

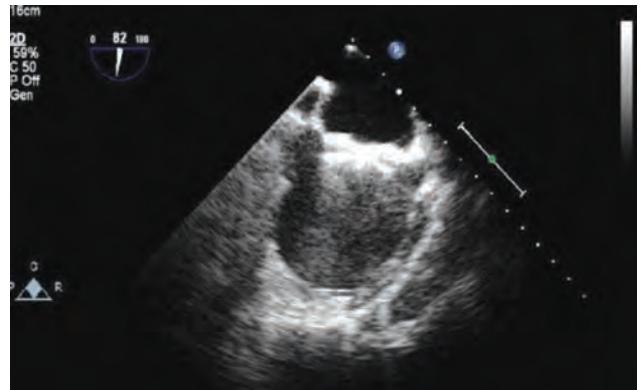


Figure 2. No image of thrombus with normal apposition of the device.