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Case report

Primitive neuroectodermal tumor mimicking left atrial myxoma

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Introduction

Primary cardiac tumours are rare; the incidence ranges from 0.001% to 0.28%.[1] Malignant tumours account for 25% of primary cardiac tumours.[1] Primary primitive neuroectodermal tumour (PNET) is derived from carcinogenic alteration of pluripotent neural crest cells and rarely presents as an organ-based neoplasm.[2] We present a rare case of primary PNET of the heart.

Case history

An 18-year-old boy presented with a 3 months' history of progressively worsening breathlessness and peripheral oedema. There was no significant family history. Echocardiography demonstrated a 58 mm × 49 mm mass, probably left atrial myxoma with no pericardial effusion and good left ventricular function. Myxoma removal was planned two days after echocardiography evaluation, intraoperatively there was firm vascular mass present in left atrium with involvement of interatrial septum and extending into right pulmonary veins, the mass was encasing aorta and pulmonary artery on lateral and posterior surface with involvement of pericardium anteriorly, so procedure was abandoned after incisional biopsy which was sent for immunohistochemistry and histopathological examination (HPE) (Fig. 1). Post-operative high resolution contrast computerized tomography imaging (Fig. 2) revealed evidence of large, lobulated ill-defined multicompartment, heterogeneously enhancing mediastinal nodal mass. The mass had non-enhancing necrotic areas within, seen contiguously involving left lower cervical, aorto-pulmonary window, pre-vascular, pre-subcarinal and bilateral hilar regions. This solid seemingly encapsulated mass was adherent to left atrium and appeared to arise from the posterior free wall of the left atrium. A vascular, nonhomogeneous mass was seen to be densely adherent to the posterior wall of the left atrium and the coronary sinus. HPE report was suggestive of tumour infiltrating the myocardium which was composed of solid sheets of atypical cells with vesicular nuclei, variably visible small nucleoli, and eosinophilic cytoplasm. Mitoses were seen and there was focal tumour necrosis. An associated brisk lymphocytic infiltrate was present in places. No evidence of gland formation, mucin production, or keratinization was apparent. Immunostaining revealed the tumour to be positive for CD 99 (Cluster of Differentiation 99) and vimentin. The tumour was negative for lymphoid markers (leukocyte common antigen, CD 20), endothelial markers (CD 31, CD 34). In view of the immunoprofile, including positivity for CD 99, the diagnosis of PNET was made. The patient was transferred to oncology unit on the sixth postoperative day for oncologic review. Chemotherapy with cyclophosphamide and radiotherapy with 5000 rads per cycle, a total of five cycles were given. A post chemotherapy computerized tomography confirmed tumour shrinkage and no evidence of tumour involvement elsewhere.

Discussion

We present a case of primary PNET of the heart being diagnosed as LA myxoma on echocardiography, received chemo-radiotherapy for the same but ultimately, he succumbed after six months of diagnosis. Since no evidence of tumour was found elsewhere, we propose that this represents a primary PNET of the left atrial free wall. Primary cardiac tumours are rare; the incidence ranges from 0.001% to 0.28%.[1] Malignant tumours account for 25% of primary cardiac tumours.[1] The most frequent primary malignant cardiac tumours are angiosarcomas. Primitive neuroectodermal tumour derives from carcinogenic alteration of

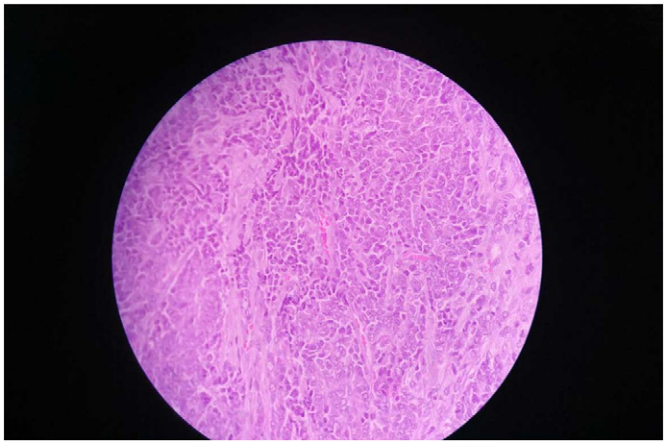


Fig. 1. Histopathological examination image of mass showing solid sheet of atypical cells with vesicular nuclei.

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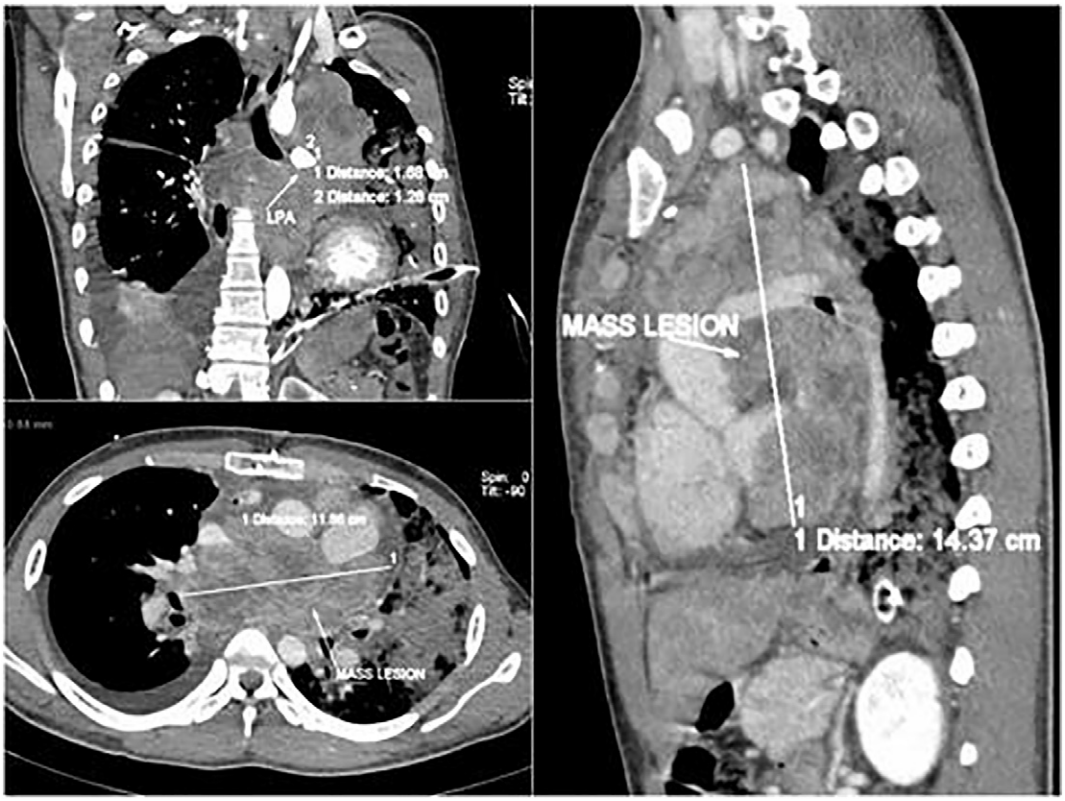


Fig. 2. Computerised tomography scan of thorax suggestive of mass lesion encasing pulmonary artery and superior vena cava.

pluripotent neural crest cells through a balanced reciprocal translocation t (11; 22) (q24; q12).[2] It rarely presents as an organ-based neoplasm and is typically seen in the soft tissues of the chest wall and paraspinal region.[2] There is no clear guidance on the treatment of PNET of the heart. Cardiac neural crest cells participate in endocardial cushion and the septation of the cardiac outflow tract into aorta and pulmonary artery. We acknowledge the possibility that this could be a cardiac metastasis behaving like a primary lesion because primary tumours can undergo spontaneous regression after treatment of metastases.[3] But as there was no evidence of gland formation, mucin production, or keratinization was on HPE, possibility of adenocarcinoma and squamous cell carcinoma can be ruled out. In a 10-year-period review of 18,751 post-mortem examinations, Bussani and associates[1] discovered 7289 cases (39%) of previously undiagnosed malignant neoplasm. Six hundred and twenty-two of these showed evidence of cardiac metastasis (9.1% of malignant tumours). However, the heart was found to be the only target of metastasis in 10 cases (10/7289; 0.14%). The highest rates of cardiac metastases are seen from pleural mesothelioma (48.4%), melanoma (27.8%), lung adenocarcinoma (21%), undifferentiated carcinoma (19.5%), lung squamous cell carcinoma (18.2%), and breast carcinoma (15.5%).[1] PNET of heart carries a poor prognosis. Myxomas are typically pedunculated, with a stalk that is attached to the interatrial septum. For echocardiographically atypical tumours a differential diagnosis should be considered by doing a detailed echocardiography before labelling it as myxoma.

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Nil.

Conflicts of interest

Nil.

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Case report

A case of twitching on the pacemaker pocket

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ABSTRACT

Pacemaker-induced extracardiac stimulation may involve the diaphragm, the pectoral or the intercostal muscles. The common causes are unipolar lead, electrode insulation defects, lead displacement and connector problems. We reported a case of twitching on the pacemaker pocket and discussed the reasons of pacemaker pocket stimulation. After evaluation of the patient we detected an insulation defect on the pacemaker lead that the cause of stimulation on pacemaker pocket.

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Case report

A 73 year old man admitted to our hospital with pain and twitching on the pacemaker pocket. His past medical history included hypertension and right pneumonectomy due to lung cancer. The patient had a VDD pacemaker on the left side, which was implanted 9 years ago due to third degree atrioventricular block. The battery had replaced one year ago due to battery depletion. The cause after pacemaker replacement had uneventful. He felt twitching on the pacemaker pocket last night. The admission electrocardiogram revealed left bundle branch block, exaggerated pacemaker spikes in all leads with sinusal p wave (Fig. 1). His physical examination was unremarkable except rhythmic contraction of the left pectoral muscle. There was no stimulation on another part of his body including diaphragm. The blood pressure was normal, 120/75 mm Hg. The chest radiogram was as shown in Fig. 2. We evaluated him with device interrogation. Electrical measurements revealed a ventricular lead amplitude of 5.0 V, sensitivity 2.80 mV and an impedance of 242Ω. The threshold of ventricular stimulation was 1.6 V at 0.40 ms. The generator of the pacemaker was unipolar. Besides the pacemaker was on VDD mode with ventricular bipolar lead while atrial parameters were stable. The cumulative ventricular pacing was 98%. We can say the ventricular lead impedance was decreased (a fall in lead impedance by more than 250Ω). While pulse amplitude is usually remained as 5.0 V at the time of the pacemaker implantation. In generally the pulse amplitude is decreased after threshold test for pacemaker longevity. In this case the pulse amplitude was remained as 5.0 V. However the main problem was not the high pulse amplitude. We reduced the pulse amplitude to 3.0 V in order to reduce muscle stimulation. After that pectoral muscle stimulation finished instantly. However he was suffering again with twitching on pacemaker pocket in the same evening. The reasons of twitching on the pacemaker pockets were decreased impedance of ventricular lead and the unipolar polarity of the generator. We planned to replace of the ventricular lead. But he refused it because of the risks of complication and decreased lung capacity.

Discussion

Pacemaker-induced extracardiac stimulation normally involves the diaphragm, the pectoral or the intercostal muscles.[1] The usual causes are positioning of the anode directly on the muscle of a unipolar pacemaker with muscle surrounding the pocket, electrode insulation defects and electrode displacement, defective coating of the pacemaker connector problems.[2] If stimulation is on the diaphragm the reason may be about increased lead amplitude. In the present case the pacemaker had unipolar polarity of the generator and decreased impedance of the lead. In this way the pectoral muscle surrounding the pocket was stimulated. Pulse amplitude is usually adjusted as 5.0 V at the time of the pacemaker implantation. But the pulse amplitude must be decreased while threshold test was done. It is more important for pacemaker longevity and pacemaker-induced extracardiac stimulation. On chest radiogram we can see double layer appearance in the lead in pectoralis and subclavian region that is normal for a VDD pacemaker which one of them is atrial sense wire and the other one is ventricular sense and pace wire. The double layer appearance is also seen in some parts of the subclavian vein and vena cava superior which disappears after atrial sense poles. We didn't see the insulation defect on the lead in chest radiogram. Insulation defect was reported that the most common cause of lead failure; insulation defect (32%), followed lead fracture

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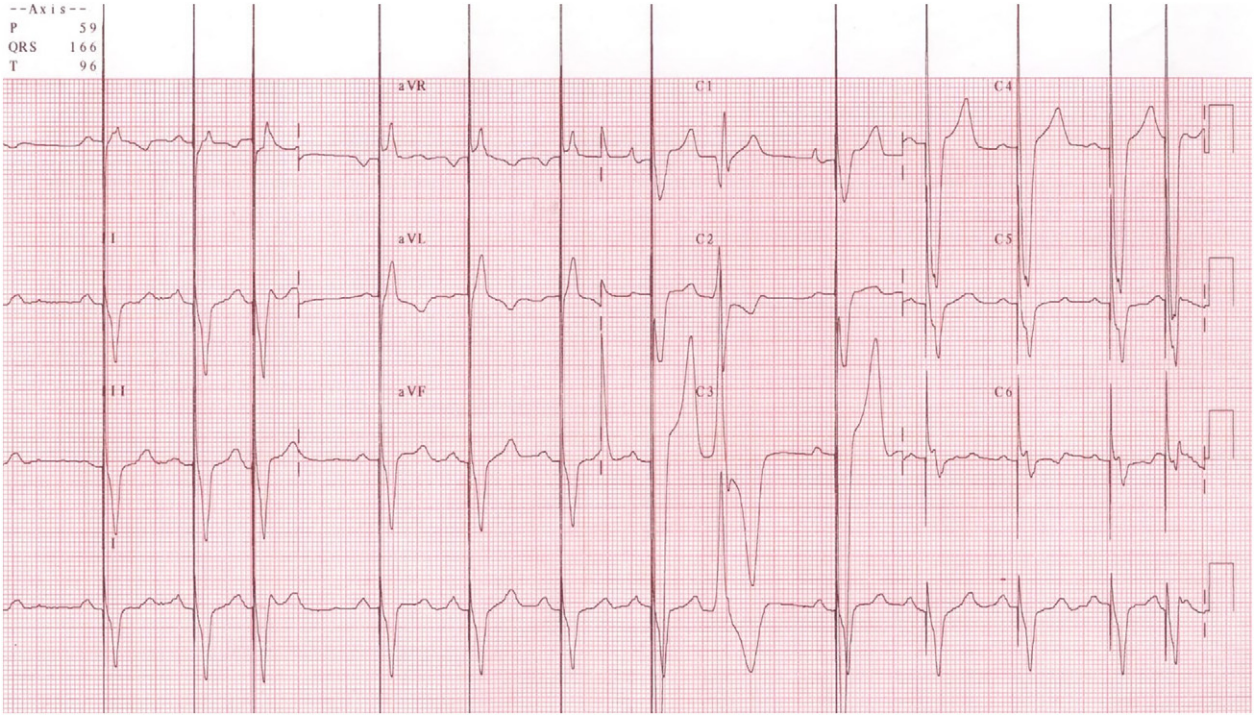


Fig. 1. Electrocardiogram revealed exaggerated pacemaker spikes in all leads and left bundle branch block with sinusal p wave.

(28%), high thresholds (21%), and late dislodgement (14%).[3] Lead insulation defects causing sensing abnormalities and extracardiac stimulation. Depending on the location of the defect, lead repair or lead replacement should be consider.

Twiddler syndrome can also a cause of pacemaker-induced extracardiac stimulation. It is defined as generator rotation with lead twisting and secondary malfunction of the leads. It is a rare but potentially lethal complication of pacemaker treatment. The predisposing factors include the creation of a large subcutaneous pocket, obesity, unconscious repetitive movements of the left arm, manipulation of the pocket, or the adoption of repeated poor postural positions.[4]

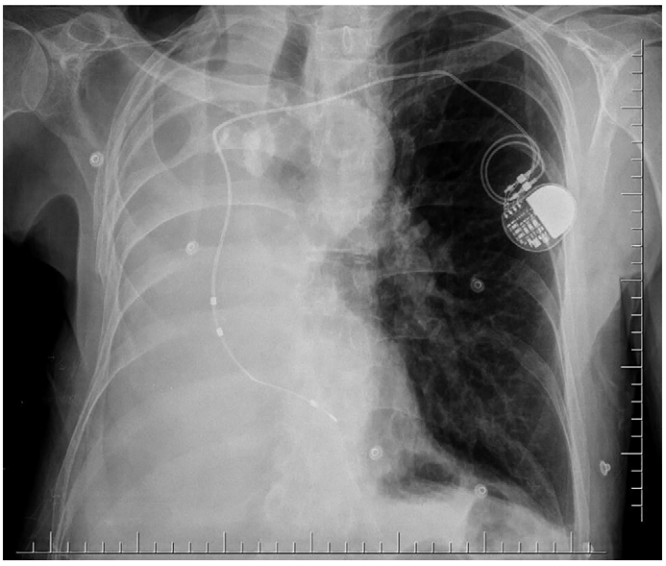


Fig. 2. Chest radiogram showing right pneumonectomy, single chamber permanent pacemaker with atrial sense part on the lead and the insulation defect around the subclavian region.

Lead dislodgement is most likely occured within hours or days of the implant with loss of capture.[5] Pacemaker interrogation revealed an elevated pacing threshold with normal impedance. Fracture of the lead is a cause of persistent sound alarm from the pacemaker. Device interrogation is revealed loss of capture in the ventricular lead and a marked increase in its impedance. Chest radiogram shows the fracture of the lead. We reported a case of pacemaker induced pectoral muscle stimulation due to insulation defect and its management.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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Short communication

Physician preferences for management of patients with heart failure and arrhythmia

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ABSTRACT

Heart failure (HF) is one of the most important causes of morbidity and mortality in the world. It is a progressive and irreversible disorder. Survey and questionnaires are helpful verify that real-life daily practice is keeping with what is recommended in the guidelines. In this questionnaire which is consisted of 32 questions about current diagnostic and therapeutic strategies of HF and arrhythmia, we aimed to evaluate awareness of physicians who are interested in HF and arrhythmias about current diagnostic and therapeutic strategies of HF.

This survey was conducted in university hospitals, state hospitals, training and research hospitals, private hospitals and medical centers in seven geographical region of Turkey between January and February 2017. The study groups were consisted of 177 physicians (163 cardiologists, 7 cardiovascular surgeons, 6 internal medicine physicians and 1 primary care physician). Less than half of the physicians (N:71, 40.1%) have used natriuretic peptides in the diagnosis of HF and 76.3% of physicians were aware of HF with mid-range EF (HFmrEF)—patients with HF and a left ventricular ejection fraction (LVEF) that ranges from 40 to 49%. Despite optimal medical therapy with diuretic, ACEIs, ARBs, BB and mineralocorticoid receptor antagonists (MRAs), the ratio of physicians who have >50% symptomatic patient population were 6.8%. In this patient, angiotensin receptor neprilysin inhibitor (ARNI) was the treatment choice among 62.7% of physicians. Interestingly, 10.2% of physicians have never heard before ARNI. While only 13.6% physicians had >30% patients with HFpEF, most of the physicians (30.5%) had between 21% and 30% patients with HFpEF.

In this questionnaire we want to see a picture from daily practice of physicians who are interested in heart failure. We aimed to measure awareness about current European Society of Cardiology (ESC) heart failure guideline recommendations, new classification of HF according to LVEF and new diagnostic and therapeutic improvements.
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Introduction

Heart failure (HF) is one of the most important causes of morbidity and mortality in the world.[1] It is a big burden to healthcare economy. The prevalence is 0.4 to 2% in general European population[2] and 5 million Americans with chronic HF are mostly attributable to inpatient hospitalization.[3] Currently approximately 26 million adults live with HF in the world ant this number is substantially increasing with aging population. The prevalence is not clearly known in Turkey, but in the HAPPY study it was estimated as 2.9% in adults.[4]

Heart failure is a progressive and irreversible disorder. Therefore, prevention of HF is of great importance. At first, it is required to control risk factors of HF and leading underlying causes and then if the disease has occurred, the guidelines recommended therapy should be implemented. Survey and questionnaires are helpful verify that real-life daily practice is keeping with what is recommended in the guidelines. Health professionals are encouraged to take the guidelines fully into account when exercising their clinical judgement, as well as in the determination and implementation of preventive, diagnostic or therapeutic medical strategies.

In this questionnaire we aimed to evaluate awareness of physicians who are interested in HF and arrhythmias about current diagnostic and therapeutic strategies of HF.

Methods

A questionnaire which is consisted of 32 questions about current diagnostic and therapeutic strategies of HF and arrhythmia was used to evaluate awareness of physicians who are interested in HF and arrhythmia. The questionnaire has been sent to physicians by e-mail and they answered the questions online. At the end of 3 months the questionnaire was finalize and answers were collected. The Statistical Package for the Social Sciences software (IBM SPSS, version 21.0, SPSS Inc., Chicago, IL, USA) was used all statistical calculations.

Results

The study groups were consisted of 177 physicians (163 cardiologist, 7 cardiovascular surgeons, 6 internal medicine physicians and 1 primary care physician). Most of them have worked as a physicians for 5–10 years (N: 103, 58.2%). There were 55 professor and associated professor (38%) and 122 assistant doctor and specialist (62%). Fifty two point five percent of physicians have been worked in state hospital and 33.9% in university hospital. Less than half of the physicians (N:71, 40.1%) have used natriuretic peptides in the diagnosis of HF and 76.3% of physicians were aware of HF with mid-range EF (HFmrEF)—patients with HF and a left ventricular ejection fraction (LVEF) that ranges from 40 to 49%. Only 5.6% of physicians were able to achieve the guidelines recommended target dose of angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) in more than 70% of patients with HF and reduced LVEF (HFrEF). Most of the physicians (69%) were only able to reach the treatment goals in the 41–50% of HFrEF patients. The guideline recommended treatment goals of beta blockers (BB) were achieved in >70% patients by 5.1% of physicians. More than half of the physicians (55.9%) achieved the treatment goals in <30% of patients. Despite optimal medical therapy with diuretic, ACEIs, ARBs, BB and mineralocorticoid receptor antagonists (MRAs), the ratio of physicians who have >50% symptomatic patient population were 6.8%. Twenty seven point one of the physicians (27.1%) had symptomatic patients <25%. In a symptomatic patient despite optimal medical therapy with diuretics, ACEIs/ARBs, BB, MRAs and with sinus rhythm, QRS duration less than 120msn on surface electrocardiogram (ECG), the 83.6% of physicians selected ivabradin therapy, 11.9% digoksin therapy and 4.5% left ventricular assist device (LVAD) or heart transplantation. In this patient, angiotensin receptor neprilysin inhibitor (ARNI) was the treatment choice among 62.7% of physicians. Interestingly, 10.2% of physicians have never heard before ARNI. Digoksin therapy was used <20%of patients with sinus rhythm on ECG among 76% of physicians. At patients with spontaneous echo contrast on echocardiography, 49.7% of physicians have selected antiplatelet therapy, 17.5% warfarin plus antiplatelet therapy and 31.6% no therapy. Among patients with stable angina pectoris, sinus rhythm, under maximum tolerated BB therapy, treatment choice was revascularization among 37.3% of physicians, trimetazidine among 8.5%, ivabradin among 16.9%, ranolazine among 12.4 and long actin nitrates among 24.9%. Most of the physicians (90.4%) have suggested searching for viability before revascularization. Carvedilol was the most chosen BB in HF patients by physicians (59.9%). Respectively, metoprolol succinate (32.8%), nebivolol and bisoprolol (3.4%) and propranolol (0.6%) were the preferred BB. Eighty three percent (83%) of participant center had the possibility of continuous positive airway pressure for non-invasive ventilation. Only 11.3% of diabetic patients were followed by cardiologists, the rest of diabetic patients (88.7%) were referred to endocrinologist. Fifty one (28.8%) patients suggested their patients ICD therapy independently of heart failure etiology, if LVEF is ≤40%. Fourty-five patients (25.4%) considered ICD therapy for their patients, if the heart failure is ischemic origin. Only small amount of patients (11.3%) considered the hypervolemia responsible for hyponatremia >30% of patients. Left ventricular assist device (LVAD) and intra-aortic balloon pump (IABP) were achievable in 117 participant center (66.1%). When the patients were discharged from hospital, symptomatic improvement in 96% of patients, decrease of natriuretic peptides (BNP and NT pro-BNP) in 18.6% of patients, euvolemic status in 73.4% of patients as a treatment success (or surrogate). Diuretic infusion was the treatment choice in the 67.2% of patients and iv. bolus diuretic was chosen in 32.8% of patients. Seventy four percent (74%) of physicians used low dose dopamine with iv. diuretic to increase urine output in their patients. Only 9.6% of patients used the levosimendan as an inotropic support therapy in >10% their heart failure patients. Eighty one percent of physicians (81.4%) said that they would have chosen cardiac resynchronization therapy (CRT) if a pacemaker was necessary in a heart failure patient. We asked the physicians if they had a patient with HFrEF, NYHA II and systolic blood pressure lower than 90 mm Hg, which therapy would they have consider? The answer was low dose ACEIs and BB simultaneously in 45.8%, only BB in 26.6%, only ACEIs in 20.3%, no therapy in 6.8% and no idea in 0.5%. Sixty one (61%) physician agreed to implant CRT-D when CRT is indicated. In a patient with acute decompensated heart failure (ADHF), pulmonary congestion and 120 mm Hg SBP, iv. diuretic and vasodilatator therapy was the most preferred choice (58.8%). Most of the physicians (70.1%) did not prefer to give n-3 PUFA to their HF patients and. In a patient NYHA IV and QRS duration 118 msn, most of physicians (53.1%) did not consider ICD If the patient is not a candidate for LVAD or heart transplantation. While only 13.6% physicians had >30% patients with HFpEF, most of the physicians (30.5%) had between 21% and 30% patients with HFpEF. In a patient on BB and ivabradin therapy if the heart rate is decreased under 55 bpm, 55.9% of physician answer was to stop ivabradin therapy, 25.4% was cessation of ivabradin therapy, 4.5% was cessation of BB therapy, 10.7% was decreasing BB dosage. Only one quarter of participant center (27.7%) had heart failure policlinic.

Discussion

Heart failure (HF) is one of the most important causes of morbidity and mortality in the world.[1] It is a big burden to healthcare economy. Currently approximately 26 million adults live with HF in the world ant this number is substantially increasing with aging population. The prevalence is not clearly known in Turkey, but in the HAPPY study it was estimated as 2.9% in adults.[4]

In this questionnaire we aimed to evaluate awareness of physicians who are interested in HF and arrhythmias about current diagnostic and therapeutic strategies of HF. Most of the physicians (92,1%) was

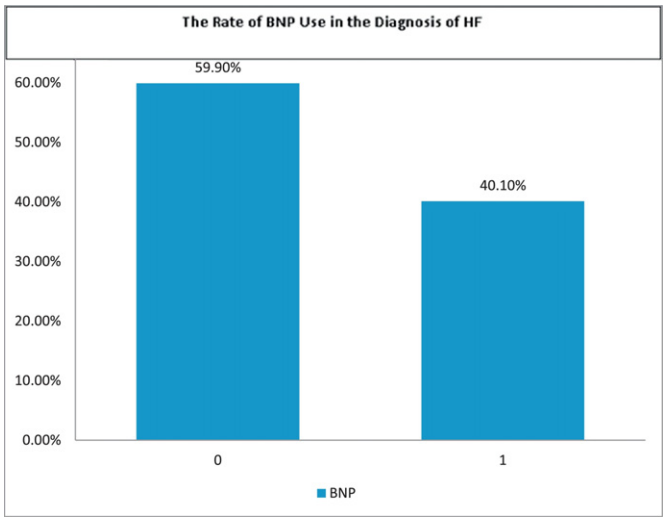


Fig. 1. The rate of BNP use in the diagnosis of HF.

working in an university hospital (3th grade health care center in Turkey) and there were much more academician (cardiologist, associated professor and professor). Natriuretic peptides (BNP and NT-proBNP) were more pronounced in the diagnosis of HF in recent European Society of Cardiology (ESC) HF guidelines (especially in the diagnosis of HFmrEF and HFpEF).[1] Less than half of the physicians (40.1%) stated to use natriuretic peptides in the diagnosis of HF (Fig. 1). 76.3% of physicians were aware of HF with mid-range EF (HFmrEF)—patients with HF and a left ventricular ejection fraction (LVEF) that ranges from 40 to 49% and agreed that it was necessary to classify HF patients as HFrEF, HFmrEF and HFpEF according to LVEF. Unfortunately only 5.6% of physicians were able to achieve the guidelines recommended target dose of angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) in more than 70% of patients with HF and reduced LVEF (HFrEF). Most of the physicians (69%) were only able to reach the treatment goals in the 41–50% of HFrEF patients. Same as ACEIs, the success rate of achievement guideline recommended BB therapy was disappointing. Only 5.1% of patients were able to achieve the treatment target >70% of HF patients. Despite optimal medical therapy

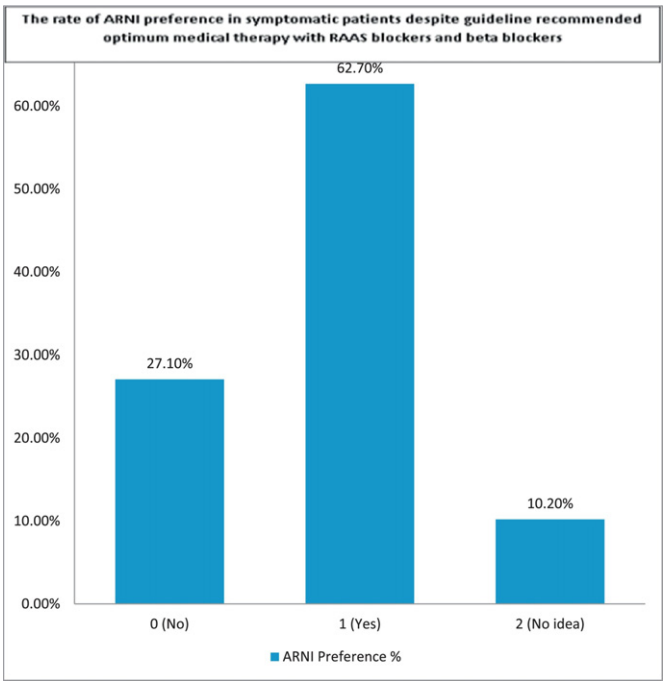


Fig. 3. The rate of ARNI preference in symptomatic patients despite guideline recommended optimum medical therapy with RAAS blockers and beta blockers.

with diuretic, ACEIs, ARBs, BB and mineralocorticoid receptor antagonists (MRAs), the ratio of physicians who have more than symptomatic patient population were 6.8% (Fig. 2). In a symptomatic patient despite optimal medical therapy with RAAS blocker and BB, most of the patients have chosen ivabradin (83.6%) or ARNI (62.7%). It shows physicians who is interested in heart failure, follows the suggestion of recent HF guidelines in daily practice. Interestingly, 10.2% of physicians have never heard before ARNI (Fig. 3, group 2). In a stable angina patient, revascularization was the most preferred treatment if the angina still continues despite optimal medical therapy. Carvedilol was the most preferred BB in HF patients same as daily practice. In an asymptomatic HF patient with LVEF 40%, most of the patients did not consider ICD

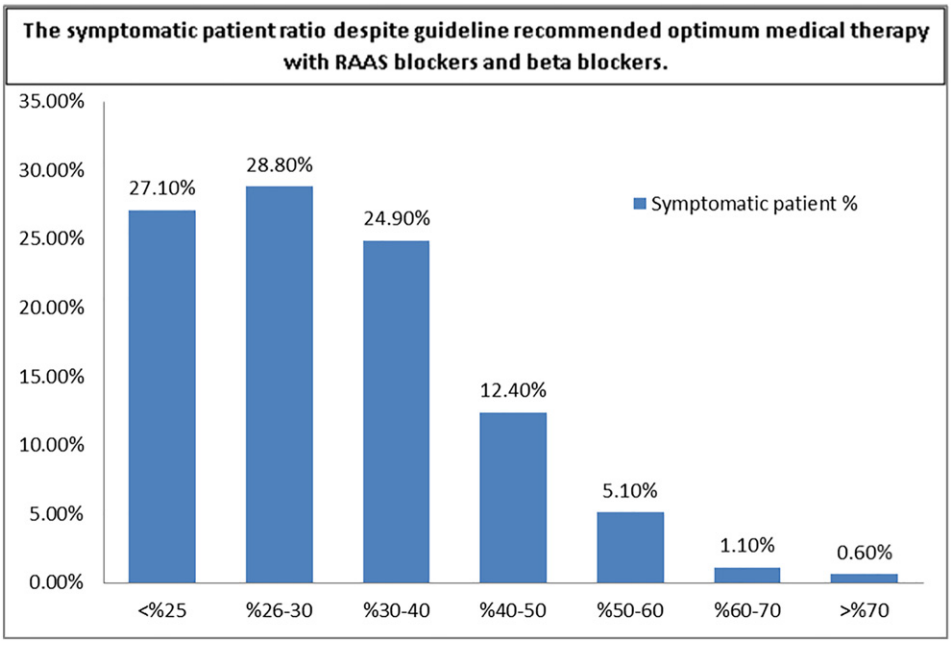


Fig. 2. The symptomatic patient rate despite guideline recommended optimum medical therapy with RAAS blockers and beta blockers.

therapy. LVAD and IABP were achievable in the most center. Most of the patients considered symptomatic improvement and euvolemia as a treatment target. Natriuretic peptides were the treatment target by only small amount of physicians (18.6%). Diuretic infusion was more preferred than iv. diuretic bolus in HF patients. This might be associated with the nature and stage of HF. If CRT is indicated most of the patients considered to implant CRT-D. Current ESC guideline on the management of HF recommends ACEIs or ARBs with BB at the same time for initiation therapy and to increase evidence-based maximum tolerated dose. The participant physicians have internalized this treatment strategy. N-3 PUFA treatment was not a highly preferred HF treatment. Current ESC guideline does not recommend ICD therapy to NYHA IV patients if they are not candidate for LVAD or heart transplantation. Most of the participants paid attention this recommendation. When we asked the proportion of HFpEF in their daily practice, 30.5% of participants emphasized that only small amount of heart failure patients in daily practice were HFpEF patients (13.6%). Only one quarter of participant center (27.7%) had heart failure policlinic.

In this questionnaire we wanted to see a picture from daily practice of physicians who are interested in heart failure. We aimed to measure awareness about current ESC heart failure guideline recommendations, new classification of HF according to LVEF and new diagnostic and therapeutic improvements.

Conflict of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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Case report

Management of iatrogenic brachial artery pseudoaneurysm☆,☆☆ Surgical treatment of iatrogenic brachial artery pseudoaneurysm

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ABSTRACT

Penetrating trauma, blunt trauma, percutaneous coronary artery intervention, drug abuse and arterial gas sampling may lead to pseudoaneurysms. Pulsatile mass, bruit, and adjacent nerve injury are the main clinical findings. Open surgery and percutaneous interventions and ultrasound-guided compression can be performed for the treatment. External compression should be applied to the brachial artery site for at least 10 min after the sheath was withdrawn for patients who underwent percutaneous coronary artery intervention. Here, we present a 55-year-old male who had an iatrogenic brachial artery pseudoaneurysm on his left upper arm.
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Introduction

Aneurysms can be either a true aneurysm or pseudoaneurysm. A true aneurysm is defined as the dilatation of all layers of the arterial wall however, pseudoaneurysm involves one or more layers and/or neointima, fibrous tissue, and thrombus. Penetrating/blunt trauma, catheterization, drug abuse and arterial gas sampling may lead to pseudoaneurysms. Pseudoaneurysms of the brachial artery usually occur as a result of either trauma (0.5%) or iatrogenic injury (3–7%) (1). Pseudoaneurysms may present weeks to years after trauma. Pulsatile mass, bruit, and adjacent nerve injury are the main clinical findings. Rupture of the pseudoaneurysm may result as infection, bleeding and distal arterial ischemia. Ultrasound, computed tomography angiography, magnetic resonance angiography or conventional arteriography can be used for diagnosing (2).

Case Report

A 55-year-old male was presented with significant swelling and pain on his left upper arm one month ago was admitted to our clinic. He had a history of coronary artery disease, hypertension, and percutaneous coronary intervention via his left arm, one month ago. On admission, his blood pressure was 125/85 mmHg and his heart rate was 84 beats/min. On physical examination both radial and ulnar pulses were palpable and a large pulsatile mass was identified on his left upper arm with a systolic bruit on auscultation (Fig. 1A). Motor function deficiency and paraesthesia were not detected. The rest of the physical examination was unremarkable. Ultrasound confirmed the presence of a 10 × 12 cm brachial artery pseudoaneurysm. We decided to perform open surgery due to the high volume of hematoma. Routine preparation for surgery was made and informed consent was taken. Under local anesthesia, a medial longitudinal incision was performed over the pseudoaneurysm. The proximal and distal brachial artery was identified. The hematoma was extracted. The puncture injury site was identified and sutured with 6/0 running prolene suture (Fig. 1A, B). The patient had an uncomplicated postoperative period and was discharged 2 days after the surgery. Six months later he was doing well with stable vital signs and pulses.

Discussion

The presentation of the pseudoaneurysm may often be delayed by weeks or years. Clinical findings are including neurological deficits, tenderness, pain and swelling, compartment syndrome, hemorrhage, hematoma, ischemia, nerve injury and venous edema which can be limb or life threatening (3). External compression should be applied to the brachial artery site for at least 10 min after the sheath was withdrawn for patients who underwent coronary artery intervention. In order to gain total hemostasis, further compression should be applied with an elastic tape. Females, patients with the peripheral vascular disease, compulsory anticoagulation, large sheaths have the higher risk for vascular complications. A pseudoaneurysm, thrombosis, dissections, median nerve injury are the major complications of the brachial artery puncture site. Early diagnosis and prompt treatment should be carried out. The treatment of pseudoaneurysm is based on its size, location,

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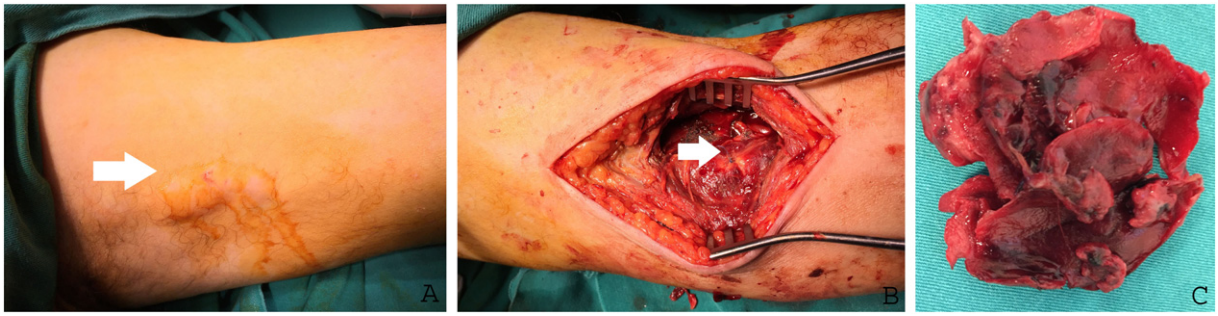


Fig. 1. External view of the pulsatile mass on the left upper arm (a), the intraoperative image of the pseudoaneurysm sac and the brachial artery injury site showed by the white arrow (b), extracted hematoma (c).

and etiology. Endovascular intervention is performed more common. The indications for open surgery consist of a rapidly expanding aneurysm, neurological deficits which are caused by local pressure and accompanying ischemia (4). Open surgical treatment includes graft interposition or primary suturing however, percutaneous interventions include bare stent or covered stent placement, coil embolization, ultrasound-guided thrombin injection and ultrasound-guided compression. Etiology, location, size and accessibility of the pseudoaneurysm should be considered for selecting the optimal treatment modality. Open surgery is preferred when there is rupture and infection (5).

In conclusion, careful and delicate puncture technique and adequate external compression to the brachial artery puncture site after the percutaneous coronary artery intervention play an essential role for cardiologists.

Conflicts of interest

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Short communication

The preference of the physicians in diagnosis and treatment of cardiovascular diseases

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ABSTRACT

Objective: To evaluate opinions and applications of physicians related to cardiovascular diseases in their daily practice about current diagnosis, treatment and prophylaxis of coronary artery disease, acute coronary syndrome and hypertension.
Method: In this study, a questionnaire survey was performed with cardiologists, internists, family physicians and neurologists about the current diagnosis, treatment and prophylaxis in cardiovascular diseases.
Results: A total of 208 physicians participate in the study. Most of the physicians were working in the specialty of cardiology (88.94%). Majority of physicians (47.6%) had a professional experience of more than ten years and 56.25% of them were working in the training and research hospitals or state hospitals.
Conclusion: The clinical applications of physicians are mostly in compliance with the treatment guidelines.
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Introduction

In recent years, new evidence has been obtained related to the diagnosis and treatment of cardiovascular diseases. As a result, many pharmacologic and nonpharmacologic treatment methods have been introduced into clinical practice.[1–6] Comprehensive improvements in diagnosis and treatment have taken place. In this questionnaire survey, evaluation of the opinions and applications of the physicians related to cardiovascular diseases in their daily practice about current methods of diagnosis, treatment and prophylaxis of cardiovascular diseases was aimed.

Methods

In this study, a questionnaire survey consisting of a total of 21 questions was performed with cardiologists, internists, family physicians, emergency medicine specialist and neurologists about current diagnosis, treatment and prophylaxis in cardiovascular diseases (Table 1). The physicians were invited to participate in the survey only via e-mails and questions were responded via e-mail. At the end of the questionnaire survey, the physicians were asked if their responses could be used for scientific processes and the responses of the assented physicians were analyzed. Approval of the local ethics committee for the study was obtained (2016/047).

Results

The questionnaire was completed by 214 physicians and 208 physicians allowed the data retrieved from the questionnaire to be analyzed within the frame of the study. The physicians participating in the study consisted of those working in the specialties of cardiology (n = 185; 88.9%), internal medicine (n = 9; 4.33%), emergency services (n = 7; 3.37%), family medicine (n = 62; 88%) and neurology (n = 1; 0.48%). The majority of the physicians (47.60%) had a professional life longer than ten years. The 56.25% of the physicians were working in training and research hospitals or state hospitals, 28.37% in universities, 12.5% in private hospitals and 2.88% in medical centers. (Table 2). The 66% prescribed ACE-inhibitors as their first choice in stage 1 hypertensive patients. More than half of the physicians had not used cardiovascular risk scoring system. The 73.5% of the attendees stated that outcomes

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Table 1
Questions (Q) of the questionnaire survey are seen.

Q1: Please select your area of specialization.
Q2: How many years have you been working as a physician?
Q3: Please indicate your academic status.
Q4: In which institution do you work?
Q5: Which the cardiovascular risk scoring systems do you use in your daily practice?
Q6: Do you require an 8-hour fasting condition while evaluating your patient's lipid panel?
Q7: Which patient population is your target in your daily practice of PCSK9 inhibitors?
Q8: Which of the following do you prefer as a primary treatment in a patient with AF who didn't undergo stenting after STEMI and has a CHA2DS2VASc score of 3?
Q9: Which of the following would you prefer as the first drug option in the pharmacological cardioversion for a patient with sudden AF, if the patient has hypertension, LVH and is hemodynamically stable?
Q10: When do you prefer Ticagrelor treatment instead of aspirin for the long-term treatment for secondary prevention?
Q11: In which order do you prefer the following combinations in the treatment of hypertension? (put them in order from 1 to 5)
Q12: At which rate do you need fixed-dose triple combination therapy in the hypertension treatment?
Q13: At which rate do you prefer Clopidogrel after ACS?
Q14: Do you think that too much statin is used in our country?
Q15: Which drug group do you prefer most in the treatment of stage 1 hypertension?
Q16: Who should evaluate CT angiography?
Q17: Which of the following patient profile(s) is/are not at high risk according to you?
Q18: What is your preference of the treatment for a case with STEMI diagnosis?
Q19: Did the results of the SPRINT study alter your target blood pressure value in high-risk group patients?
Q20: A 42-year-old asymptomatic patient who had an anterior MI one month ago and underwent primary PCI was referred to your polyclinic for routine control. You see that the medical treatment continues as suggested by guidelines, but the LVEF value is between 25–30%. What would be your ICD preference for this case?
Q21: For which patients do you prefer a bioresorbable stent?
Q22: Do you allow the answers given in the current survey to be used for scientific purposes?

of SPRINT study would not change target blood pressure value. Fixed-dose triple-combination was required >20% in clinical practice of 18.27% of the attendees. The 29% believed that statins are prescribed much more than needed. Most of the physicians participating in the study would prefer PCSK9 inhibitors mostly in cases with familial hypercholesterolemia. The 67% of participants performed PCI for seven days a week and 24 h a day or they transferred these patients to a center where PCI could be performed. However, the half of them still preferred clopidogrel following acute coronary syndrome (ACS). Moreover, 53% of participants did not use any cardiovascular risk-scoring tool.

Table 2
The table shows the institutions in which physicians participated in the survey are working.

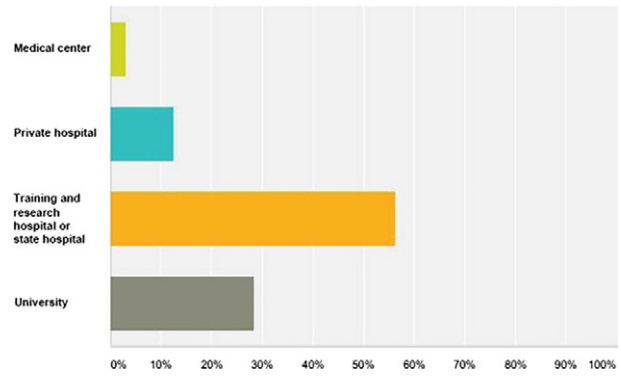
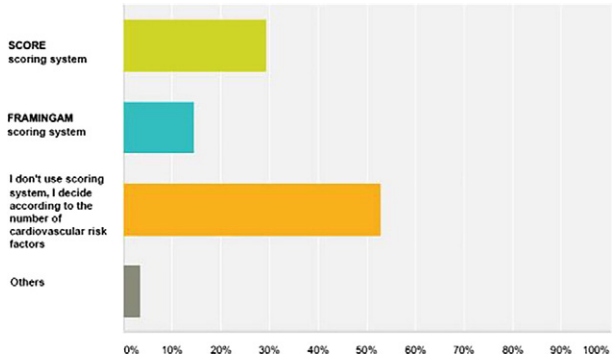


Table 3
The cardiovascular risk scoring systems preferred by the physicians in daily practice are seen.



Discussion

Cardiovascular diseases are among important causes of morbidity and mortality worldwide. Their prevalence gradually increases because of prolongation of expected life span, variations in life style and technologic developments in the field of medicine.[7–9] In recent years, despite raised awareness of cardiovascular diseases and steps taken during the fight against risk factors, these diseases are important causes of mortality both in developed and developing countries.[10,11]

Cardiovascular disease is still the main cause of cardiac death worldwide. Therefore, the identification of risky population and assessing the risk of cardiovascular disease is essential.[12,13] Numerous risk-scoring models like WHO model, SCORE, JBS-2, QRISK, and ASSIGN are available.[14,15] In our study, we have detected that more than half of the physicians had not used cardiovascular risk scoring system however the majority of users had a preferred SCORE risk scoring system (Table 3).

Atherosclerosis is the most frequent cause of cardiovascular diseases. Lipids play a role in the development of atherosclerosis. Treatment of hyperlipidemia is associated with decreased cardiovascular mortality.[16] Decreasing LDL-cholesterol levels gains priority in the cardiovascular prophylaxis.[17] In clinical application, lipid panel is evaluated after 8–12 h of fasting. However, most of the individuals eat three main meals and snacks every day and feel fullness all day long. This condition has created concerns in that fasting lipid panel would not reflect cardiovascular risk. However, when fasting and postprandial lipid panels were compared, the difference was detected to be clinically insignificant.[18] Hyperlipidemia Guideline of European Society of Cardiology indicated that lipid panel could be performed during both fasting and state of satiety. However, the guideline-recommended follow-up of the patients during fasting state in case of hypertriglyceridemia.[19] In the study, during evaluation of lipid panel by physicians, most of the physicians indicated that they stipulated for fasting condition. A small percentage

Table 4
Table shows the answers regarding whether physicians were looking for 8 h of fasting before investigating lipid profile.

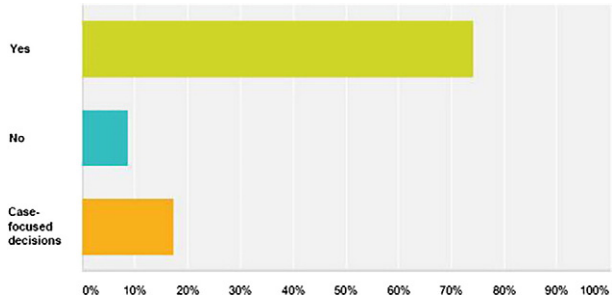
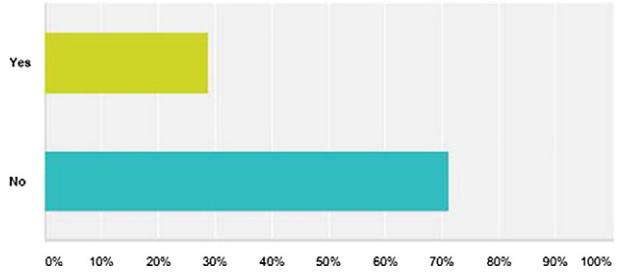


Table 5
The table shows the answers of the physicians participated in the survey regarding whether statins were overused in our country.



(17.3%) of the participants reported that they made case-focused decisions (Table 4).

Currently, statins are the most frequently used agents in the treatment of hyperlipidemia. They are recommended for primary and secondary prophylaxis against cardiovascular diseases.[20,21] However, under statin treatment target LDL-C level is achieved at a low level. Nearly 10–20% of the patients cannot use statin because of the development of statin tolerance. Whereas, ezetimibe and niacin decrease LDL-C only slightly and their effect on cardiovascular prophylaxis is debatable.[22] Statins are the very much-debated drugs among cardiovascular drugs in the media. Debates about the uselessness of these drugs or their many side effects are continuing. However, the great majority of our attendees believe that statins are not over-prescribed in Turkey (Table 5).

PCSK9 inhibitors in the treatment of hyperlipidemia are a novel and promising alternative. With these drugs, much more decrease in LDL-C levels has been achieved and cardiovascular events have also demonstrated a tendency to decrease.[23] In the light of available evidence PCSK9 inhibitors have found an indication of use in patients with high risk who cannot attain target LDL-C levels under statin therapy and in patients with familial hyperlipidemia.[19] Most of the physicians participating in the study stated that they would prefer this drug and use mostly in cases with familial hypercholesterolemia while 19% of them expressed that they wouldn't feel a need to use this new group agent at all (Table 6).

Hypertension is an important public health problem prevalent in the community and causes life-threatening complications. The objective of antihypertensive treatment is to decrease blood pressure, cardiovascular mortality and morbidity.[24] Decreasing blood pressure is known to decrease unwanted events. However, the extent of this drop has been a matter of debate.

In ACCORD study performed on diabetic patients with increased cardiovascular risk, the incidence of unwanted events did not differ between patients who were under strict blood pressure (<120 mm Hg) control and patients in whom normal blood pressure levels (<140 mm Hg)

Table 6
The answers of the physicians regarding the place of PCSK9 inhibitors in daily practice are seen.

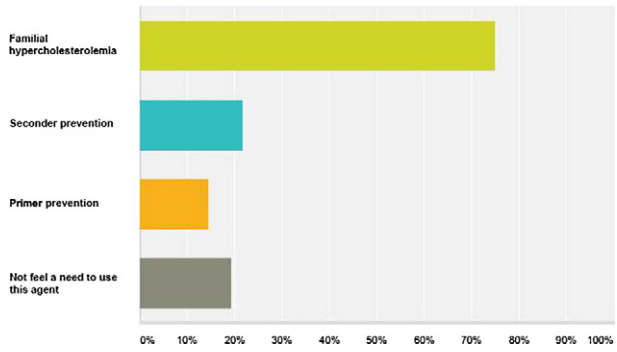
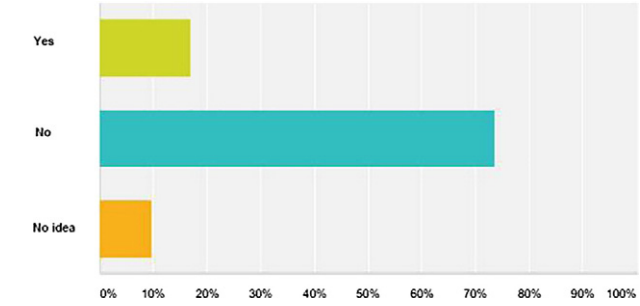


Table 7
The table shows the answers concerning whether the results of the "SPRINT" study have changed the target blood pressure levels in high-risk patients.



were achieved.[25] Still as demonstrated in SPRINT study where non-diabetic patients with an increased cardiovascular risk were enrolled, selection of target blood pressure as 120 mm Hg rather than 140 mm Hg, decreased fatally and nonfatal cardiovascular events.[26] Possibly, with SPRINT study, the necessity to determine target blood pressures again in patients with increased cardiovascular risk will arise. However, in our questionnaire survey, most of the physicians (73.5%) stated that outcomes of SPRINT study would not change target blood pressure value (Table 7).

Blood pressure control in the treatment of hypertension is mostly achieved by combination treatment rather than monotherapy. In a meta-analysis, it has been determined that combination of two different drugs at lower concentrations induced a more dramatic decrease in blood pressure when compared with the maximum dose of a single drug.[27] When we asked the physicians about their selection of antihypertensive combination, most frequently angiotensin-converting enzyme inhibitor (ACE-I) plus diuretic combination was preferred, followed by (in order of decreasing frequency) ACE-I + calcium canal blocker, angiotensin II receptor blocker (ARB) + diuretic, and ARB + calcium canal blocker and β-blocker + diuretic (Table 8). Fixed triple dose combination was preferred by <10% of the physicians.

As is known, beta-blocker, diuretic, calcium canal blockers and renin-angiotensin system inhibitors decrease blood pressure and unwanted cardiovascular events. Nowadays, most frequently RAS blockers are used for the treatment of hypertension. The two most frequently preferred members of this group are ACE-I, and ARBs.[28] In our questionnaire, survey physicians reported their first preference for the treatment of stage I hypertension as ACE-I (Table 9).

Left ventricular hypertrophy (LVH) is one of the important target organ injuries caused by hypertension. LVH is a strong independent risk factor for arrhythmias as atrial fibrillation (AF).[29] Achievement of sinus rhythm leads to symptomatic improvement and increase in quality of life. The selection of the drug to be used for pharmacologic cardioversion is based on underlying pathology. In hypertensive heart disease, the first drug to be selected is dronedarone followed by amiodarone.[30] In our questionnaire survey, the first preference was amiodarone for

Table 8
The preferences of the physicians on antihypertensive drugs are seen.

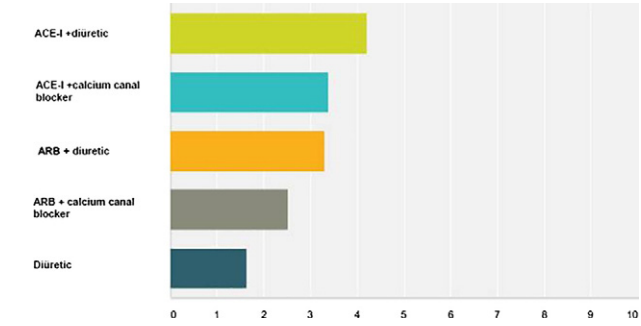
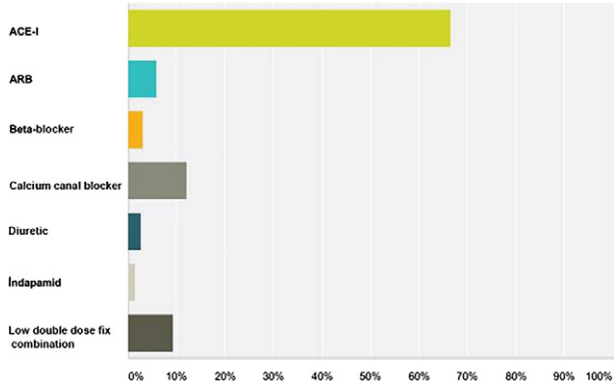


Table 9
The preferences of the physicians on drugs in Stage 1 hypertension.



pharmacologic cardioversion in hypertensive LVH patients with the stable hemodynamic state who experienced sudden AF attacks, which was consistent with guideline recommendation (Table 10).

The main component of the treatment for ST-elevation myocardial infarction (STEMI) is the achievement of coronary reperfusion. Coronary reperfusion can be achieved with thrombolytic treatment or percutaneous coronary intervention (PCI). If it can be performed by an experienced team within the first 120 min after the first medical contact, PCI is more advantageous than thrombolytic treatment.[31] Most of the study participants stated that they could perform PCI for seven days a week and 24 h a day or they transferred these patients to a center where PCI could be performed. Only a relatively low percentage of physicians used thrombolytic treatment.

As the part of new developments in stent technology, absorbable stents have been introduced into clinical use. Nearly one-third of the physicians reported that they would prefer to use the stent for diabetic patients with middle-long lesions of LAD, while other one-third of them indicated that they would never advise them.

The Cardiac computed tomography angiography (CCTA) is most commonly performed by radiologist however; the 81% of attendees were against the evaluation of CCTA by only radiologists.

Following an episode of ACS long-term use of dual anti-aggregant is required. In addition, the use of ticagrelor as an adjunct to aspirin is known to decrease major unwanted hemorrhagic effects and mortality rates when compared with the aspirin-clopidogrel combination.[32] Therefore, independent from the initial therapy, it is based on recommendations that all patients under moderate-high risk of ischemic event including those previously treated with clopidogrel should receive ticagrelor and if the patients could not use ticagrelor, then

Table 10
The preferences of the physicians in the pharmacological cardioversion of a hemodynamically stable, hypertensive patient with left ventricular hypertrophy for recently started atrial fibrillation are seen.

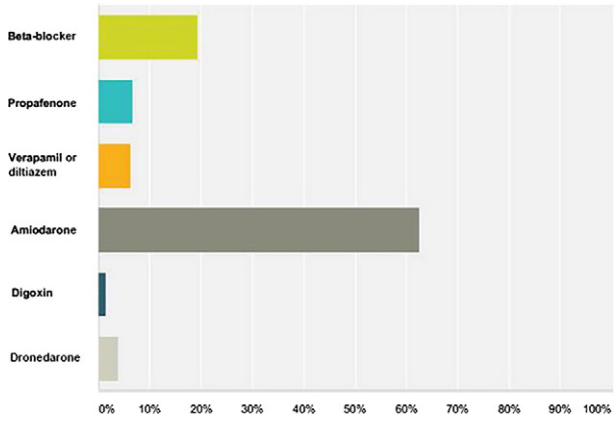
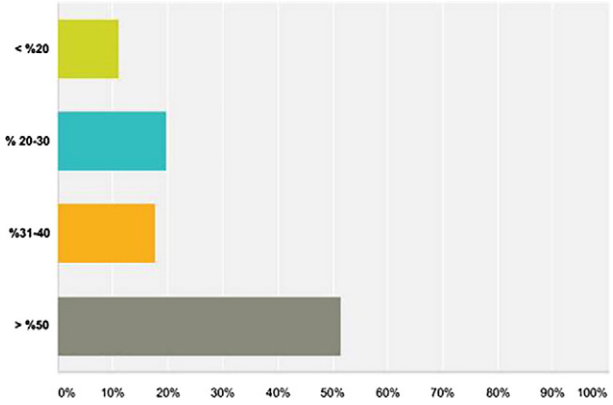


Table 11
Clopidogrel preference rates of the physicians after acute coronary syndrome are seen.



clopidogrel should be used.[33] However, we detected very high rates of preference for clopidogrel in our study despite the recommendations of the guideline (Table 11).

Aspirin + ticagrelor therapy exerts favorable effects on clinical events within one year after an episode of ACS. However usefulness of maintaining this treatment for longer periods has been a debatable issue. In a study performed on this subject a marked decrease in the risk of cardiovascular death, myocardial infarction and stroke have been detected.[34] In a questionnaire survey, the physicians were questioned about the preference of ticagrelor for secondary prevention. It has been determined that very scarce number of physicians preferred ticagrelor and most of them inclined to use aspirin (Table 12).

Because of AF, dual antiplatelet use in patients under oral anticoagulant therapy may create problems. In patients who had undergone stenting procedure triple therapy (aspirin + clopidogrel + anticoagulant therapy) should be used. While for patients scheduled for medical or surgical treatment, alternative dual (oral anticoagulant and aspirin or clopidogrel) therapy is recommended.[35] When physicians were questioned about their preference for the treatment of patients with a CHA2DS2VASc score of 3 who did not undergo stenting procedure after an episode of STEMI, 53% of them preferred to use anticoagulant + clopidogrel therapy. This preference complied with the recommendations stated in guidelines.

Patients experiencing acute myocardial infarction (MI) should be evaluated before their discharge from the hospital and 6–12 weeks later with echocardiographic tests. Post-MI ejection fraction <40% increases the risk of sudden cardiac death. In symptomatic MI patients whose ejection fraction dropped dramatically (NYHA Class 2–3), a waiting period of at least 40 days is recommended for primary prophylaxis for the implantation of intracardiac defibrillator in selected cases. (incomplete revascularization, history of lower EF, development of polymorphic ventricular tachycardia or ventricular fibrillation 48 h following myocardial infarction) Implantation of ICD may be considered within the first 40 days. Due to lack of randomized controlled studies

Table 12
The preference rates of the physicians of ticagrelor rather than aspirin for long-term secondary protection are seen.

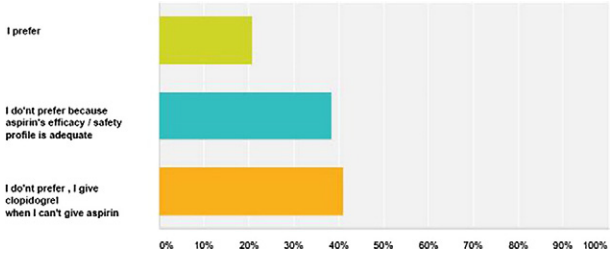
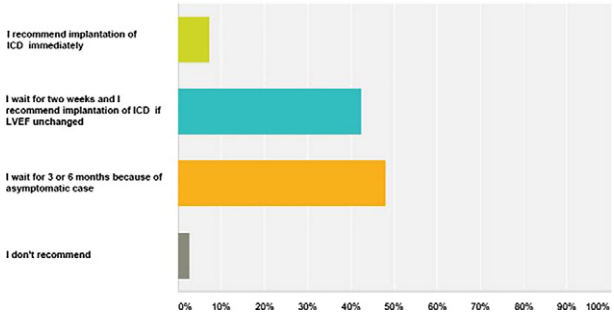


Table 13
A 42-year-old asymptomatic patient who underwent primary PCI for an anterior myocardial infarction one month ago admitted to our polyclinic for routine control examination. Although the patient was treated according to the guidelines, LVEF was found to be 25–30%. The physicians were asked for ICD treatment in this patient.



in asymptomatic cases with low EF, implantation of ICD is not recommended.[36] In our questionnaire when the physicians were asked about their preference of ICD for an asymptomatic patient with an LVEF of 25–30% who experienced an episode of MI one month previously, most of the physicians preferred to make a decision after a waiting period of 3 or 6 months, 42% of them recommended ICD implantation after waiting period of 2 weeks in case of persistence of LVEF. Relatively small number of physicians did not recommend implantation of ICD at all (Table 13).

Conclusion

This questionnaire survey aimed to determine daily practical approaches of the physicians. As a result of the study, apparently clinical applications and preferences of the physicians in the diagnosis and treatment of cardiovascular diseases comply with most of the recommendations in treatment guideline of European Society of Cardiology.

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Short communication

Monocyte-to-high density lipoprotein ratio (MHR) can predict the significance of angiographically intermediate coronary lesions☆

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Plateletcrit

ABSTRACT

Purpose: Monocyte-high density lipoprotein ratio (MHR) has recently emerged as a marker of inflammation and oxidative stress in the cardiovascular disease. We aimed to investigate whether baseline MHR is associated with functional significance of intermediate coronary artery lesions.
Methods: Three hundred and one consecutive patients, 215 males and 86 females, who underwent fractional flow reserve (FFR) measurement for angiographically intermediate coronary stenosis (40–70% in quantitative coronary analysis) in the left anterior descending coronary artery were enrolled into the study. An FFR value of ≤0.80 was accepted for hemodynamic significance.
Results: Of the 301 patients, 115 (38.2%) exhibited significant functional stenosis (FFR ≤ 0.80) in the FFR measurement. Patients with hemodynamically significant lesions had higher MHR values (11.6 ± 3.3 vs. 12.6 ± 2.5 p = 0.003).In stepwise multivariate logistic regression analysis, total cholesterol (OR = 1.008, 95% CI = 1.002–1.013, p < 0.010), plateletcrit (OR = 1.310, 95% CI = 1.097–1.564, p = 0.013) and MHR (OR = 2.993, 95% CI = 1.365–6.561, p = 0.008) were independent predictors of significant functional stenosis. An MHR value of 12.1 had 65% sensitivity and 55% specificity for prediction of hemodynamically significant coronary artery stenosis.
Conclusions: Increased MHR values were associated with functional significance of angiographically intermediate coronary artery stenosis.
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Introduction

Coronary angiography is one of the principal methods used for diagnosing and assessing coronary artery lesions.[1] However, qualitative evaluation of coronary artery stenosis with coronary angiography is not always reliable and the visible anatomic stenosis may not be hemodynamically significant. FFR measurement is a well-established method for functional assessment of lesion severity, which involves the measurement of a coronary artery lesion's hemodynamic significance when the coronary artery stenosis is particularly between 40 and 70% (i.e. at an intermediate level).[2]

Inflammation, oxidative stress, platelet activation and endothelial dysfunction assume an important role in both the development and progression of atherosclerosis.[3,4] Monocytes and macrophages are the main types of cells that secrete proinflammatory cytokines, which play a central role in in the pathogenesis of atherosclerosis.[4] Studies have shown that the adverse effects of low-density lipoproteins (LDLs) on endothelial cells can be limited by high-density lipoprotein cholesterol (HDL-C), which prevents the oxidation of LDL.[5–7] HDL-C is believed to have both antioxidant and anti-inflammatory properties. A new cardiovascular disease marker that has appeared in recent times is the monocyte to HDL-C ratio (MHR), which combines the prognostic and predictive effectiveness of two widely used and accessible laboratory parameters.[8–10] The aim of the present study was to examine the relationship between MHR and the functional significance of intermediate level coronary artery stenosis evaluated with FFR measurement.

Subjects and methods

This retrospective study was conducted between January 2012 and March 2016 with a total of 301 consecutive patients, including 86 female and 215 male patients, diagnosed with single intermediate-grade coronary stenosis (40–70%, based on quantitative coronary analysis) on their left anterior descending coronary artery who were examined with fractional flow reverse (FFR) measurement. Patients who had

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undergone coronary angiography with an indication of stable angina pectoris involved in the present study. Patients with the following conditions were excluded from the study: acute coronary syndrome; moderate or severe valvular heart disease; significant arrhythmia; hemodynamic instability; second lesion at the index coronary artery; another coronary artery with a ≥40% luminal narrowing (determined by coronary angiography); a history of previous surgical or percutaneous coronary artery intervention; acute or chronic inflammatory or infectious diseases; anemia, chronic renal failure, and malignancy.

Hospital files and records were examined to determine the patients' demographic, clinical and angiographic data. Blood samples were collected from veins within 24 h after hospital admission. Measurements included lipid profile, serum creatinine and complete blood count. The patients' baseline MHR was determined by dividing their monocyte count with their HDL cholesterol level. The institutional local ethics committee approved the study protocol.

FFR measurements for intermediate-grade lesions with a 40–70% stenosis rate were performed based on the cardiologists' decision and discretion. Following the intra-arterial administration of a 5000 unit heparin bolus, the coronary artery was examined by employing a guiding catheter without side holes. A 0.014 in. pressure monitoring guidewire (PrimeWire, Volcano, San Diego, Calif., USA) was positioned distally to the stenosis after calibration. A 200 μg bolus of nitroglycerin was administered intracoronarily prior to FFR measurements. The patients' distal intracoronary pressure was recorded at baseline, and hyperemia was triggered by applying intracoronary adenosine at gradually increasing doses until the FFR value ceased to decrease any further. FFR was determined as the ratio between the mean distal intracoronary pressure and the mean aortic pressure, at the moment the highest level of hyperemia is observed.

An FFR value of ≤0.80 was defined as functionally significant. Patients with an FFR value of >0.80 formed group I and patients with an FFR value of ≤0.80 formed group II.

Statistical analysis

Statistical analysis was performed using the SPSS 18.0 for Windows (SPSS Inc., Chicago, IL, USA) package software. Whether the data exhibited normal distribution was determined using the Kolmogorov–Smirnov test. The chi-square test was used for comparing categorical variables, while the Mann–Whitney *U* test or Student's *t*-test were used for comparing continuous variables. Percentages were used for presenting categorical variables, while mean ± standard deviation or median (interquartile range) were used for presenting continuous variables. Possible confounding factors for the coronary artery lesions' severity were determined using the univariate and multiple logistic regression analysis. The multiple regression model was used to test variables in the univariate regression analysis that has p values below 0.10. To determine optimum MHR cut-off value for predicting the hemodynamic significance of the coronary artery stenosis, the receiver-operating characteristic (ROC) curve was employed. Statistical significance level was set at <0.05.

Results

The baseline characteristics of the patients are shown in Table 1. Among the 301 patients included into the study, 115 (38.2%) were found to have significant functional stenosis. Group II has a lower mean age than group I (58 ± 12 vs. 61 ± 11 years, p = 0.007). While group II had a higher ratio of male patients, the difference between the two groups was not significant (68% vs. 77%, p = 0.072). There were also no statistically significant differences with regards to the coronary risk factors, including smoking, hyperlipidemia, hypertension and diabetes mellitus (Table 1).

The laboratory parameters of two groups are reported in Table 2. Group II had higher level of total cholesterol (203 ± 46 vs. 188 ± 42 mg/dL, p = 0.004). LDL-C level was also higher in group II, although the difference was not statistically significant (125 ± 39 vs. 117 ± 38 mg/dL, p = 0.078). Group II exhibited higher monocyte count (50 (40–56) vs. 55 (45–58), p = 0.004), plateletcrit (0.21 ± 0.01 vs. 0.20 ± 0.01, p = 0.002) and MHR (12.67 ± 2.59 vs. 11.65 ± 3.33, p = 0.003). Group I had a higher lymphocyte-to-monocyte ratio (LMR), although this difference was not statistically significant (0.044 (0.034–0.057) vs. 0.042 (0.030–0.054), p = 0.227). Other laboratory data did not differ significantly between the two groups (Table 2).

Univariate and multiple logistic regression analysis indicated that MHR (OR = 3.401, 95% CI = 1.378–8.391, p = 0.008), plateletcrit (OR = 1.276, 95% CI = 1.052–1.549, p = 0.013) and total cholesterol (OR = 1.025, 95% CI = 1.006–1.044, p = 0.010) are independent predictors of significant functional stenosis (Table 3). ROC analysis revealed that an MHR value of 12.1 had a specificity of 55% and a sensitivity of 65% in predicting hemodynamically significant coronary artery stenosis (Fig. 1).

Table 1
Basal characteristics of the study groups.

Variable	Insignificant FFR (n = 186)	Significant FFR (n = 115)	p value
Age, year	61 ± 11	58 ± 12	**0.007**
Basal FFR	0.93 ± 0.03	0.86 ± 0.08	<**0.001**
Smoking, n (%)	91 (49)	55 (48)	0.853
Male gender, n (%)	126 (68)	89 (77)	0.072
Diabetes mellitus, n, %	56 (30)	30 (26)	0.453
Hypertension, n, %	69 (37)	42 (37)	0.920
Hyperlipidemia, n, %	51 (28)	24 (21)	0.198

FFR - fractional flow reserve. Data are expressed as mean ± standard deviation for normally distributed parametric variables, median (interquartile range) for non-parametric variables and percentage for categorical variables.

Discussion

In this study, MHR levels were independently associated with functionally significant coronary artery lesions assessed with FFR

Table 2
Comparison of laboratory parameters between patients with hemodynamically significant coronary artery lesions and patients with hemodynamically non-significant coronary artery lesions.

Variable	Insignificant FFR (n = 186)	Significant FFR (n = 115)	p value
Glucose, mg/dL	110 (94–153)	112 (94–147)	0.838
Urea, mg/dL	34 (28–39)	34 (27–40)	0.951
Creatinine, mg/dL	0.91 (0.79–1.04)	0.93 (0.80–1.03)	0.678
Total cholesterol, mg/dL	188 ± 42	203 ± 46	**0.004**
Trygliceride, mg/dL	146 (107–195)	154 (112–242)	0.122
HDL-C, mg/dL	42 (35–50)	41 (37–45)	0.495
LDL-C, mg/dL	117 ± 38	125 ± 39	0.078
WBC count, $\times 10^9$/L	8.5 ± 2.7	8.9 ± 2.3	0.233
Neutrophile count, $\times 10^9$/L	5.46 ± 2.41	5.81 ± 2.03	0.199
Lymphocyte count, $\times 10^9$/L	2.22 ± 0.84	2.25 ± 0.83	0.709
Monocyte count, $\times 10^{12}$/L	50 (40–56)	55 (45–58)	**0.004**
Eosinophile count, $\times 10^{12}$/L	23 (11–38)	30 (20–40)	0.054
Hb, gr/dL	14.2 (13.0–15.4)	14.5 (13.3–15.4)	0.241
RDW	13.7 (13.0–14.6)	13.5 (13.1–14.3)	0.212
Platelet count, $\times 10^9$/L	254 ± 62	247 ± 65	0.369
MPV, fL	8.4 ± 0.6	8.4 ± 1.4	0.641
Platecrit (PCT)	0.20 ± 0.01	0.21 ± 0.01	**0.002**
PDW	16.9 (16.3–18.0)	16.8 (16.2–17.5)	0.338
NLR	2.39 (1.75–3.19)	2.56 (1.74–3.54)	0.196
PLR	121 (93–151)	117 (91–155)	0.369
LMR	0.044 (0.034–0.057)	0.042 (0.030–0.050)	0.227
MHR	11.65 ± 3.33	12.67 ± 2.59	**0.003**

FFR - fractional flow reserve; HDL - high-density lipoprotein; LDL - low-density lipoprotein; MPV - mean platelet volume; NLR - neutrophil-to-lymphocyte ratio; PLR - platelet-to-lymphocyte ratio; RDW - red cell distribution width; WBC - white blood cell; LMR - lymphocyte-to-monocyte ratio; MHR - monocyte count -to -HDL-C ratio. Data are expressed as mean ± standard deviation for normally distributed parametric variables and median (interquartile range) for non-parametric variables.
Bold values indicate significance at p < 0.05.

Table 3
Univariate and multiple logistic regression analyses performed to find out possible confounding factors of hemodynamically significant coronary artery stenosis.

Variable	Univariate analysis			Multivariate analysis		
	Odds ratio	95% CI	p value	Odds ratio	95% CI	p value
T chol	1.008	1.002–1.013	0.005	1.025	1.006–1.044	**0.010**
Age	0.971	0.950–0.992	0.008	0.984	0.961–1.008	0.193
LDL-C	1.006	0.999–1.012	0.079	0.984	0.967–1.000	0.053
PCT	1.310	1.097–1.564	0.003	1.276	1.052–1.549	**0.013**
MHR	2.993	1.365–6.561	0.006	3.401	1.378–8.391	**0.008**
Male gender	1.603	0.939–2.738	0.084	1.436	0.788–2.615	0.237

T chol - total cholesterol; LDL-C - low-density lipoprotein cholesterol; PCT -plateletcrit; MHR - monocyte count -to -HDL-C ratio.
Bold values indicate significance at $p < 0.05$.

measurement. In addition, total cholesterol and plateletcrit remained independent predictors of functional significance on multivariate analysis in the current study.

Oxidative stress and inflammation are central mechanisms in the development and progress of atherosclerosis.[3,4,9] Monocytes play a particularly important role in these processes.[4,5] The interaction of activated monocytes with activated or damaged endothelium leads to the overexpression of adhesion molecules and proinflammatory cytokines, such as the intercellular adhesion molecule-1, the vascular cell adhesion molecule-1, and the monocyte chemotactic protein-1 ligand.[4,11,12] Monocytes then differentiate into the macrophage cells, which absorb oxidized LDL cholesterol molecules, forming harmful foamy cells in the process.[12] On the contrary, HDL-cholesterol molecules reduce macrophage accumulation and promote removal of oxidized cholesterol from arterial wall.[13–15] Recent studies also showed that HDL-cholesterol can inhibit monocyte activation, adhesiveness, and inflammation.[5–7] In addition to its antioxidative and antiinflammatory effects, HDL also increases the expression of nitric oxide synthase in endothelial tissues and promotes vasorelaxation.[16] Higher HDL levels are known to provide protection against atherosclerosis, and are associated with better prognosis among patients with atherosclerosis.[17] Therefore, HDL-C counteracts the predominantly prooxidant and proinflammatory effects of monocytes.

MHR is a new and promising marker that indicates the inflammatory state of the patient. Based on the current literature, we assumed that higher MHR values might serve as an effective predictor of functional significance of coronary artery stenosis.

Qualitative evaluation of coronary artery stenosis with coronary angiography is not always reliable and the visible anatomic stenosis may not be hemodynamically significant.[2,18] FFR measurement is a well-established method for functional assessment of lesion severity. FFR technique is based on pressure decline between proximal and distal parts of the stenosis after obtaining maximal hyperemia and linearly related to maximum blood flow to the myocardium supplied by the respective coronary artery. Thus, in this study FFR measurement was utilized to determine the functional significance of coronary lesion severity and its relationship with the MHR level.

MHR is a newly discovered marker for oxidative stress and inflammation. Kanbay et al. previously suggested that in patients with chronic kidney disease, high MHR values are linked with worse cardiovascular prognosis.[8] Another study reported that high MHR values are correlated with a greater severity and occurrence of isolated coronary artery ectasia.[10] Kundi et al. demonstrated, based on SYNTAX score assessments, a relationship between higher MHR and greater coronary atherosclerosis severity.[19] Cetin et al. demonstrated that in patients with acute coronary syndrome MHR was an independent predictor of stent thrombosis and severity of coronary artery disease as well as future cardiovascular events.[20,21] Also in a study of Cicek et al., MHR was found to be significantly associated with both short- and long term mortality in patients with ST elevation myocardial infarction who underwent primary percutaneous coronary intervention.[22]

In our study, we found that MHR is significantly higher in patients with hemodynamically significant lesions assessed by FFR (FFR ≤ 0.80). There are some possible explanations of our findings. First of all, it was clearly shown that patients with hemodynamically significant coronary lesions have a poor prognosis and increased MHR has been closely linked with worse outcomes.[8,20–22] Secondly, higher MHR can

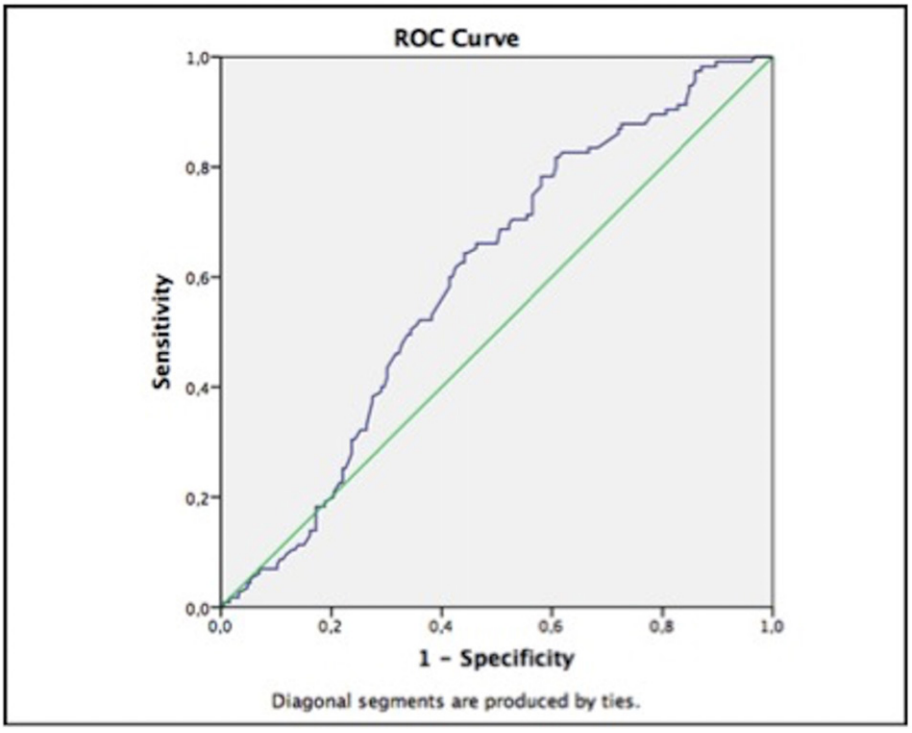


Fig. 1. The receiver-operating characteristic curve analysis for MHR in predicting functionally significant coronary stenosis in FFR measurements. MHR (monocyte count -to -HDL-C ratio) value of 12.1 had 65% sensitivity and 55% specificity for prediction of hemodynamically significant coronary artery stenosis. AUC: 0.598, 95% CI = 0.534–0.662, p = 0.004.

be viewed as a reaction to coronary artery lesions of greater severity. Based on this view, we consider that ischemias can lead to various inflammatory responses that alter WBC distribution and count. There is evidence suggesting that higher monocyte levels are linked with the development of proinflammatory and atherosclerotic responses.[3,4,9,11–12] It is known that HDL-C molecules can inhibit the activation and movement of monocytes.[13–15] The observation that coronary artery disease patients with high SYNTAX levels (≥23) tend to have with increased MHR values is in line with the present study's findings that increased MHR may serve as a predictor of functional significance in patient with intermediate-grade coronary artery stenoses.[19,23] In addition, previous studies indicate that severe CAD with good collaterals tend to have high monocyte counts, while those which poorer collaterals have lower HDL levels. This finding is also in agreement with the results of our study.[24–26] Considering that the degree and duration of coronary stenosis is the one of the most important determinants of coronary collateral development, it can be said that MHR values can help predict significant coronary stenosis that leads to myocardial ischemia.

In contrast to other studies, we determined during the multivariate analysis that plateletcrit and total cholesterol are both independent predictors for significant functional stenosis. These findings are in line with the known pathogenesis of coronary atherosclerosis. But there is insignificant correlation between functional stenosis, MHR and LDL-Cholesterol. Previous similar studies showed that there was not statistically significant difference between lipid biomarkers and functional coronary stenosis severity in stable coronary artery disease.[27–29] The difference in results between our work and other studies, can be explained by the he effects of antihyperlipidemic treatment; and genetic, environmental (including obesity, physical inactivity, and cigarette smoking), and unmeasured variables.

Studies have demonstrated that the platelet-to-lymphocyte ratio (PLR) and the neutrophil-to-lymphocyte ratio (NLR) are both significant markers of inflammation, and that they are linked with a variety of cardiovascular diseases.[30–32] In two studies similar to our own on the FFR, red cell distribution width (RDW) and NLR showed close relationship with the functional significance of intermediate-grade coronary artery stenoses.[28–29] However, in our study, it was observed that PLR, NLR and RDW did not function as independent predictors hemodynamically severe coronary artery stenosis. Similarly, Sels et al.'s study failed to demonstrate any relationship of FFR with inflammatory markers – such as the tumor necrosis factor, interleukin-6, interleukin-8 and WBC count.[33] These different results might be explained with differences in patient characteristics and numbers in different studies. Also, these differences might also be accounted by the different approaches of researchers towards coronary stenotic lesions (e.g. percutaneous, medical and sintigraphy approaches).

The evaluation of platelet indices such as platelet counts, mean platelet volume (MPV), platelet distribution width (PDW) and plateletcrit (PCT) is easy and practical and can play a role in the onset and progression of atherosclerosis.[34] İn our study, only higher PCT is independent predictors for significant functional stenosis but there is weak correlation. There are few studies investigating the relationship between PCT and coronary artery disease in the literature. Previous studies suggested that high PCT values on admission are independently associated with long-term adverse outcomes in patients with ST-segment elevation myocardial infarction (STEMI), non STEMI (NSTEMI), coronary slow flow phenomenon and saphenous vein grafting disease.[35–38] In the light of these findings PCT may show correlation with significant functional stenosis of angiographically intermediate coronary lesions, but this may be a coincidence arising from the coexistence of atherosclerosis severity and not a causal relationship.

Study limitations

The present study had a number of limitations. First of all, the study has a retrospective design with limited number of patients. Secondly, many other important markers of inflammation – such as the hs-CRP – were not used in this study (although it is certainly unfeasible to conduct a study that can include and examine all types of inflammatory markers). Thirdly, our study analyses were based on a single MHR values; in other words, we did not follow temporal changes and variations in MHR. Finally our study does not provide a mechanistic explanation for the effect of specific monocyte subsets on the severity of CAD and MHR.

Conclusion

The present study demonstrated that in FFR assessments, MHR values exhibit an independent relationship with the functional significance of angiographically intermediate coronary artery stenosis. As they are based on commonly used and low cost parameter found in lipid panels and complete blood counts, MHR values can be readily determined to predict the likelihood of hemodynamically significant coronary artery stenosis in clinical settings. Nevertheless, the results of this study should be further expanded and confirmed through studies involving larger samples and prospective designs.

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Case report

An interesting case of a self-apposing stent implantation in an aneurysmatically dilated artery in acute myocardial infarction with high quality optical coherence tomography images☆

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ABSTRACT

56-Year-old man with non-ST-segment elevation myocardial infarction. Coronary angiography showed aneurysmatic changes of the left circumflex artery with near occlusion of this vessel. We have decided to implant a Self-Apposing® Coronary Stent Xposition S (Stentys SA, Paris, France). In optical coherence tomography a good stent apposition has been confirmed. A complete distal flow in the infarct-related artery was achieved. Implantation of DES in a large vessel, especially with aneurysmatic dilatation is limited due to difficulties in choosing a proper stent size. Undersizing may cause stent malapposition which carries an increased risk of late and very late stent thrombosis. Oversizing may lead to dissection or even vessel perforation. In presented case choosing a proper stent size was not so easy because of aneurysmatic changes of the artery. The vessel diameter in the aneurysm area was about 6 mm. Regular DES are commercially available in maximal sizes up to 4.5–5.0 mm. In this case we have chosen the largest Stentys stent size available (3.5–4.5 × 27 mm) which is designed to target vessels of diameter between 3.5 and 6.0 mm. Self-apposing stents present useful features which might have advantages over conventional drug-eluting stents in specific angiographic situations.
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Introduction

Implantation of a new-generation drug-eluting stent (DES) is a preferred method of reperfusion in patients with acute myocardial infarction (MI) in most angiographic situations.[1,2] However, these devices also have some limitations. They are commercially available in maximal sizes up to 4.5–5.0 mm. Implantation of DES in a large vessel, especially with aneurysmatic dilatation is limited due to difficulties in choosing a proper stent size. Undersizing may cause stent malapposition which carries an increased risk of late and very late stent thrombosis. On the other hand, oversizing may lead to dissection or even vessel perforation.[1,3]

Case report

A 56-year-old Caucasian man was transferred to our department because of non-ST-segment elevation MI. On admission the patient was clinically stable. The ECG showed sinus rhythm, 50 beats per minute, negative T waves in III, aVF. Transthoracic echocardiography revealed hypokinesis of the middle and apical segment of the lateral wall with left ventricular ejection fraction of 60%. The patient was treated with a loading dose of acetylsalicylic acid and unfractionated heparin and was transferred to the cathlab for a coronary angiogram. Coronary angiography performed through the radial access showed aneurysmatic changes of the left circumflex artery with near occlusion of this vessel (90% stenosis by visual assessment) (Fig.1). A loading dose of ticagrelor has been administered and an immediate PCI of the infarct-related artery with guiding catheter Launcher 6FEBU 4.0, and BMW guidewire was performed. Pre-dilatation with balloon-catheter Maverick 3.5 × 15 mm up to 8 atm. Due to ectatic dilatation of the artery we have decided to implant a Self-Apposing® Coronary Stent Xposition S (Stentys SA, Paris, France). Post-dilatation was performed with 4.5 × 15 mm balloon catheter Maverick XL inflated to 6–8 atm. In optical coherence tomography (OCT) a good stent apposition has been confirmed with a few not adhering struts. The maximal distance between stent struts and the vessel wall was 0.5 mm (Figs. 1 and 2). A complete distal flow

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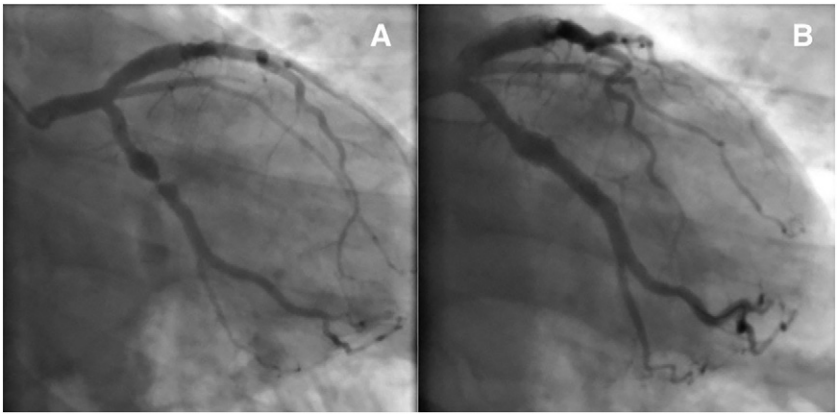


Fig. 1. Fluoroscopy image of the left coronary artery. A – left circumflex artery before the procedure; B – left circumflex artery after the procedure.

in the infarct-related artery was achieved. After four days of hospitalization the patient was discharged home in a good general condition with a recommendation for the use of dual antiplatelet therapy (acetylsalicylic acid and ticagrelor) for 12 months.

Discussion

Recently, Stentys company has presented a sirolimus-eluting version of Stentys stent (Xposistion S) with a new balloon delivery system aimed to facilitate stent positioning. The stent is now mounted on a semi-compliant balloon and is restrained by a splittable sheath. The balloon is inflated to relatively low pressures, only in order to split the sheath and release the stent. After deflation the balloon and sheath are withdrawn leaving the stent apposed to the vessel wall. This overcomes a forward jump complication of the device know from the previous trigger delivery system.[4–6] The latest version of Stentys Xposition S presents radio-opaque stent end markers to improve precise placement of the device. Self-apposing stents are particularly effective in angiographic situations where precise sizing is difficult.[1] In presented case choosing a proper stent size was not so easy because of aneurysmatic changes of the artery. The vessel diameter in the aneurysm area was about 6 mm (Figs. 1 and 2). Regular DES are commercially available in maximal sizes up to 4.5–5.0 mm. In this case we have chosen the largest Stentys stent size available (3.5–4.5 × 27 mm) which is designed to target vessels of diameter between 3.5 and 6.0 mm (Fig. 2).

The efficacy and safety of Stentys Self-Apposing stent in ST-segment elevation MI patients has been confirmed previously. No malapposed

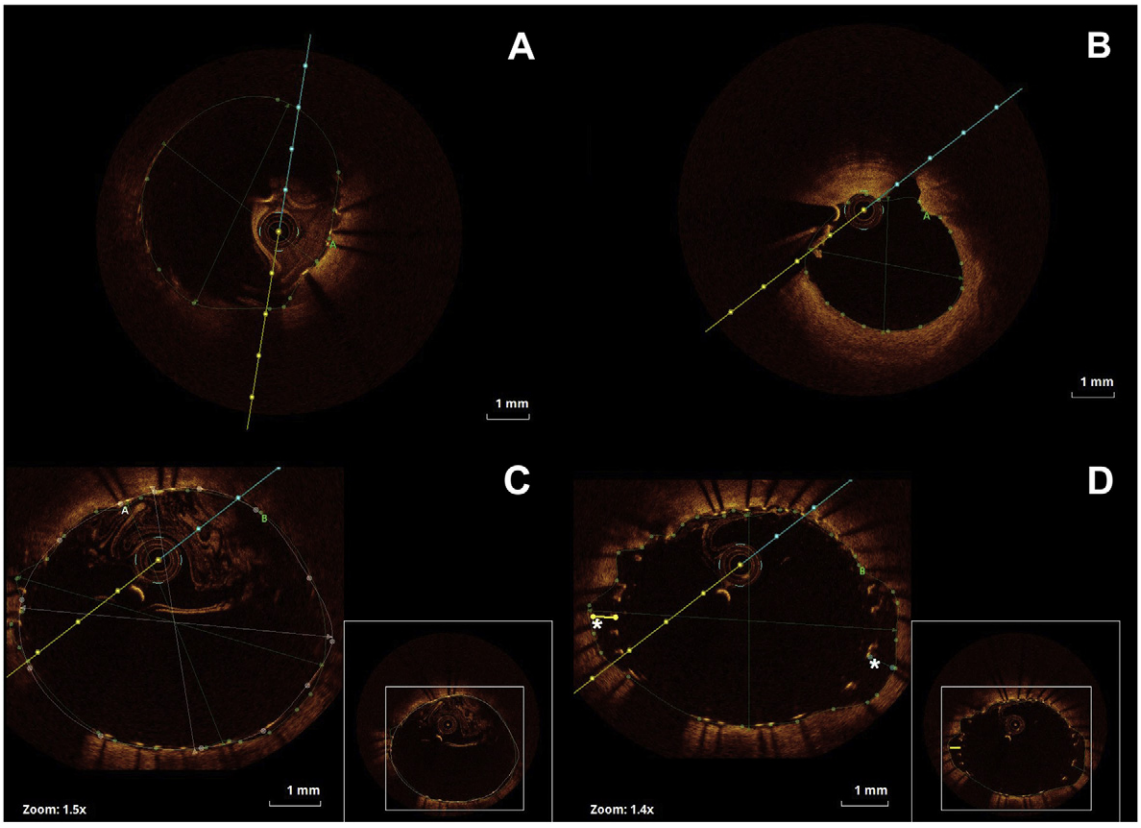


Fig. 2. Optical coherence tomography imaging of the left circumflex artery. A - distal part of the lesion with mean diameter of 4.87 mm and max diameter of 5.26 mm; B - proximal part of the lesion with mean diameter of 3.43 mm and max diameter of 3.76 mm; C - mid part of the lesion with mean diameter of 5.56 mm and max diameter of 6.29 mm; D - result of after stent implantation without complete stent apposition −0.5 mm (asterisk).

stent struts at 6 months, and lower rate of acute stent malapposition (at 3 days) as compared to balloon-expandable stents was reported.[7–9] In our case we performed post-dilatation after stent implantation, which is currently recommended to decrease the risk of late in-stent thrombosis.[10]

Self-apposing stents present useful features which might have advantages over conventional DES in specific angiographic situations. Thus, self-apposing stents could become a new therapeutic option for selected patients.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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Case report

Caseoma: A rare echocardiographic finding of mitral annulus☆

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ABSTRACT

Caseoma (caseous calcification of mitral annulus) is rare variant of common echocardiographic finding. Chronic caseous degeneration in the internal material of mitral annular calcification is the underlying mechanism of this condition. On the echocardiographic evaluation, the calcification is around the mass with central echolucent area, typically located at the base of the posterior leaflet. This echocardiographic finding can be mistaken for cardiac tumors, vegetations or abscesses.

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Introduction

The calcification of mitral annulus is a common echocardiographic finding in elderly individuals. Caseoma (caseous calcification of mitral annulus) is rare variant of common echocardiographic finding of mitral annulus. Chronic caseous degeneration in the internal material of mitral annular calcification is the underlying mechanism of this condition. On the echocardiographic evaluation, the calcification is around the mass with central echolucent area, typically located at the base of the posterior leaflet. This echocardiographic finding can be mistaken for cardiac tumors, vegetations or abscesses. The differential diagnosis is important.

Case report

A 78-year-old woman with a history of hypertension, diabetes mellitus and chronic obstructive pulmonary disease was admitted for dyspnea upon minimal exertion. Her white blood cell count was normal, erythrocyte sedimentation rate and CRP levels were minimally elevated. PA chest X-ray showed massive pleural effusion at right hemithorax and elevated cardiothoracic rate. Transthoracic echocardiography (TTE) showed a large calcified mass of 27 × 24 mm in the posterior mitral annulus that extended to the most basal area, the P1–P2 scallops, without causing valve stenosis (E velocity: 1,1 m/s) (Fig. 1) and with moderate mitral regurgitation (Fig. 2). Left atrial chamber and right heart chambers were dilated, left ventricular ejection fraction was 46% (modified Simpson) on echocardiographic evaluation. The patient was offered for transesophageal echocardiography, but patient refused invasive cardiac procedures. During the follow-up period, the patient was treated with supportive medical treatment.

Discussion

Echocardiographic series report a low prevalence of caseoma, between 0.06% and 0.07%, and up to 0.6% of patients with calcification of the mitral annulus (CMA).[1] In most patients, because of the asymptomatic course of caseoma, the condition is mostly underdiagnosed. Caseoma can be symptomatic, symptoms are associated with mitral valvular dysfunction, both stenosis and regurgitation. The initial diagnosis is made on echocardiography. Transthoracic echocardiography shows a spherical mass, with calcified borders and an echolucent area.[1,2] A calcified mass may be shown in the cardiac silhouette of the chest X-ray. Due to the structural deformation, caseomas associated with mitral valvular dysfunction, both stenosis and regurgitation. Transesophageal echocardiography will provide more information about the internal material of CMA, also the diagnosis will be greatly aided by computed tomography (CT) and cardiac magnetic resonance imaging.[3] The calcified envelope can be clearly detected by computed tomography. Cardiac magnetic resonance imaging gives the characteristics of the various sequences which will help to make differential diagnosis (especially tumors). The MRI signal is normally in T1- and T2-weighted sequences; contrast uptake is not expected at the periphery of the mass. In our hospital, transesophageal echocardiography and magnetic resonance imaging were not possible, and CT imaging was not performed because of the caution of radiation exposure. The differential diagnosis should include cardiac abscesses and tumors. The relationship between CMA and atrial fibrillation, aortosclerosis, and coronary artery disease has been studied before.[4] Because the mitral annulus is close to the region of the conduction system, any extension of calcified mass would affect how this

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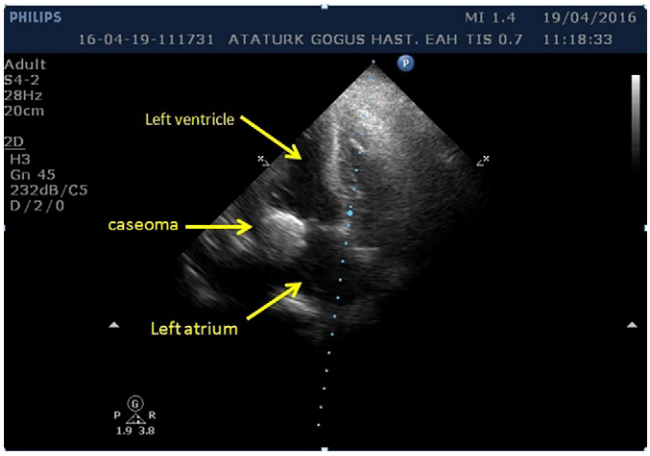


Fig. 1. Caseoma on TTE with apical four chamber view.

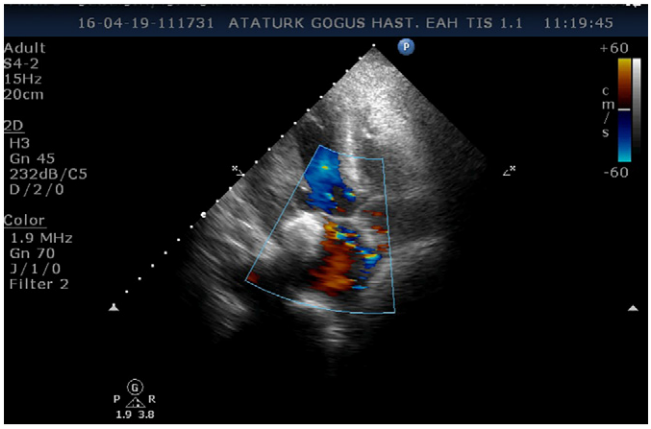


Fig. 2. Eccentrical mitral regurgitation caused by caseoma.

electrical conduction function, leading to intraventricular conduction delays. Due to the asymptomatic course, only monitoring of the patient's progress is recommended in most cases. Surgery should be chosen for patients with significant valvular dysfunction or an uncertain diagnosis.[2,3] In our case, due to the age of the patients and patient's chose, surgery was not performed. Because of that, no biopsy data were obtained. In conclusion, caseoma is a rare variant of calcification of the mitral annulus, but should be evaluated for the differential diagnosis with other cardiac masses to prevent unnecessary surgery.

Conflict of interest

None declared.

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Case report

Percutaneous VSD closure of a baby weighed 3 kg☆

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Introduction

The incidence of muscular VSD of 2.7 cases per 1000 live births.[1] The expected fate of muscular Ventricular Septal Defects (VSD) is that they decrease in size and finally close. Ventricular myocardium grows and fills the defect therefore small sized VSDs close spontaneously. It was reported that 33% of VSD closed in utero, 44% within the first postnatal year, and 23% did not close.[2] Large muscular defects if not treated may cause pulmonary hypertension and may affect the growth of children. Surgery can be an option but because of their location it is very hard to find such defects and approach because of coarse trabeculations. Therefore, firstly hybrid approaches, later percutaneous closures are done for treatment. Various devices are used for this purpose, nowadays Amplatzer Duct Occluder (ADO) II are common.

Case

Two months old baby was referred to our clinic with tachypnea, tachycardia and growth failure. She was born 2800 g 30 weeks of gestation and now she is 3000 g. Her mother recognized that she had dyspnea during feeding and irritable. Pansystolic murmur 2–3°/6 in degree was found by physical examination. Telecardiography showed mild cardiomegaly. Apical 4 mm large VSD was detected in the echocardiography, left side of heart was enlarged. 20 mm Hg gradient was detected between two ventricles. Tricuspid insufficiency was 2nd degree. Anti-congestive treatment was started but despite to the treatment, her symptoms continued. She failed to gain weight and hospitalized for lower respiratory tract infections twice. Surgical closure was discussed with the family and the surgeons but because of VSD location is apical which is hard to localize during surgery and the bodyweight of patient was low it was decided that morbidity and mortality of surgery would be high. Family was informed about percutaneous closure procedure; its risks, benefits and they accepted Transcatheter closure. After left ventriculography, we decided to close the defect from left ventricle side. The VSD was passed through by using a partly cut pigtail catheter. A hydrophilic glide wire was passed across the defect into the right ventricle and the defect was closed by 5 × 4 ADO II AS (St. Jude Medical, Plymouth, MN) device (Fig. 1). We did not face any complications like atrioventricular block during or after the procedure. No vascular complication was seen up till now. In control transthoracic echocardiography no aortic or tricuspid insufficiency was detected. She was discharged on the next day after the procedure (Fig. 2).

Discussion

Surgery is not the choice for the closure of muscular and apical VSDs because risk of residual shunting and reoperation is high. In each intervention mortality and morbidity increases.[3–5] Transcatheter closure of VSD is performed successfully in older children for years. Percutaneous VSD closure has certain risks like complete heart block, aortic, tricuspid insufficiency. In order to minimize such risks; appropriate device should be selected according to the type, location, and the size of the defect. Amplatzer muscular VSD occluders were used as device, later Amplatzer duct occluders (ADO) have been started to be used. ADO II was safe and effective for aneurysmatic perimembranous VSD closure as shown before.[6]

Transcatheter retrograde closure of VSD with ADO II (like our case) is possible and mentioned in the literature previously.[7] Also additional risks were found in infancy period like sheath size, stiffness of delivery. Diab et al.[8] reported the largest series of infants <1 year of age whose muscular VSD were closed with device. The smallest patient that they closed the VSD was 3.2 kg with periventricular and 3.8 kg with percutaneous approach. Zartner et al.[9] reported transvascular closure of single and multiple muscular VSDs in neonates and infants < 20 kg. They were able to close VSD by percutaneous approach in infants as small as 2.2 kg. However acute complete atrioventricular (AV) block occurred, the device was removed immediately. Then AV block was resolved and surgical closure was performed.

The smallest case was reported by Polat et al. They reported percutaneous closure of 13 days old newborn weighed 2.9 kg.[10]

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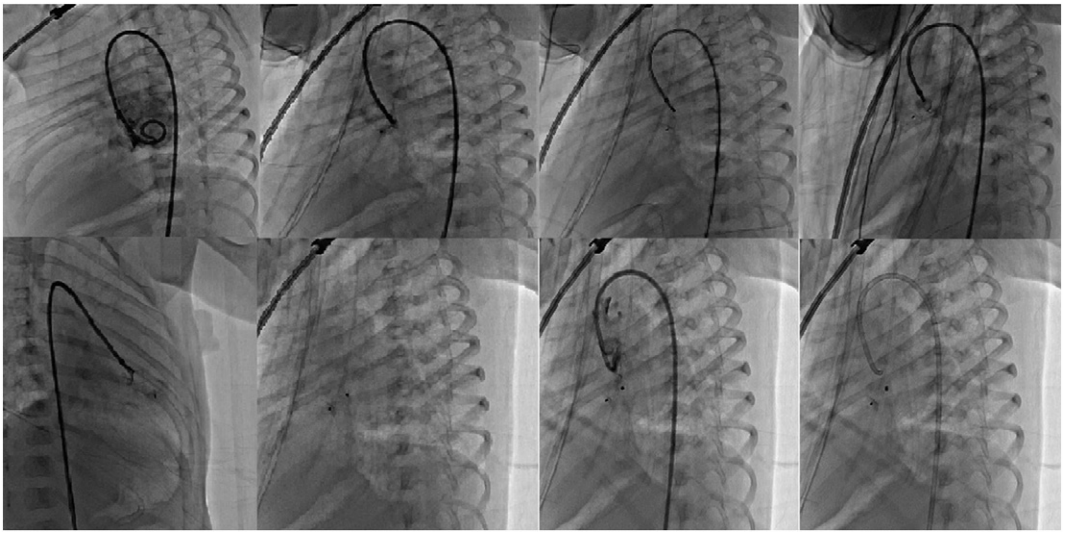


Fig. 1. Angiography images of percutaneous VSD closure.

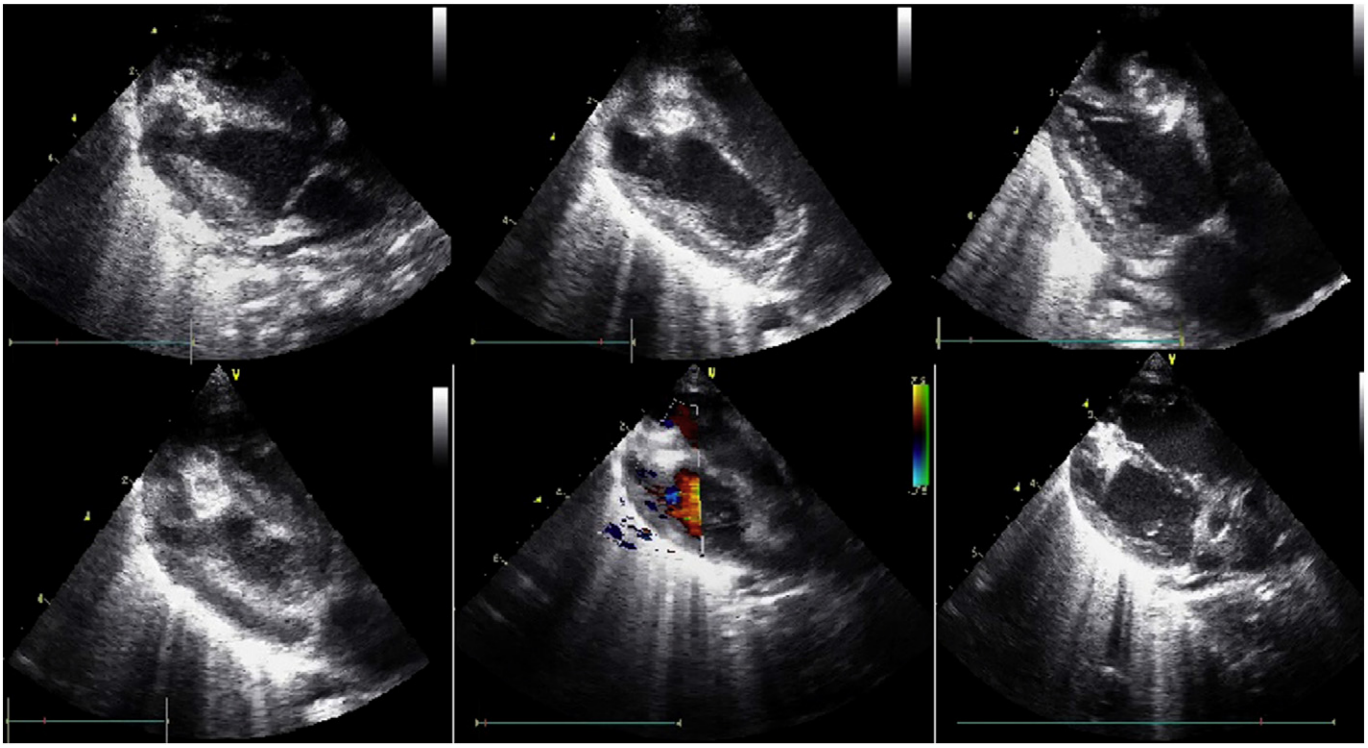


Fig. 2. Transthoracic echocardiography images after VSD closure.

Conclusion

We emphasized that firstly; if appropriate device with appropriate size was used with good technique in experienced hands percutaneous muscular VSD closure is a safe procedure second; this can be done with ADO 2 device form left ventricle side easily.

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Case report

Acute pulmoner embolism mimicking acute coronary syndrome

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ABSTRACT

Clinical and electocardiographic (ECG) features in pulmonary embolism (PE) lack of specificity and may mimic an acute coronary syndrom (ACS). We here report a case of a 56-year-old woman presenting with chest pain secondary to pulmonary artery embolism which was initially diagnosed as ACS due to electrocardiographic changes and raised troponin. PE presenting with negative T-wave inversion can mimic ACS and misdirect the diagnostic approach. Simultaneous T-wave inversions in anterior and inferior leads are important clues suggesting PE. Most common ECG findings in PE are anteroseptal T-wave inversion/ST-elevation or depression along with complete or incomplete right bundle branch block, sinus tachycardia, low QRS-complex voltage, an S1Q3T3 pattern, and right axis deviation. The reasons for the ECG changes that seem like ischemia are sudden RV strain, hypoxemia and the release of catecholamines. So we have to be aware that PE can present as acute coronary syndrome with ECG changes preoccupy ischemia

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Introduction

Clinical and electocardiographic (ECG) features in pulmonary embolism (PE) lack of specificity and may mimic an acute coronary syndrom (ACS). We here report a case of a 56-year-old woman presenting with chest pain secondary to pulmonary artery embolism which was initially diagnosed as ACS due to electrocardiographic changes and raised troponin.

Case

A 56-year-old diabetic, hypertensive, obese woman with a history of smoking was admitted to the emergency service with chest pain and nausea. She did not have any other relevant symptoms. Clinically, she was oriented, her cardiovascular and chest examination was unremarkable. Her pulse was regular at 93 beats/min, blood pressure was 142/89 mm Hg and respiratory rate was 18 breaths/min. The room air oxygen saturation was 99%. In her blood investigations everything was normal except troponin I which was 0.98 ng/ml (normal < 0.06 ng/ml). On her 12 lead ECG, the rhythm was sinus. There was ST segment depression in leads V3–V6 and T wave inversion in anterior and inferior leads and minimal ST segment elevation in AVR (Fig. 1). She was diagnosed as non-ST elevation myocardial infarction and referred to a third step hospital for diagnosis and treatment. Cause of her insistent chest pain invasive coronary evaluation planned. The coronary angiography revealed normal. Transthoracic echocardiography demonstrated good left ventricular systolic function but a dilated right ventricle (RV). There was mild tricuspid regurgitation with calculated RV systolic pressure of 45 mm Hg. So thoracic computed tomographic angiogram planned and it showed extensive PE of both right and left main pulmonary arteries extending distally into lobar, segmental and sub segmental branches (Fig. 2). A Doppler scan of the legs revealed no deep vein thrombosis. Her thrombophilia profile was normal. The

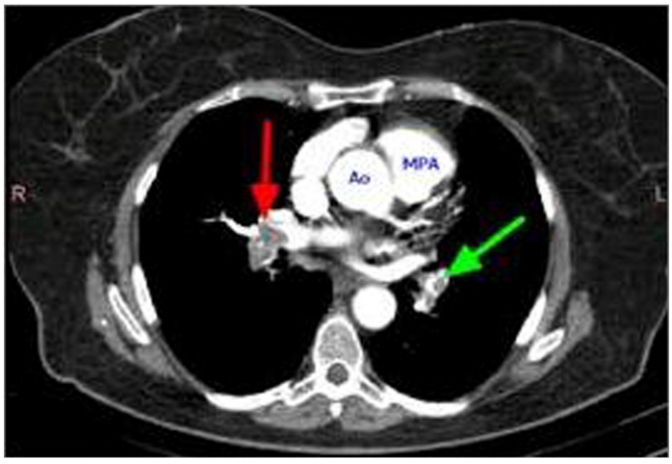


Fig. 1. Patient s 12 lead electrocardiogram on admission.

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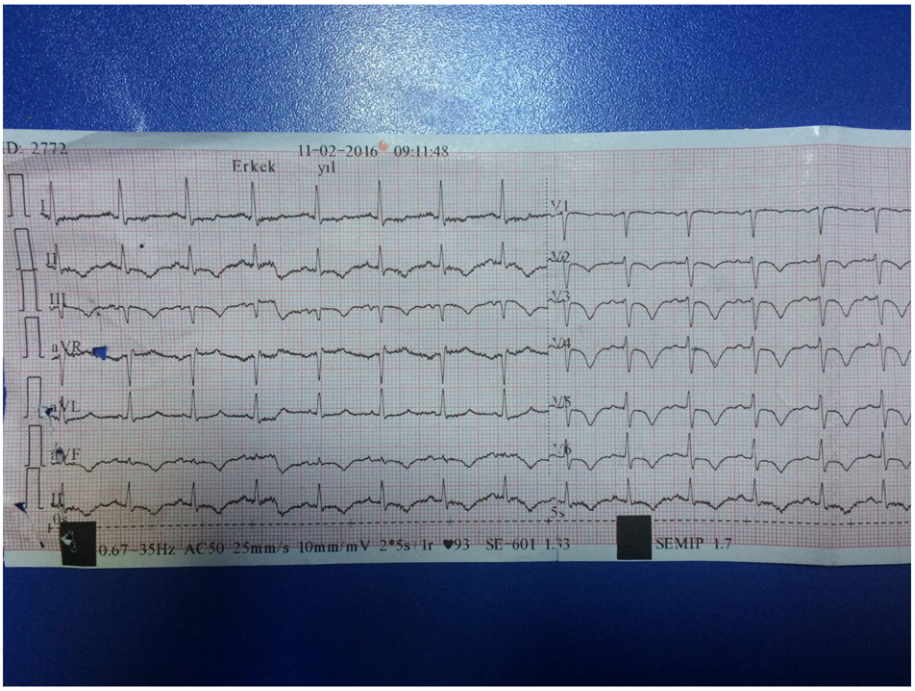


Fig. 2. Pulmoner embolism seen on pulmoner CT angiogram.

patient was treated with low molecular weight heparin (LMWH) along with overlap warfarin.

Conclusion

PE presenting with negative T-wave inversion can mimic ACS and misdirect the diagnostic approach. Simultaneous T-wave inversions in anterior and inferior leads are important clues suggesting PE. Most common ECG findings in PE are anteroseptal T-wave inversion/ST-elevation or depression along with complete or incomplete right bundle branch block, sinus tachycardia, low QRS-complex voltage, an S1Q3T3 pattern, and right axis deviation. The reasons for the ECG changes that seem like ischemia are sudden RV strain, hypoxemia and the release of catecholamines. In our case, patients' oxygen saturation was normal so we don't expect a catecholamine surge which is generally induced by severe hypoxemia. We think sudden pressure load on a noncompensatory right ventricle and global or focal ischemia induced by this additional strain may be the reason of the ECG changes. As a result, we have to be aware that PE can present as acute coronary syndrome with ECG changes preoccupy ischemia. Otherwise, the diagnosis of PE can be easily missed and patients may not receive appropriate treatment resulting in increased mortality.

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Case report

Surgical treatment of pulseless pediatric supracondylar humerus fracture☆

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ABSTRACT

Supracondylar humerus fractures in childhood may be accompanied by a vascular injury that is worse than the fracture. The early recognition of the vascular complications is crucial to perform an adequate, and rapid treatment that provides a good prognosis. Early and precise treatment of acute vascular injuries is necessary to avoid a delay which may lead to a permanent disability. In this article, a pediatric case of supracondylar humerus fracture with a brachial artery impairment and the successful surgical treatment is presented.
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Introduction

Supracondylar humerus fractures are one of the major pediatric elbow fractures that account for 55 to 80% of all upper limb fractures.[1] Most of these fractures occur as the result of a fall on an outstretched extremity.[2] Falling onto an outstretched hand with a hyperextended elbow results in displacement of the distal portion of the humerus.[3] The closeness of the vascular structures to the distal part of the humerus makes them susceptible to injury in case of a supracondylar humeral fracture.[4]

The prevalence of vascular injury in pediatric patients with Gartland type-3 supracondylar humeral fractures has been reported in a range from 14% to 18.6% in different series.[1,4,5] The mechanism of injury is associated with the mechanical displacement of the bone fragments over the neurovascular structures including the median nerve, radial nerve, and the brachial artery.[6] The posterolateral displacement has seen 64% of the brachial artery injuries related to the supracondylar humerus fractures whereas the posteromedial displacement is seen in 36% of the cases.[5] Vascular injury mechanism can either be an intimal damage or a total cut. Whatever the injury mechanism is, the vascular traumas have a high risk of morbidity associated with limb loss, and they require urgent management.[6] In this paper, successful surgical management of a child with brachial artery injury related to a supracondylar humerus fracture is presented.

Case report

A nine-year-old, otherwise healthy, right-hand-dominant boy was referred to the emergency department after he falls onto his left elbow. He described falling backward on a slippery surface while running and feeling immediate pain in the left elbow. On inspection, his humerus was immobile with a massive medial displacement. On physical examination, a closed supracondylar humerus fracture was determined. There was no hematoma or ecchymosis over the fracture region. The left antecubital brachial artery pulse, as well as the radial and ulnar pulses, was absent. The left hand was pale and cold that are indicative of poor perfusion. Neurological examination was normal with a normal motor and sensory function. Doppler ultrasound (DUS) examination revealed the cessation of brachial artery circulation at the level of the humeral fracture. Similarly, transverse slices of the Computerized Tomography (CT) angiography revealed an obstructed brachial artery image compatible with the site of dislocated humeral fracture line (Fig. 1).

The patient was taken to the operating room emergently by a team of orthopedics and cardiovascular surgery. Under general anesthesia, the humeral fracture was stabilized with percutaneous Kirschner wires following a closed reduction that was performed by the orthopedist. Despite the reduction, alignment, and fixation of the displaced fragments of the fractured humerus, distal pulses did not return. DUS examination revealed the same preoperative vascular signs without expected reflow. Traumatic brachial artery intimal injury was considered, and the surgical exploration of the brachial artery was performed. A discrete lesion of subintimal hematoma consistent with the arterial dissection was observed after the exploration of the brachial artery (Fig. 2). The injured arterial segment was resected (Fig. 3). Reconstructive anastomosis was performed in end-to-end fashion using 7-0 polypropylene suture (Fig. 4). Distal pulses returned soon after the release of vascular

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Fig. 1. Subsequent transverse slices of Computerized Tomography (CT) angiography revealed the cut brachial artery at the site of the humeral fracture line. Arrow is showing the brachial artery opacification.

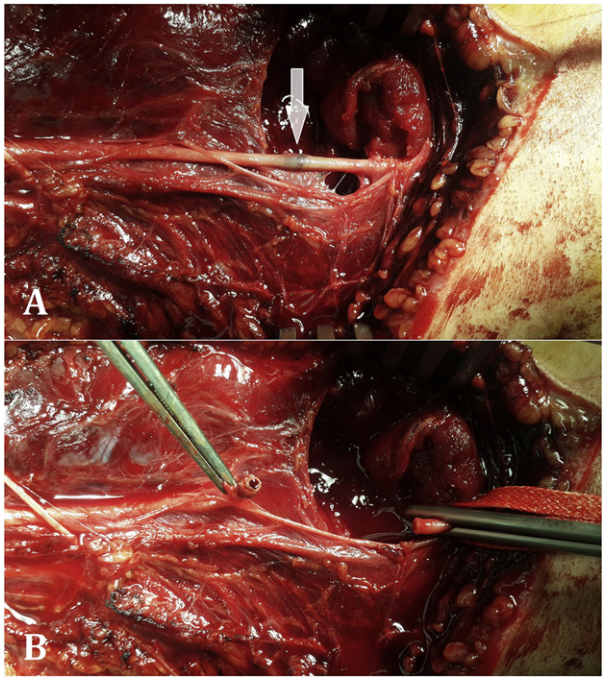


Fig. 2. (A) Brachial artery injury, the arrow indicates the injured segment with a subintimal hematoma. (B) Division of the injured part reveals a dissected brachial artery in which the intima and media layers were separated.

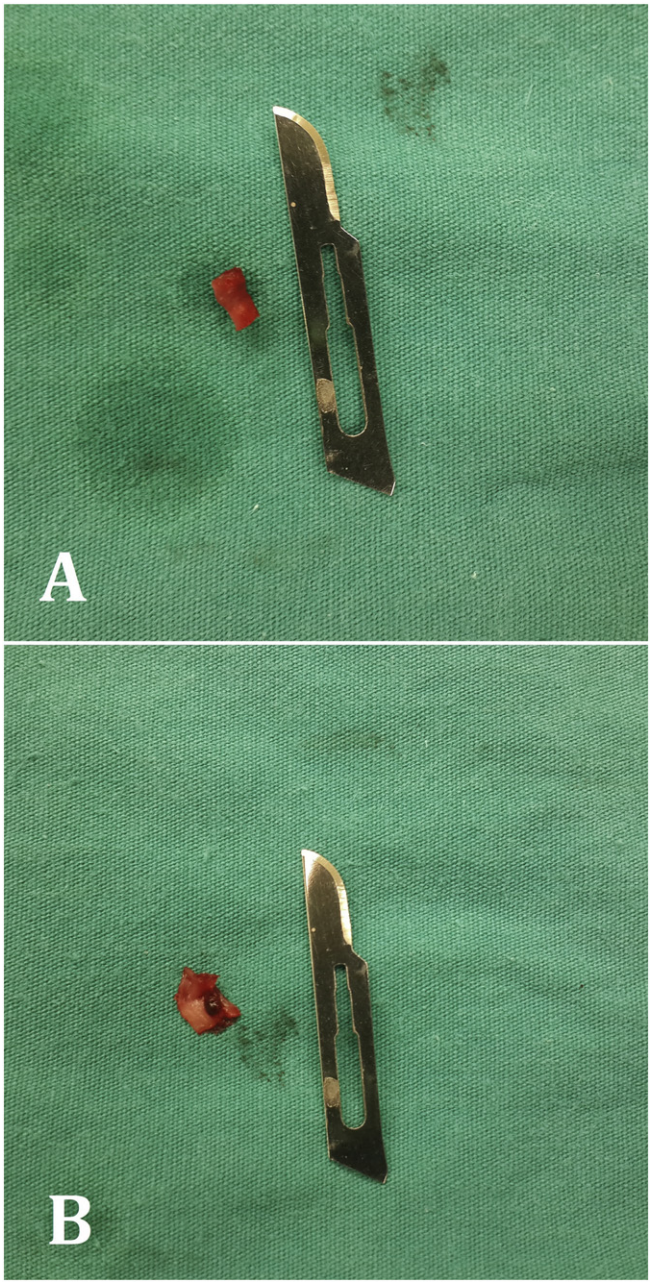


Fig. 3. (A) Macroscopic view of the resected brachial artery segment with an intimal injury. (B) Transsection of the injured segment reveals subintimal hematoma.

clamps. The patient was discharged fifth postoperative day with an arm-split and had no further complication.

Discussion

Pediatric supracondylar humerus fractures with a pulseless and poor-perfused hand should be operated emergently. If the limb still remains pulseless and ischemic after the surgical fracture fixation, vascular exploration, and repair (if needed) should be done. A pediatric supracondylar humerus fracture with a pulseless but well-perfused hand should be operated urgently. If the limb still remains pulseless after the surgical fracture fixation, vascular status should subsequently be reassessed. However, there are some controversies exist regarding the optimal management of this type of pediatric supracondylar humerus fractures with a pulseless but well-perfused hand. The opinions include an immediate vascular exploration or a twenty-four to forty-

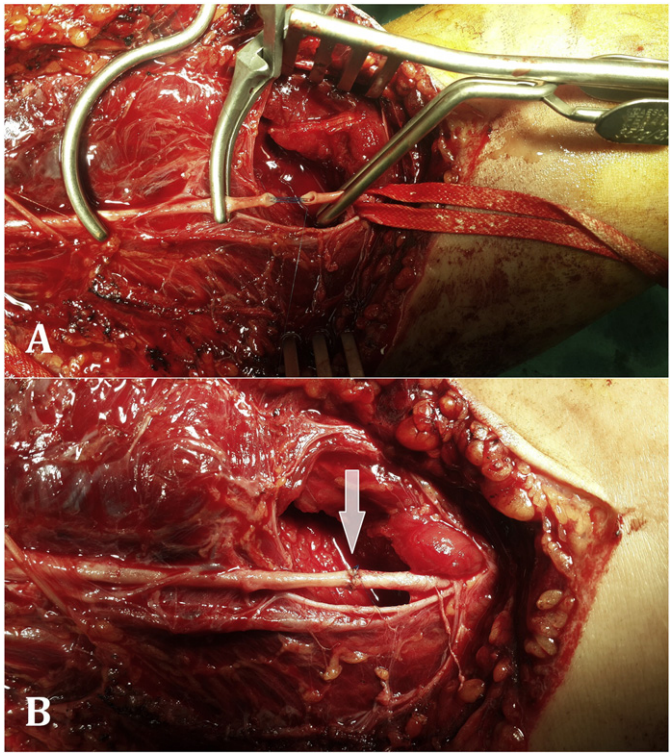


Fig. 4. (A) Reconstruction with end-to-end anastomosis of the clear edges. (B) The arrow shows the anastomotic line of the repaired brachial artery.

eight hour observation. The patient is returned to the operating room immediately for an emergent vascular management if perfusion of the hand is compromised during this period of observation.[7] For patients whose palpable pulses do not return after reduction and fixation, many authors recommend surgical exploration if there is lack of a normal DUS signal.[4]

In this case, it was aimed to be emphasized that the vascular injury should always be considered after a supracondylar humerus fracture in children. Because displacement of the supracondylar humerus fractures in children often causes serious injuries to vascular structures of the affected limb. The brachial artery is mostly affected due to its intimacy with the humerus. Thus, pulseless and poorly perfused hands require emergent evaluation for the brachial artery injury in case of humeral fractures. Postponing the surgical intervention may cause permanent vascular dysfunction or even limb loss. Therefore, a prompt surgical management that is crucial for the limb protection should rapidly be constituted.

Conflict of interest

None declared.

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Case report

Rheumatic valvulitis – Forgotten, but not gone☆

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ABSTRACT

Acute rheumatic fever is an exceedingly rare condition in the developed world. It is thought to have nearly disappeared in the United States. Through the case of a 41-year-old Caucasian female who presented with progressive dyspnea and symptoms of heart failure, due to subacute rheumatic valvulitis, the importance of multimodality cardiovascular imaging assessment of patients presenting with severe, multi-valvular dysfunction is highlighted. This case also serves as a powerful reminder for clinicians that rheumatic valvulitis should remain in the differential diagnoses for patients with otherwise unexplained severe, multi-valvular dysfunction.
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Introduction

Acute or subacute rheumatic fever in adults in the developed world is exceedingly rare, such that most clinicians assessing patients presenting with multi-valvular dysfunction would not even consider rheumatic valvulitis as a differential diagnosis. We report the case of a 41-year-old Caucasian female who developed severe multi-valvular regurgitation as a result of subacute rheumatic valvulitis in the United States. This case serves as a powerful reminder for clinicians that rheumatic valvulitis is an important and relevant condition, which is not yet "*gone, but not forgotten*", but rather "*forgotten, but not gone*".

Case record

A 41-year-old Caucasian female first developed dyspnea on exertion in August 2015. Her relevant medical history includes obesity and chronic rhinitis complicated by post-nasal drip. Her dyspnea was initially attributed to possible asthma and lower respiratory tract infections. A trial of inhaled bronchodilators and antibiotics did not help. By March 2016, she was in New York Heart Association functional class III, becoming short of breath with routine activities of daily living. She also reported four-pillow orthopnea, paroxysmal nocturnal dyspnea and peripheral edema. Echocardiography performed at a local hospital demonstrated severe aortic, mitral and tricuspid regurgitation. N-terminal pro-brain-derived natriuretic peptide was significantly elevated at 14,900 (normal: <125 pg/mL). Erythrocyte sedimentation rate (ESR) was moderately elevated at 50 mm/h (normal: 0–15 mm/h). Serum C-reactive protein (CRP) was mildly elevated at 10.5 (normal: 0–1.0 mg/dL). She was managed with standard heart failure therapies. Her symptom status improved following oral diuretic treatment. At this stage, the patient was referred to our center for further assessment.

On further history, the patient recalled an episode of severe pharyngitis six years ago. She had to take time off work for two days at that time. She did not seek medical attention at that time. The patient did not have a prior history of valvular heart disease. There was also no family history of valvular heart disease or sudden cardiac death. The patient did not use illicit substances. The patient has not had any pharmacological anti-obesity treatment. In particular, she has not taken fenfluramine or dexfenfluramine. An extensive auto-immune screen including lupus anticoagulant, anti-cardiolipin, beta-2 glycoprotein, anti-double stranded deoxyribonucleic acid, anti-nuclear, anti-Smith, anti-Jo-1, anti-Ro, anti-La, anti-ribonucleoprotein, cytoplasmic and peri-nuclear anti-neutrophil cytoplasmic antibodies, and rheumatoid factor, were unremarkable. Serum complement levels were within normal limits. Testing for syphilis and human immunodeficiency virus were negative.

The patient underwent further imaging investigations at our center. Repeat transthoracic echocardiography demonstrated a dilated left ventricle with preserved left ventricular ejection fraction. The mitral valve was abnormally thickened with restricted diastolic opening. There was severe (4+) pan-systolic mitral regurgitation (Fig. 1; Supplementary Videos 1 and 2). The aortic valve was also severely thickened with restricted leaflet opening. There was severe (4+) pan-diastolic aortic regurgitation, associated with significant flow reversal within the upper descending thoracic aorta (Fig. 2; Supplementary Video 3). Additionally, the tricuspid valve was also thickened. There was severe tricuspid regurgitation (Fig. 3). Aortic valve morphology was difficult to appreciate on echocardiography, due to abnormal thickening of the

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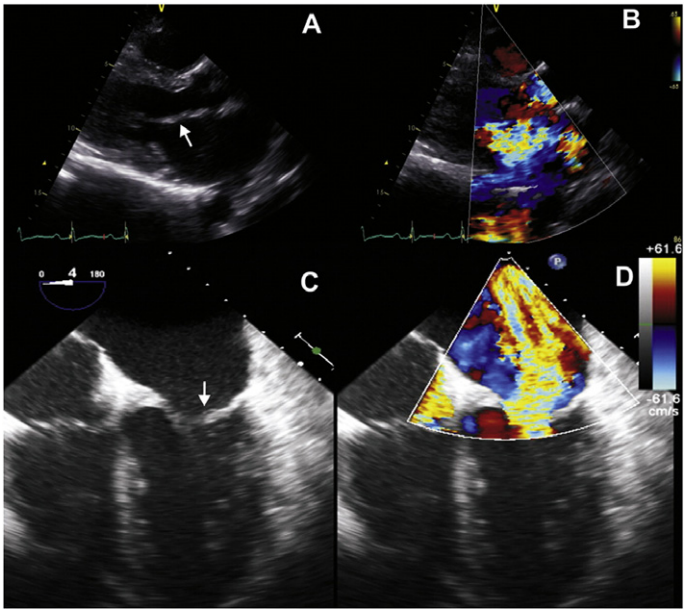


Fig. 1. Abnormally thickened mitral valve leaflets with restricted opening on echocardiography (A: parasternal long-axis view on transthoracic echocardiography; C: mid-esophageal four-chamber view on transesophageal echocardiography; arrows point to thickened mitral valve leaflets). Severe, torrential mitral regurgitation on color Doppler analysis (B: parasternal long-axis view on transthoracic echocardiography; D: mid-esophageal four-chamber view on transesophageal echocardiography).

valve leaflets. Multi-detector cardiac computed tomography (MDCT) demonstrated a thickened trileaflet aortic valve (Fig. 4). Cardiac magnetic resonance imaging (CMR) was performed to exclude pan-aortitis. There were no supportive features of Takayasu arteritis. Significant multi-valvular regurgitation was confirmed by CMR (Fig. 5; Supplementary Videos 4 and 5).

The patient underwent mechanical mitral valve replacement (#25 On-X), mechanical aortic valve replacement (#21 On-X), and tricuspid valve repair (#28 Carpentier Classic annuloplasty ring). At the time of operation, the aortic and mitral valves were found to be severely thickened and retracted. Histological examination of the aortic and mitral valves demonstrated severe fibrosis with neovascularization consistent with post-inflammatory scarring. The gross appearance and microscopic features of the valve pathology were most consistent with rheumatic valvulitis (Fig. 6). Post-operatively, the patient has remained clinically well, in New York Heart Association functional class I. She has been commenced prophylactic antibiotic therapy, as secondary prevention for rheumatic fever. Due to the unusual nature of the patient's presentation, and the rare occurrence of rheumatic fever in adults in the United States, rheumatic fever was not even considered as a differential diagnosis at the time of

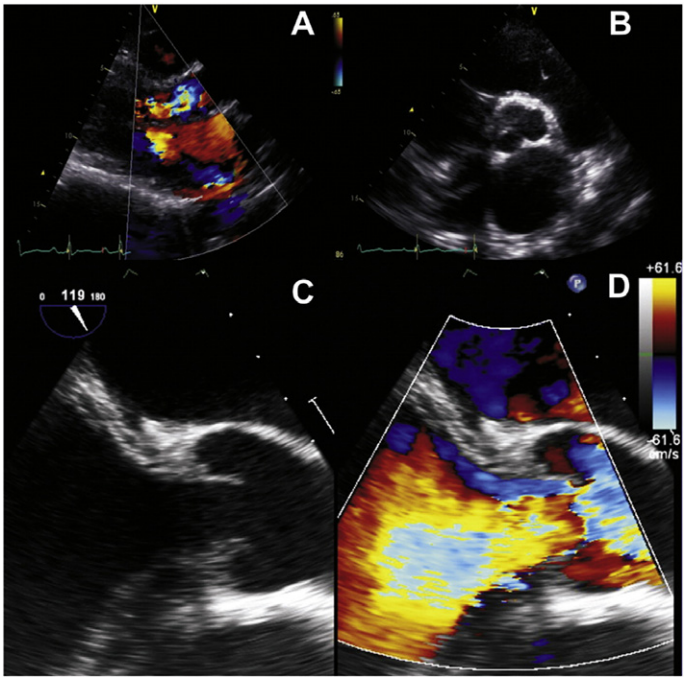


Fig. 2. Severe, torrential aortic regurgitation demonstrated on color Doppler analysis (A: parasternal long-axis view on transthoracic echocardiography; D: mid-esophageal long-axis view on transesophageal echocardiography). Aortic valve leaflets were abnormally thickened (C: mid-esophageal long-axis view on transesophageal echocardiography), however, the morphology of the aortic valve was rather difficult to appreciate on echocardiography (B: parasternal short-axis view on transthoracic echocardiography).

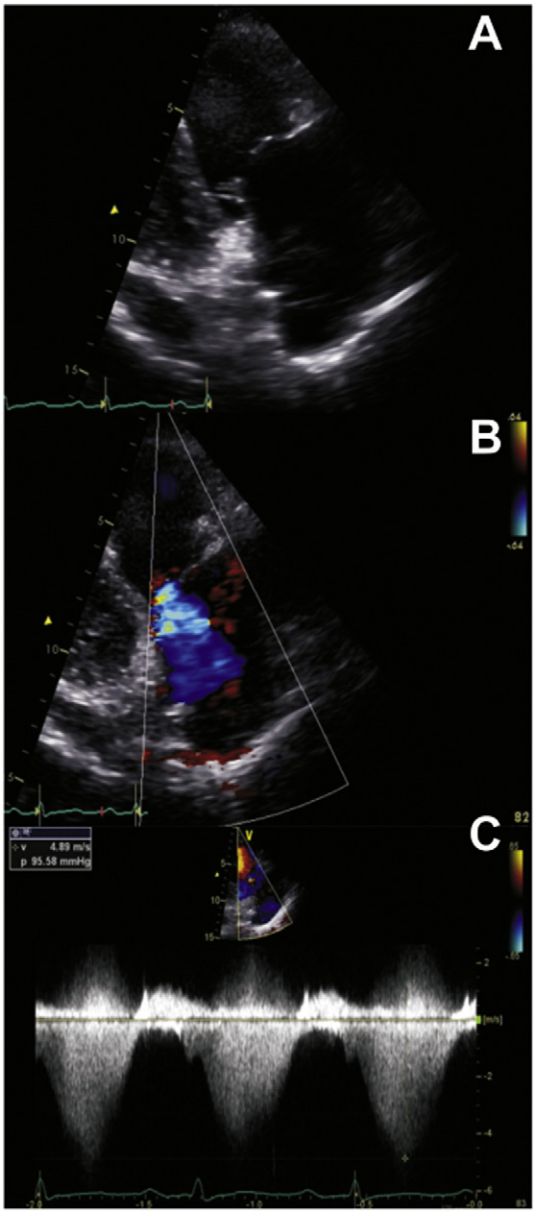


Fig. 3. Mildly thickened tricuspid valve leaflets (A: right ventricular inflow view on transthoracic echocardiography), with significant tricuspid regurgitation (B: color Doppler analysis in right ventricular inflow view on transthoracic echocardiography). Estimated right ventricular systolic pressure was severely elevated (C: continuous wave Doppler recording through tricuspid regurgitant signal in right ventricular inflow view on transthoracic echocardiography).

initial clinical assessment. Six weeks post-operatively, the patient underwent serum anti-Streptolysin O titer and anti DNAse B titer testing. At that stage, her serum anti-Streptolysin O titer was 88 (normal range: 0–530 IU/mL). Her anti DNAse B titer was 165 (normal range: 0–300 U/mL).

Discussion

We report a case of subacute rheumatic valvulitis causing severe aortic, mitral and tricuspid regurgitation in a 41-year-old Caucasian female in the United States. Nowadays, acute rheumatic fever is an exceedingly rare condition in the developed world, and is thought to have nearly disappeared in the United States.[1] To the best of our knowledge, this is the first reported case of rheumatic valvulitis in an adult in the developed world in recent English literature. The global incidence of acute rheumatic fever in children between 5 and 14 years of age is estimated at 300,000 to 350,000 per year.[2] The diagnosis of rheumatic fever can be made using the revised Jones criteria.[3] In low-risk populations, the diagnosis can be made with two major manifestations (carditis, arthritis, chorea, erythema marginatum, subcutaneous nodules), or one major and two minor criteria (polyarthralgia, fever, elevated ESR and/or CRP, prolonged PR interval).[3] The most recent rheumatic fever outbreak in the pediatric population in the United States was due to isolates of the rheumatogenic group A streptococcus M-type in Utah in 1998.[4] The fundamental pathophysiology of rheumatic fever results from molecular mimicry between group A streptococcal antigens and host antigens, in a genetically susceptible host with an abnormal host immune response.[2] However, the specific mechanisms that render each individual host susceptible to developing rheumatic fever are less clear. Even though the patient's serum anti-Streptolysin O and anti DNAse B titers were within the normal range, the tests were performed post-operatively, and it is very likely that the elevated titers may have normalized. The extensive negative auto-immune screen, the history of preceding severe pharyngitis six years ago, the unusual echocardiographic findings of abnormally thickened valves with severe regurgitation, the rheumatic appearance of the aortic and mitral valves at the time of operation, and the severe fibrosis with post-inflammatory scarring on histological examination of the valves, lend support to rheumatic valvulitis being the etiology of the patient's multi-valvular dysfunction.

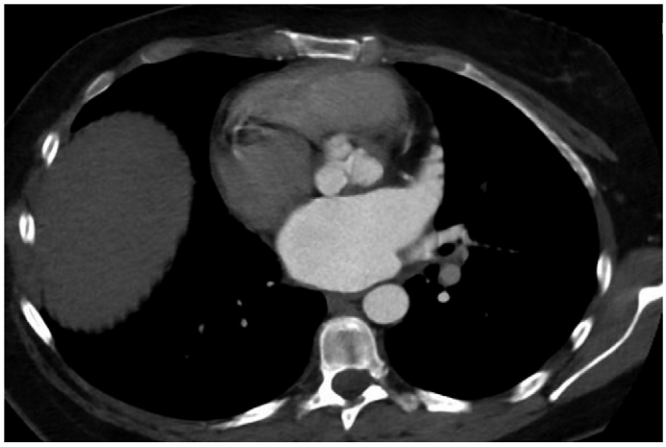


Fig. 4. The morphology of the aortic valve was well demonstrated to be trileaflet on multi-detector computed tomography (contrast-enhanced axial image).

This case is a powerful reminder for clinicians, highlighting the fact that rheumatic valvulitis, although exceedingly rare in the developed world, should still be considered as an etiology for otherwise unexplained severe multi-valvular dysfunction. The comprehensive management of rheumatic valvulitis should incorporate multi-modality cardiovascular imaging assessment, appropriate medical and surgical management of valvular dysfunction, and long-term secondary antibiotic prophylaxis.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ijcac.2017.05.001.

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Conflict of interest statement

The authors have no conflicts of interest to declare.

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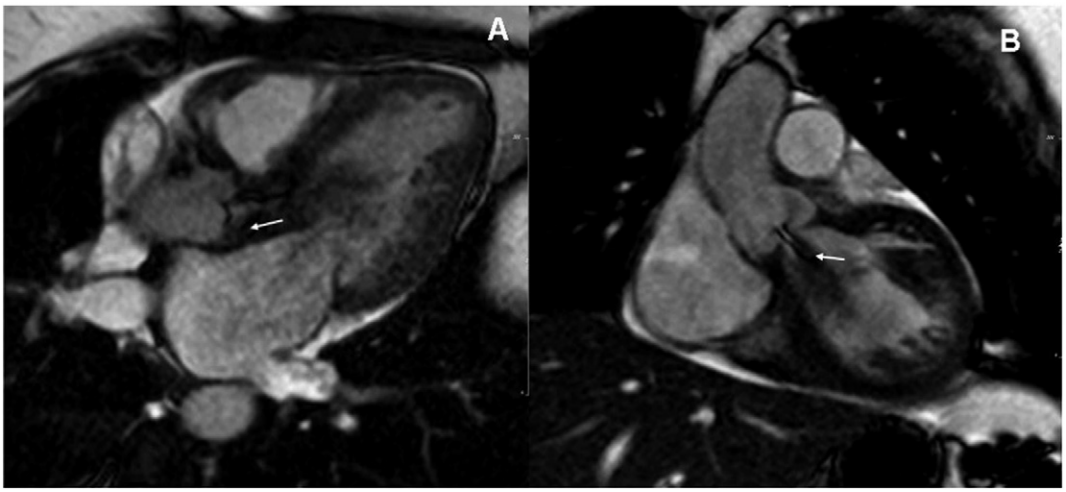


Fig. 5. Cardiac magnetic resonance imaging confirmed the presence of severe aortic regurgitation, represented by signal dephasing (arrows; A: axial image through the left ventricular outflow tract on the steady-state free precession sequence; B: oblique image through the left ventricular outflow tract on the steady-state free precession sequence). There was no evidence of Takayasu's arteritis on cardiac magnetic resonance imaging.

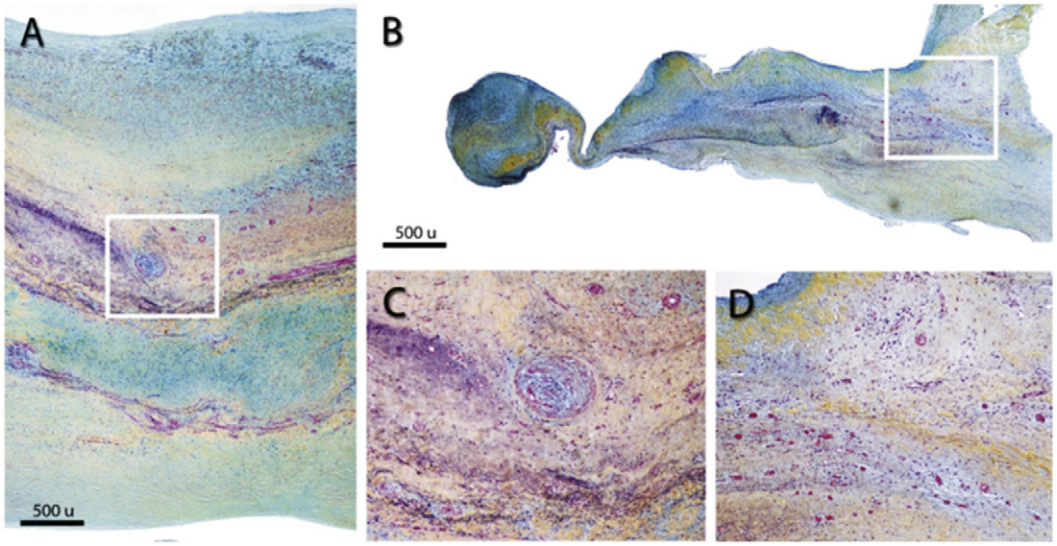


Fig. 6. Histologic sections of the mitral (A) and aortic valves (B) stained with Movat pentachrome shown in the same scale demonstrated fibromyxoid thickening of the leaflets which was more severe in the mitral valve. The boxed areas in A and B are shown in higher magnifications in C and D, respectively. In addition to the fibrosis (yellow material), there was proliferation of numerous small vessels as a result of valvulitis.

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Case report

Kounis syndrome due to deksketoprofen trometamol administration☆

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ABSTRACT

Kounis syndrome is a rarely seen syndrome which is also called allergic angina syndrome. Contacted allergic agents cause allergic reaction, and then it leads to vasospastic angina pectoris or myocardial infarction. When the patient contact allergic agent, histamine releases from mast cells, and vasospasm or plaque rupture occurs at coronary arteries. A lot of allergic agents is defined which causes Koinus syndrome. Most of these allergic agents are antibiotics, analgesics, antineoplastics, contrast agents, anethesics, non-steroid antiinflammatory drugs, proton pump inhibitors and venoms. Our patient is the first case report which is the Kounis syndrome after taking deksketoprofen trometamol.
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Introduction

Imbalance of myocardial oxygen supply and demand cause myocardial ischemia which leads to development of angina pectoris. One of the reasons of myocardial ischemia is coronary vasospasm which is resulted from the contraction of epicardial coronary arteries. If coronary vasospasm become longer, it may causes myocardial infarction.

Myocardial ischemia or infarction is sometimes occurred due to allergic agents. This entity is defined as allergic angina syndrome which is also called Kounis syndrome by Kounis and Zafras at 1991,.[1] After contacting with allergens, inflammatory mediators like histamine and leukotriens, releasing from mast cells, causes vasospasm or plaque rupture of coronary artery. Plaque rupture, vasospasm and thrombus of coronary arteries cause myocardial ischemia and myocardial infarction,.[2,3] Histamine is an important biochemical mediator and its effects via H1 histamine receptors at smooth muscle cells and coronary arteries,.[4] The other reasons of allergic ischemia are collagen destruction and plaque rupture, caused by metalloprotein activators like triptase and kimase,.[4,5]

In this paper we present a case developing vasospastic angina pectoris after taking deksketoprofen trometamol.

Case

A 49-year-old man admitted to emergency department with a complaint of itching and angina pectoris. His symptoms had begun 30 min after taking 25 mg deksketoprofen trometamol for his headache. On admission patient's blood pressure was 110/70 mmHg and heart rate was 86 pulse/min. There was no abnormality on his cardiovascular and other system examinations. He had an only allergy anamnesis for shaving foam. He was a smoker (35 years; 20 pieces/day). At emergency department, 5 mg intravenous feniramine and intravenous 20 mg methyl prednizolone had been administrated as a first intervention for his itch. Since the patient had also angina pectoris we performed twelve derivations electrocardiogram (ECG) which revealed ST segment elevation in inferior derivations (D2, D3, aVF),(Fig. 1). However ST segment elevation had disappeared while he was transferring to cardiology clinic (Fig. 2). Patient was hospitalized at coronary intensive care unit for medical treatment.

There was no abnormality in biochemical and cardiac markers and transthoracic echocardiography revealed 60% ejection fraction with no segmental motion abnormality. Coronary angiography was demonstrated that there were some plaques on left anterior descending artery, circumflex artery, and right coronary artery (RCA) (Fig. 3a). In this context we thought that, the patient's clinic was vasospastic angina and we performed ergonovine test to evaluate vasospastic angina pectoris diagnosis. We gave 10 μg and 20 μg methyl ergonovine to the right coronary artery because of ST elevation on inferior derivations. Patient described chest pain and pressure on his chest, after giving 20 μg methylergonovine. We performed an ECG and there was ischemic T wave negativity on inferior derivations. Simultaneously coronary angiography showed the vasospasm on RCA (Fig. 3b).Intravenous nitroglycerine was given and then his symptoms and ECG findings were disappeared.

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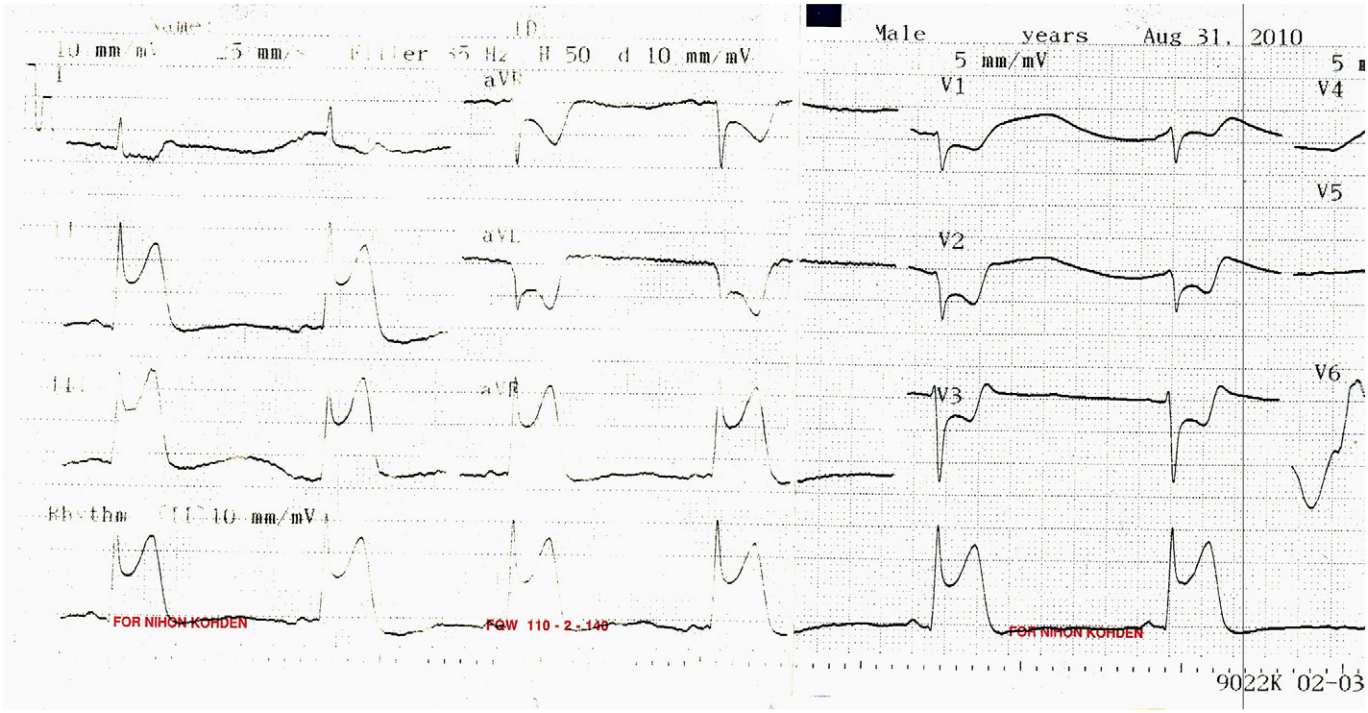


Fig. 1. Electrocardiogram revealed ST segment elevation in inferior derivations after taking 25 mg deksketoprofen trometamol.

Discussion

Kounis syndrome called as "allergic angina syndrome" was first described by Kounis and Zarvas in 1991.[1] Kounis syndrome is mostly seen in elderly patients Most of whom have an atopy history.

After contacting with allergens (drugs, venom, etc.…), antigene or antigene-anticore complex causes mast cell activation or type 1 anaphylaxis. Histamine, neutral protease (kimase, tripase), asite hidrolase and platelet activated factors are released from mast cells following allergic reaction. These chemical mediators lead to hypotension, vascular collapse, tachycardia, ventricular arrhythmia, vasospasm on coronary arteries and plaque rupture on cardiovascular system which ultimately result in myocardial infarction or cardiac arrest,.[4]

There are two types of Kounis syndrome. Type 1 Kounis Syndrome is seen in patients with normal coronary arteries and low cardiovascular risk factors,.[2] Whereas type 2 Kounis syndrome generally seen in patients having coronary heart disease or plaque on coronary arteries. Cardiac markers may increase in both types. Myocardial damage is generally developed on inferior wall at Kounis syndrome but anterior wall may be affected as well.[5]

Antibiotics, analgesics (aspirin, dipyrone), antineoplastics, contrast agents, corticosteroids, anesthetics, thrombolytics, anticoagulants, proton pump inhibitors, Skin disinfectants, bee and snake venoms may cause Kounis syndrome. Non-steroid antiinflammatory drugs (alclofenac, diclofenac, naproxen) can also cause Kounis syndrome. Majority of the cases with Kounis syndrome have an atopy history.[2] Our

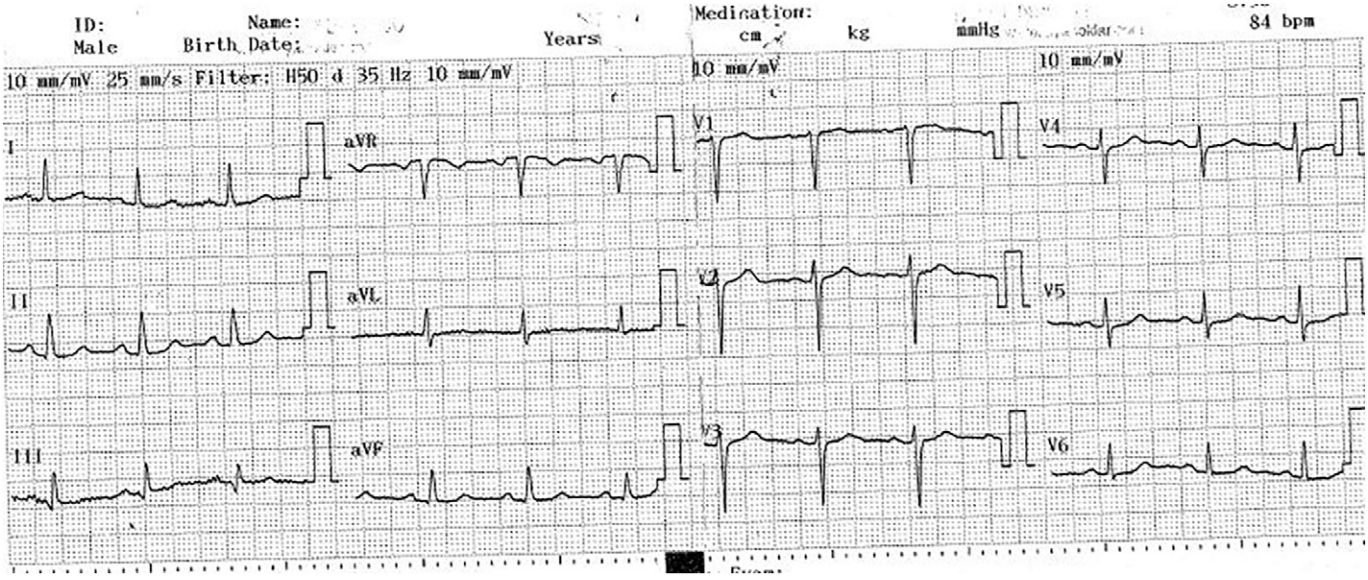


Fig. 2. Electrocardiogram revealed ST segment resolution following the treatment of the allergic reaction.

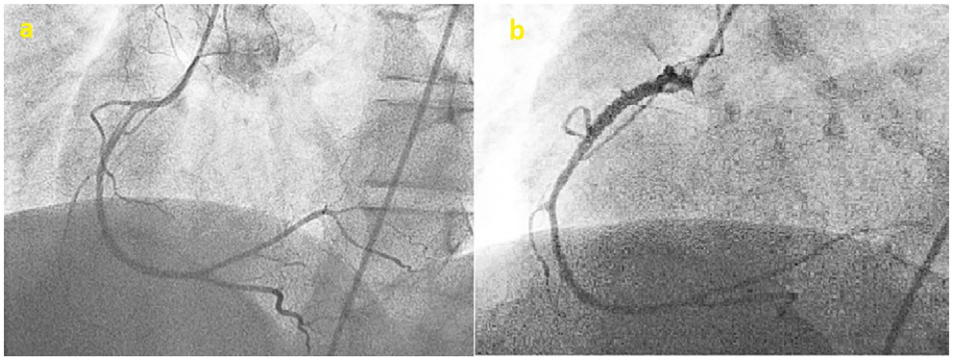


Fig. 3. a Shows non-critical atherosclerotic plaque formation in right coronary artery. b Shows 70% and more spasm in mid right coronary artery and 90% and more focal spasm in osteal posterior descending and posterolateral branch of right coronary arter artery following ergonovine administration.

case has been the first reported Kounis syndrome due to deksketoprofen trometamol which is the member of non-steroid antiinflammatory drugs.

After definite diagnosis, treatment should be directed to the reason and treatment should be targeted to the allergic reaction.

In this case, we thought Kounis syndrome because of his symptoms began after taking deksketoprofen trometamol. He has an atopy history (shaving foam) also. He had itching all of his body, nausea, dizziness associated with anaphylaxis after taking deksketoprofen trometamol. Simultaneously patient has described typical angina pectoris and there was inferior ST segment elevation on ECG. ST elevation disappeared after taking intravenous feniramine and intravenous methyl prednizolone. Cardiac markers did not increase. We saw vasospasm on right coronary arteries and T wave negativity at ergonovin test. These findings confirmed that Type 2 Kounis syndrome.

Conclusion

In conclusion, this is the first case defined as Kounis syndrome due to deksketoprofen trometamol. Patients with Kounis syndrome or atopy anamnesis should be carefully evaluated while taking any medicine. If a drug has not been used before by the patient with Kounis syndrome, the first dose should be administrated in hospital.

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Case report

Pulmonary embolism presenting with ST segment elevation in inferior leads☆

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ABSTRACT

Acute pulmonary embolism is a form of venous thromboembolism that is widespread and sometimes mortal. The clinical presentation of pulmonary embolism is variable and often nonspecific making the diagnosis challenging. In this report, we present a case of pulmonary embolism characterized by ST segment elevation in inferior leads without reciprocal changes in the electrocardiogram.

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Introduction

Acute pulmonary embolism (PE) is a form of venous thromboembolism (VTE) that is widespread and sometimes mortal.[1] The clinical presentation of PE is variable and often nonspecific making the diagnosis challenging.[2] Patients can be diagnosed and therapy administered quickly to reduce the associated morbidity and mortality.[3] Electrocardiographic (ECG) changes are very unspecific and range from most common sinus tachycardia, rightward shift in QRS axis, complete or incomplete right bundle branch block, precordial T wave inversion, S1Q3T3 pattern and more uncommon ST segment elevation.[4] In this report, we present case of pulmonary embolism characterized by ST segment elevation in inferior leads without reciprocal changes in the electrocardiogram (ECG).

Case report

76 year-old female patient was admitted to our emergency department because of chest pain and acute onset breathlessness. Her past medical history included hypertension and her coronary angiography showed normal coronary artery two years priorly. She was getting immobile because of left arm fracture for six months. On physical examination, her blood pressure was 102/65 mm Hg, heart rate was 102 bpm and oxygen saturation was 88%. Electrocardiography (ECG) showed ST segment elevation in inferior leads without no reciprocal changes (Fig. 1). Transthoracic echocardiography revealed normal left ventricular systolic function without segmental contraction defects and slightly right ventricular enlargement. Systolic pulmonary pressure was 45 mm Hg and tricuspid annular plane systolic excursion (TAPSE) and tricuspid annular velocity were 16 mm and 10 cm/s, respectively. Laboratory test results were unremarkable but troponin-I level increased. Re-tests 3 h later, there was no changes according to first troponin level. On the basis of physical examination, past medical history, ST segment elevation without no reciprocal changes, pulmonary embolism was suspected. Then we performed contrast-enhanced chest computed tomography. Chest computed tomography scan showed pulmonary embolism (Fig. 2). At the time of diagnosis of pulmonary embolism she was hemodynamically stable, therefore she received continuous infusion of unfractionated heparin. After 5 days heparin infusion, ECG changes was getting normal and ST segment elevation in inferior leads was resolved (Fig. 3). In a few days heparin, infusion was substituted for warfarin and enoxaparin. International normalized ratio (INR) level became therapeutic range and she discharged successfully.

Discussion

The symptoms and clinical features of pulmonary embolism are variable.[2] As stated previously, pulmonary embolism present several electrocardiographic (ECG) changes but ST segment elevation is rare condition. We demonstrated this rare condition in pulmonary embolism. This case highlights the fact that pulmonary embolism still remains one of the biggest masqueraders in acute coronary syndrome. Physicians should be careful in patients presenting with ST segment elevation and pulmonary embolism should be kept in mind differential diagnosis of acute coronary syndrome.

Conflict of interest

None.

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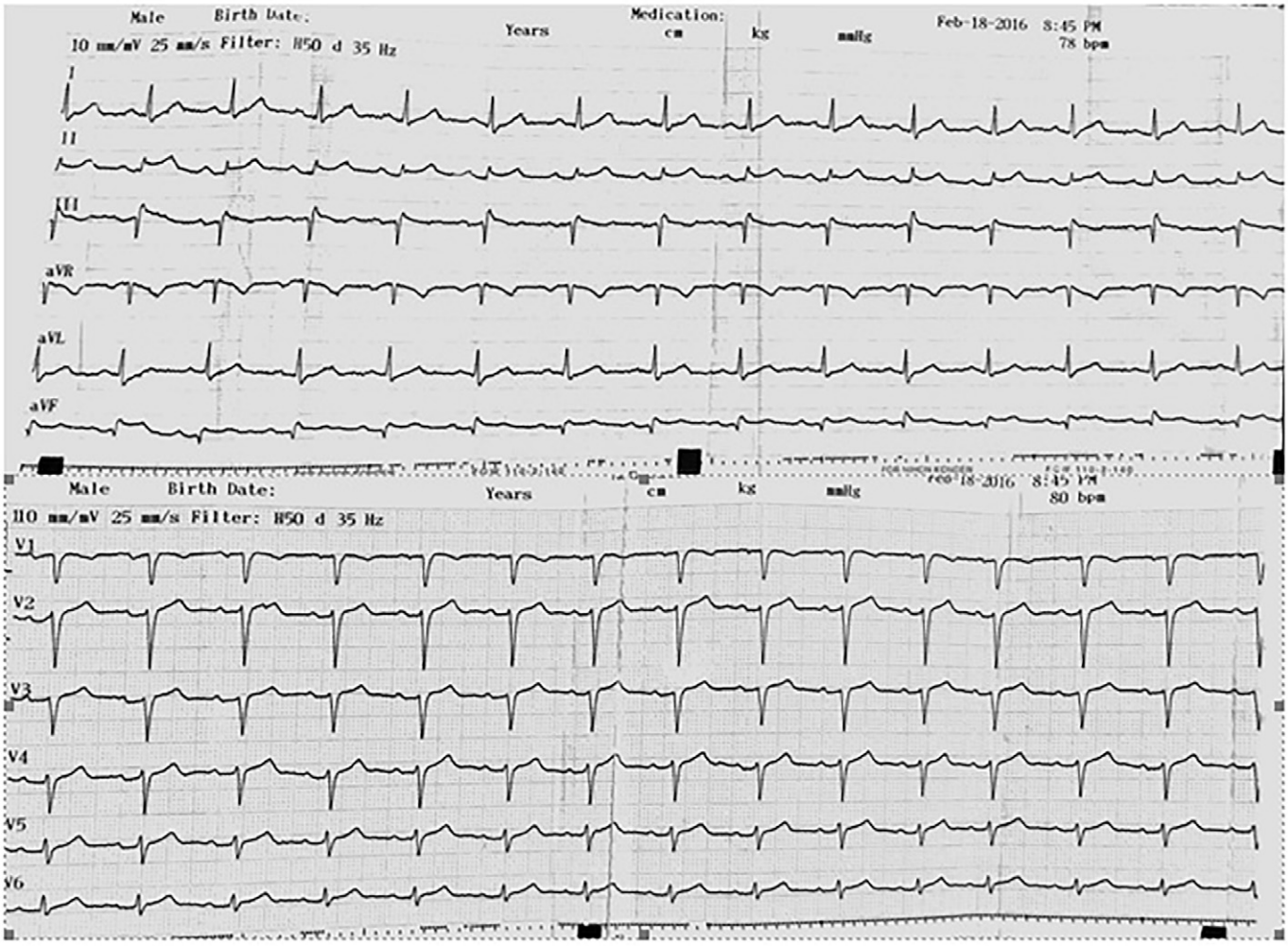


Fig. 1. The ECG shows ST Segment elevation in inferior leads without reciprocal changes.

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Fig. 2. Computed Tomography shows pulmonary embolism.

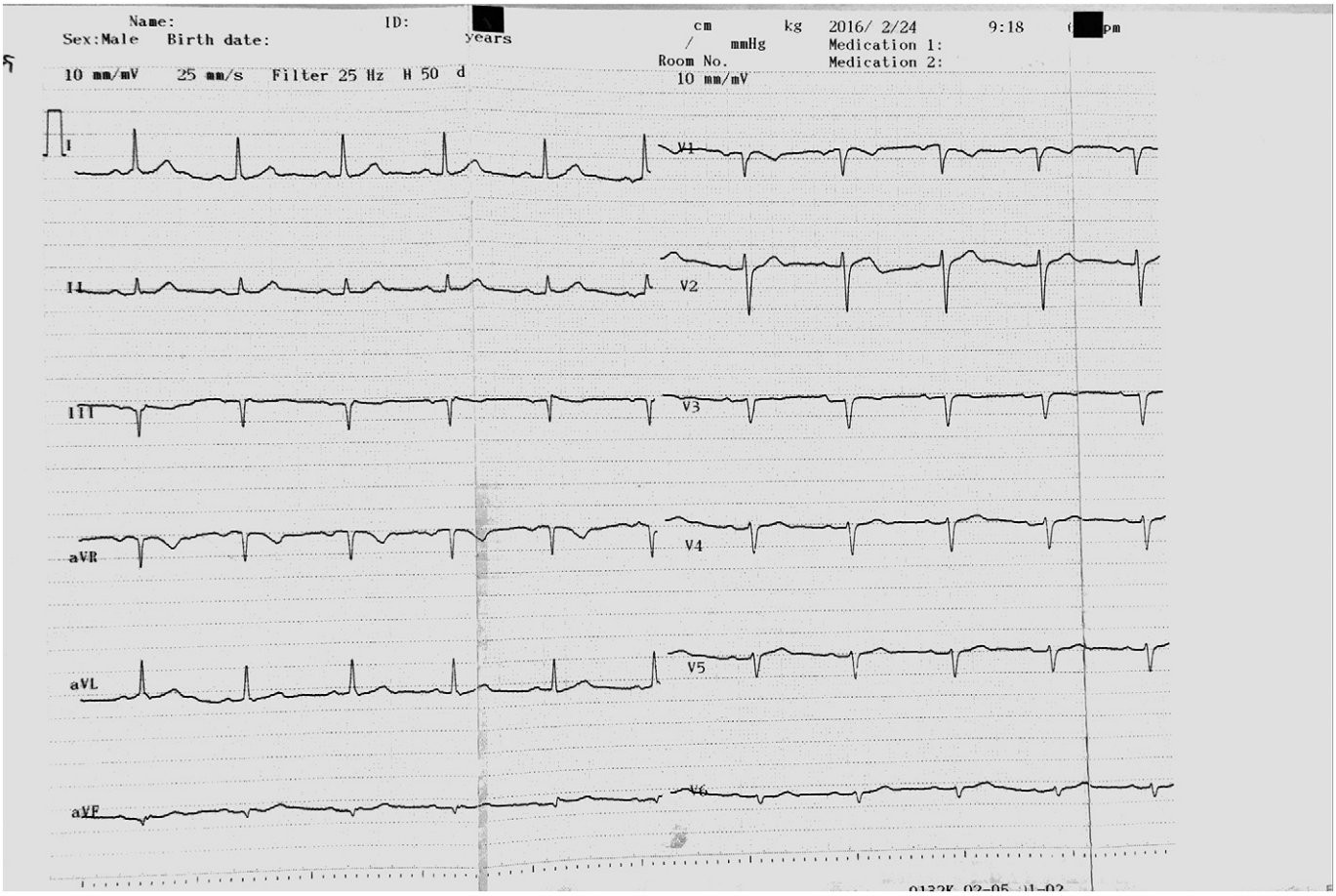


Fig. 3. The ECG shows resolution of ST segment elevation after anticoagulation treatment.

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Case report

Rare complication of diagnostic coronary angiography: Perforation☆

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Introduction

Diagnostic coronary angiography (CA) and percutaneous coronary interventions (PCI) are being performed in increasing numbers. However, several complications may also be seen in daily practice. Most are vascular access site complications. Acute myocardial infarction, coronary artery perforation, stroke, and death are rare complications, with an incidence of around 1%.[1] The coronary artery perforation is usually seen in PCI, and we present a case of coronary artery perforation of the three major branches of the right coronary artery.

Case

The patient, a 45-year-old female renal transplant recipient, presented with chest pain accompanied by shortness of breath, which is precipitated by exertion, and relieved by rest. She did not have any symptoms of congestive heart failure. Her family history was significant for hypertension and hyperlipidemia. She had been attending hemodialysis three-times a week for 8 years for chronic renal failure. She had been undergoing hemodialysis through a temporary double lumen subclavian catheter for 1 week due to the nonfunctioning dialysis fistula. The electrocardiogram (ECG) revealed sinus rhythm with T-wave inversion in leads II, III, and aVF. The transthoracic echocardiography (TTE) showed an ejection fraction of 55%, normal cardiac chamber dimensions, and impaired inferior wall motion, which might be associated with coronary fistulae. TTE also showed minimal tricuspid, and mitral regurgitation. The patient was advised to undergo CA, based on symptoms and findings consistent with the coronary artery disease. The CA was performed using the standard femoral approach with a 6-Fr diagnostic catheter. The CA revealed mild plaques in the left anterior descending artery and left circumflex artery (Figs. 1–2). In the left anterior oblique view, fistulas were seen between proximal right coronary artery (RCA) and right atrium. In right anterior oblique view, the small localized contrast staining was seen, which was consistent with the perforation of distal three branches of RCA (Figs. 3–5). The ECG was obtained due to the new-onset chest pain. It showed no change from previous ECG. The cardiac panel showed that CK-MB levels were minimally elevated, and troponin-I levels were normal. The TTE revealed a small pericardial effusion without tamponade. She was admitted to the coronary intensive care unit for follow-up monitorization. The pericardial effusion did not change during a 48-hour follow-up. The patient was advised to undergo percutaneous coil embolization of fistulas of RCA by our heart team. She was referred to the tertiary cardiovascular center.

Discussion

The complications of CA can be divided into three groups: major, minor, and contrast-associated. Minor complications include vascular access site bleeding, bruising, and infections. Major complications include ventricular fibrillation, acute myocardial infarction, stroke, aortic

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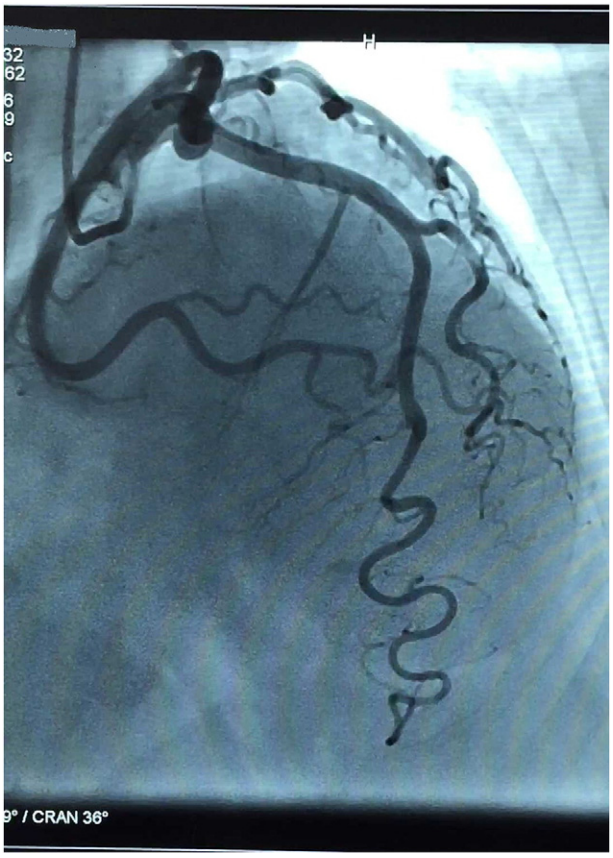


Fig. 1. Right Anterior Oblique views (RAO) with cranial angulation. Normal LAD.

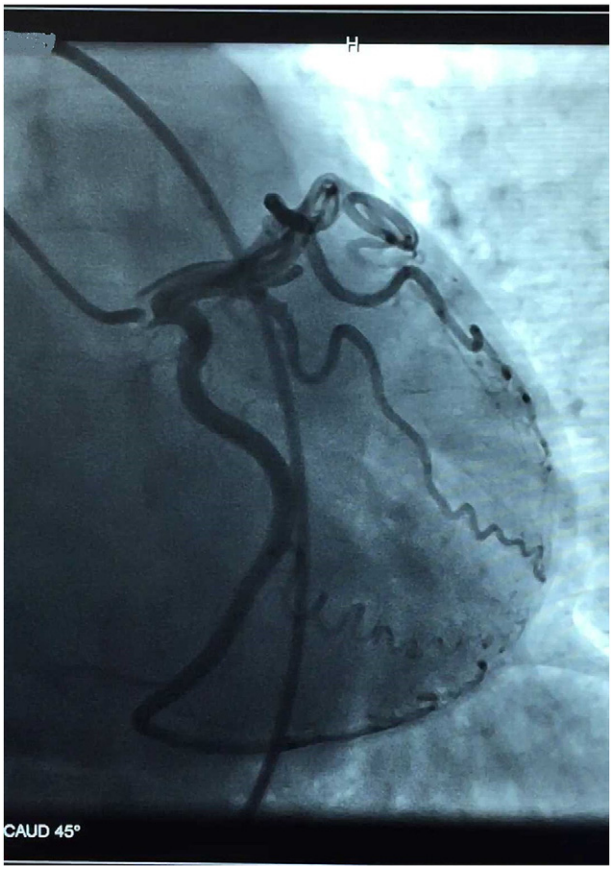


Fig. 2. Right Anterior Oblique views (RAO) with caudal angulation. Normal CX.

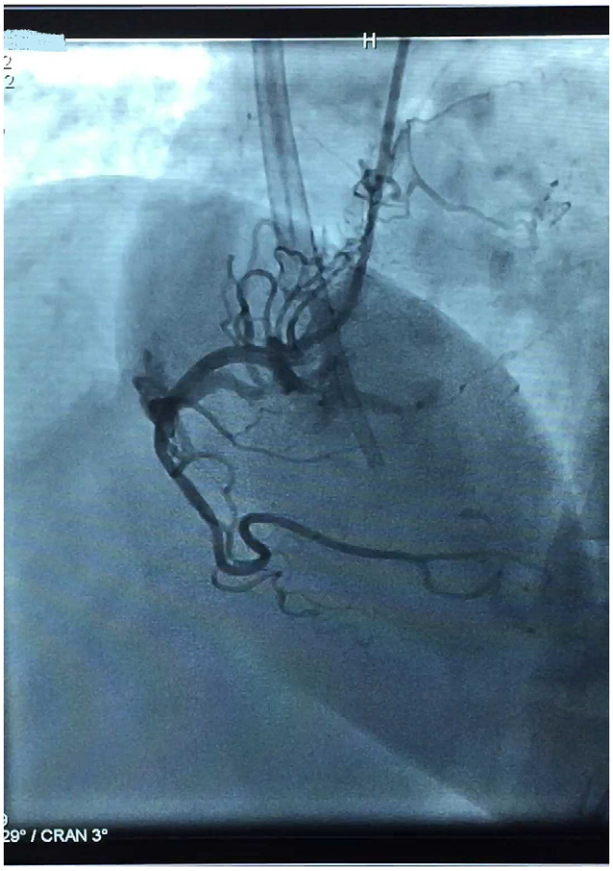


Fig. 3. Left Anterior Oblique views (LAO) with cranial angulation. Fistulas between proximal right coronary artery and right atrium.

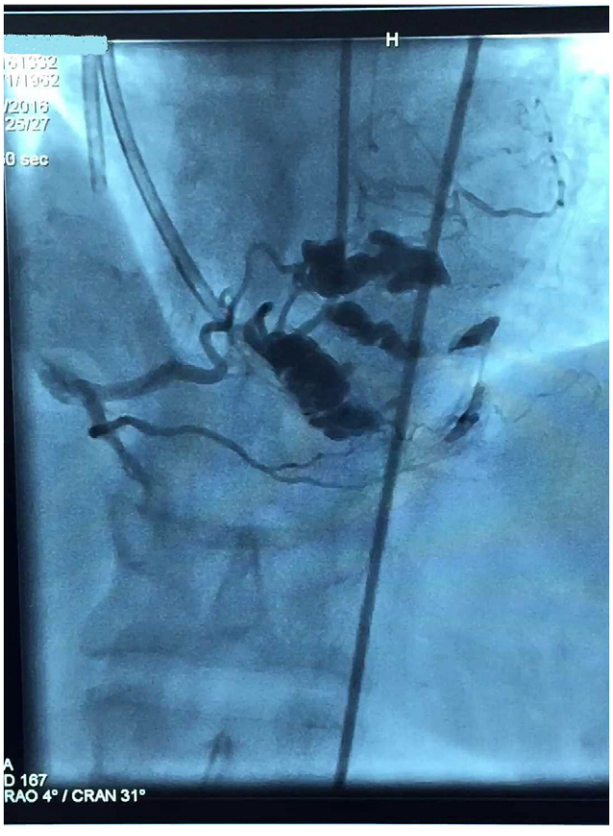


Fig. 4. Left Anterior Oblique views (LAO) with cranial angulation. Perforation of distal parts of proximal side branches of RCA.

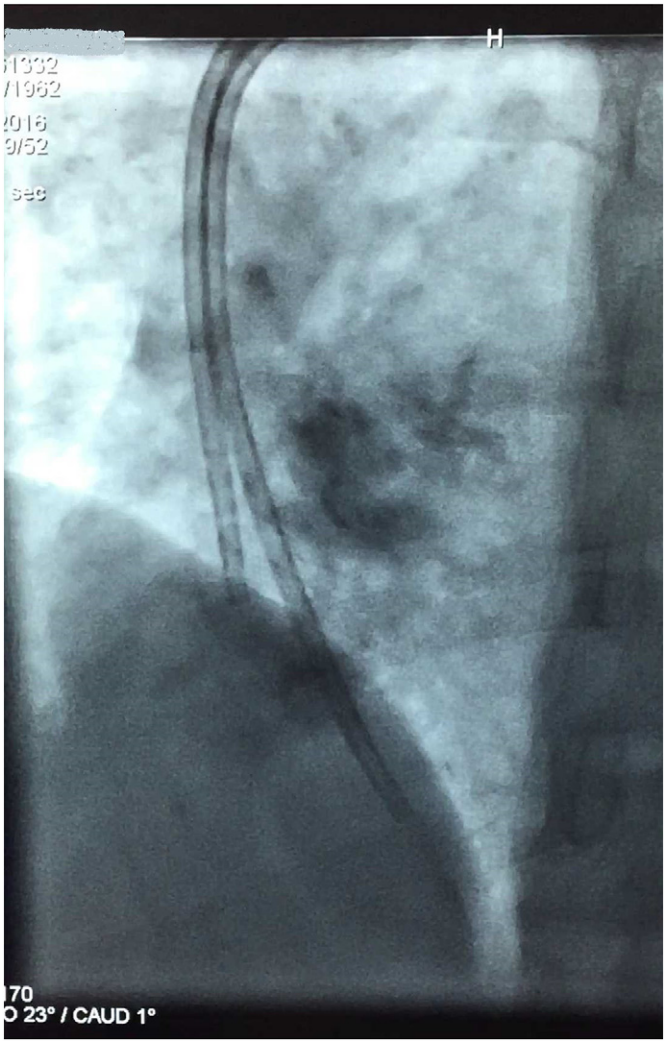


Fig. 5. Left Anterior Oblique views (LAO) with cranial angulation. The small localized contrast staining.

dissection, ventricular perforation, and coronary artery perforation.[2–3] The coronary artery perforation is a complication of PCI rather than CA. The incidence of coronary perforation ranges from 0.29% to 0.59%.[4–6] The risk factors for coronary perforation include age, gender (female), chronic kidney disease, and non-ST segment elevation myocardial infarction.[6] Ellis et al.[7] classified coronary perforation into three types: Type I (extraluminal crater), II (myocardial or pericardial blushing), and III (contrast streaming or cavity spilling). The coronary perforation in diagnostic CA is very rare and has been reported for the first time by Scöbel et al.[8] The selective cannulation of the conus branch of RCA was presented in their case report. The 5 F right Judkins (4 cm) diagnostic catheter was used to view RCA in their case. The patient was managed conservatively. The control CA showed no evidence of contrast extravasation. Timurkaynak et al.[9] presented another coronary perforation caused by deep cannulation of proximal branches of RCA. The 7 F right Judkins (4 cm) guiding catheter was used in their case. They did not find any pericardial effusion, and the patient was managed conservatively. Duyuler et al.[10] reported a perforation of the intermediate coronary artery due to deep cannulation with the diagnostic catheter. The patient developed acute pericarditis and was managed conservatively.

In our case, the type II perforation occurred due to the deep cannulation of proximal RCA. The patient was managed conservatively even though the lesion could be managed with covered stents. Since the amount of pericardial effusion did not increase. According to our current knowledge, the avoidance of deep cannulation of coronary artery and forceful contrast injection can prevent coronary artery perforation.

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Case report

Percutaneous closure of a postoperative residual atrial septal defect☆

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ABSTRACT

Atrial septal defect (ASD) is the most common adult congenital heart disease. The aim of the treatment of this disease is the surgical or percutaneous closure of the defect. Although surgical method has been used for a long time, primarily percutaneous closure is preferred currently in many centres for appropriately selected patients. Percutaneous closure can be used for the primary treatment of newly diagnosed patients and also for cases who are found to have residual defect after surgical closure. In the literature, however, only a limited number of patients who underwent percutaneous ASD closure were reported. In this report, we present our case who was successfully treated through percutaneous closure of postoperative residual ASD.
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Introduction

Atrial septal defect (ASD) is the most common adult congenital heart disease.[1] Open heart surgery has been performed for its treatment for many years.[2] Today, however, percutaneous closure of ASD is preferred for appropriate patients.[3] During follow-up, residual defects may develop in 7–8% of patients who have undergone surgery.[4] Medical follow-up is sufficient for hemodynamically insignificant residual defects while reintervention is needed for large defects. In the literature, there are only a limited number of patients reported to have developed postoperative residual defects and undergoing percutaneous closure.

Case report

A twenty-five year old male patient presented to our outpatient clinic with the complaint of shortness of breath and fatigue. His history revealed that he underwent patch repair for ASD and mitral ring repair due to secundum ASD and cleft mitral valve 6 months before. During his physical examination, fixed splitting on S2 was detected. His electrocardiography showed sinusal arrhythmia and right bundle block. Transthoracic echocardiography was performed for the patient; a 14-mm defect was detected on interatrial septum and the color Doppler showed left-to-right shunting). Value of Qp/Qs is 2.9. Mitral valve structure was normal (Fig. 1). Then transesophageal echocardiography was performed; which showed 14 * 12 * 12-mm residual defect on the interatrial septum. The morphological features and rims of the residual ASD were considered to be appropriate for percutaneous closure. Under deep sedation, percutaneous ASD closure was performed successfully for the patient with 17-mm Amplatzer occluder under transesophageal echocardiography. 34 mm balloon sizing catheter used in closure. After the procedure, the control echocardiography showed that the device occluded the defect completely and the color Doppler did not show any shunting (Fig. 2). There is no difference from routine percutaneous ASD closure. The patient was discharged on the following day without any problem.

Discussion

Secundum ASD is the most common congenital heart disease among adults.[2] Patients with ASD are usually asymptomatic at early stages. The volume loading of the right atrium and right ventricle increases in time and pulmonary arterial hypertension develops. The progression of pulmonary hypertension results in right ventricular insufficiency and Eisenmenger syndrome. Furthermore, arrhythmia and paradoxical embolism are common in these patients. Therefore, the defect should be closed as soon as the diagnosis is established if there is no contraindication.[5]

Surgical closure is the conventional method for the treatment of ASD. In this method, the defect is occluded through primary sutures or patch. Mortality and morbidity rates are low in this method compared to coronary artery surgery. However, it has some disadvantages such as sternotomy-thoracotomy requirement, postoperative pain, infection and permanent scar tissue.[5]

Today, the other defects except secundum ASD are treated surgically. However, transcatheter occlusion is preferred to surgical closure in patients with secundum ASD who are suitable for percutaneous closure. To determine the eligiblity of patients for percutaneous closure; the size of the defect, length of the total septum, sufficiency of the rims and

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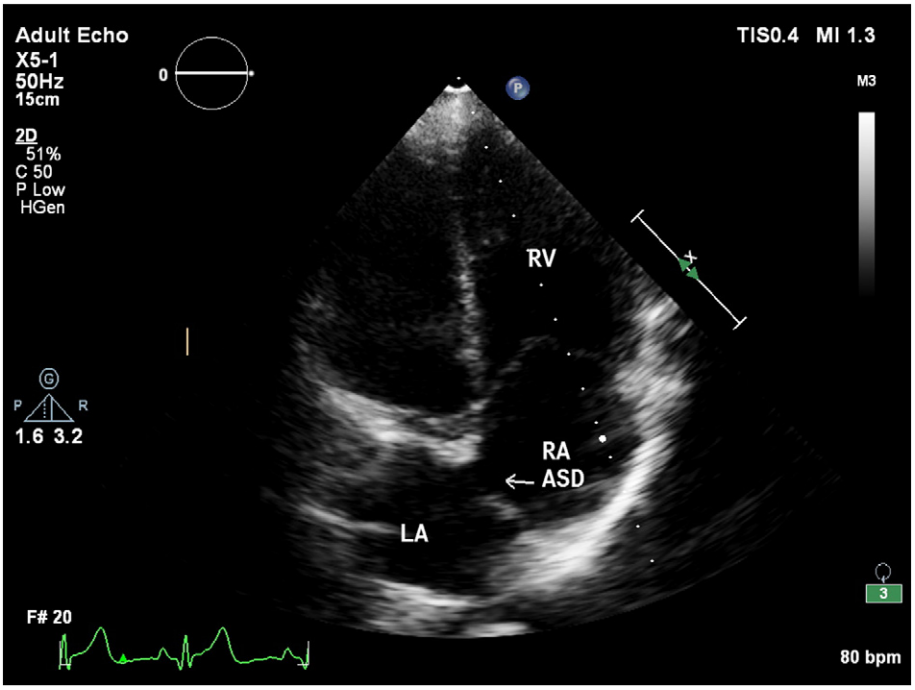


Fig. 1. Echocardiographic view of residual atrial septal defect.

comorbid congenital anomalies are assessed on transesophageal echocardiography.[6]

Percutaneous ASD closure is usually a safe procedure. However, major complications may rarely develop after transcatheter ASD occlusion, which includes perforation of the atrial wall, atrial arrhythmias and embolization of the device. The rate of such complications is less than 2%. The complication rates increase as the size of the device used gets bigger.[6]

Postoperative residual defects rarely develop in patients who are operated for secundum ASD. The development of the residual defect may be associated with the problems in the surgical technique or degeneration of the graft. The presence of pulmonary hypertension and arrhythmia is important for the choice of treatment in patients who develop postoperative residual ASD.[7] The hemodynamic significance of the shunt in patients who have residual ASD and their eligibility for percutaneous closure should be assessed. Like in our case, percutaneous

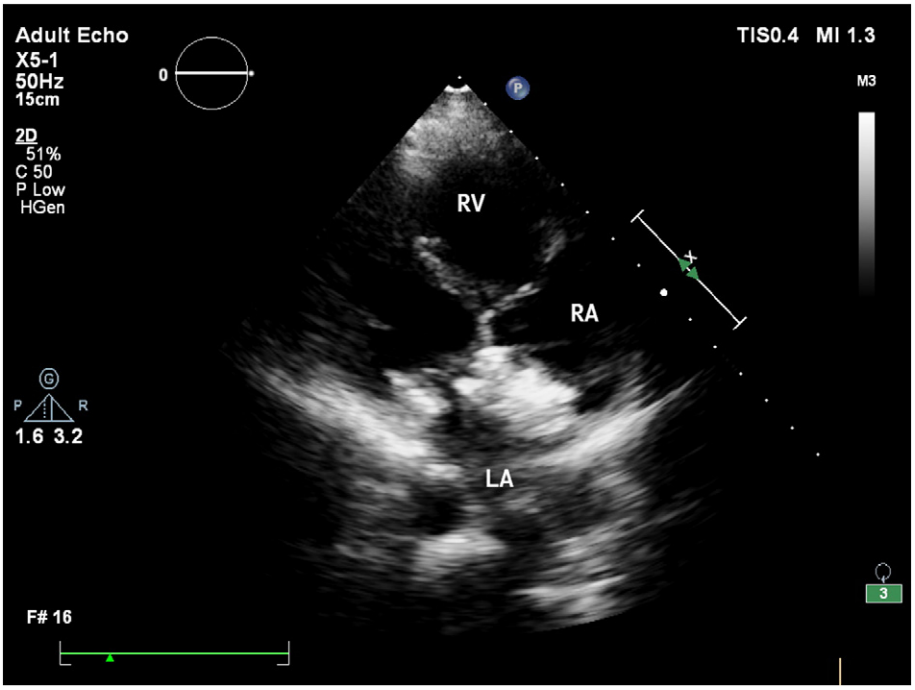


Fig. 2. Echocardiographic view of amplatzer occluder in intraatrial septum.

closure should be preferred to surgery for appropriate patients since it is a less invasive method.

Conclusion

The eligibility of the patients who develop postoperative residual ASD for percutaneous closure should be assessed taking into account the hemodynamic significance of the shunt and the morphological features of the defect. Percutaneous closure is a safe and effective treatment option if the morphological features are appropriate in these patients like newly diagnosed patients with secundum ASD.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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Case report

Circumferential dissection of ascending aorta☆

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Bentall's procedure

ABSTRACT

Acute type A aortic dissection is a surgical emergency. Unfortunately, the early outcome of surgical correction has not improved significantly over the past 20 years (Chiu and Miller, 2016). There is still conflict regarding operative extent and optimal conduct of the operation (Chiu and Miller, 2016). Stanford type A aortic dissection deforms the aortic root and causes aortic regurgitation. On rare occasion, there can be circumferential intimal disruption. Because of the critical, general and hemodynamic state of patients, surgery carries high risk. Two cases with circumferential Stanford type A aortic dissection were detected; Case I had interposition graft while Case II underwent Bentall's procedure.

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Introduction

Aortic Dissection is a life threatening condition. It requires immediate surgical intervention to prevent the grave consequences. Despite continuous advances in diagnostic methods, operative technique and perioperative care; acute Stanford type A aortic dissection remains a major unsolved cardiovascular surgical challenge, as illustrated by the most recent International Registry of Acute Aortic Dissection (IRAD) report showing an 18% operative mortality in a contemporary cohort [2010 – 2013].[2] We came across circumferential dissection of ascending aorta on two such occasions.

Case reports

Case I

A 54 years old patient was admitted to our hospital with acute chest pain, dyspnea (NYHA class iii) and vomiting for 2 days. On examination, there was significant difference in blood pressure of upper (90/50 mmHg) and lower limbs (120/70 mmHg); pulsation in both upper limbs had low volume while in both lower limbs it was high volume. Both carotid pulses appeared low volume with similar character. He had no neurological deficit and adequate urine output. Cardiac examination revealed normal heart sounds with grade 2/6 systolic and 3/6 diastolic murmur over left sternal border.

Investigations

Chest radiography revealed enlarged cardiac silhouette and pulmonary congestion. Twelve leads electrocardiography showed normal sinus rhythm with no ischemic changes and LV strain pattern. Biochemistry and hematological profiles were within normal limits. Two-dimensional Trans-thoracic echocardiography revealed moderate dilatation of aortic root and severe aortic regurgitation with normal LV dimension and LVEF of 55%. However, aortic valve leaflets were normal in morphology. There was a large flap seen in ascending aorta extending to arch and great vessels that was visualized on supra-sternal view. Epigastric view showed flap in descending aorta.

Surgery

Two arterial invasive monitoring lines were placed in right radial artery and left femoral artery. CPB was established with femoral and right atrial cannulation and the patient was cooled to 28 °C. Large clots were found all around the heart on opening the pericardium. All clots were evacuated. Ascending aorta was dilated tapering towards great vessels. A small area was cleared proximal to innominate artery for aortic cross clamp. Aorta was vertically opened at the most dilated part after cross clamping. Dissection had complete intimal disruption circumferentially with intact surrounding adventitia (Fig. 1). The dissection was well away from both coronary ostia (Fig. 1). Aortic valve was found to be morphologically normal (Fig. 1). We decided to preserve aortic valve and coronary ostia. We created proximal and distal cuff of aorta with Teflon felt taking care to suture adventitia and intima in a single layer. We replaced ascending aortic segment with number 24 knitted polyester graft. Patient was weaned from cardiopulmonary bypass. Radial and

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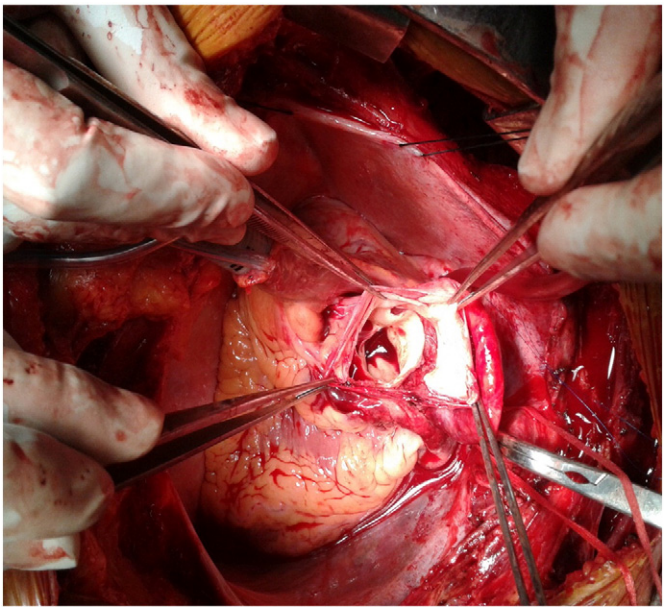


Fig. 1. Dissection is away from coronary ostia.

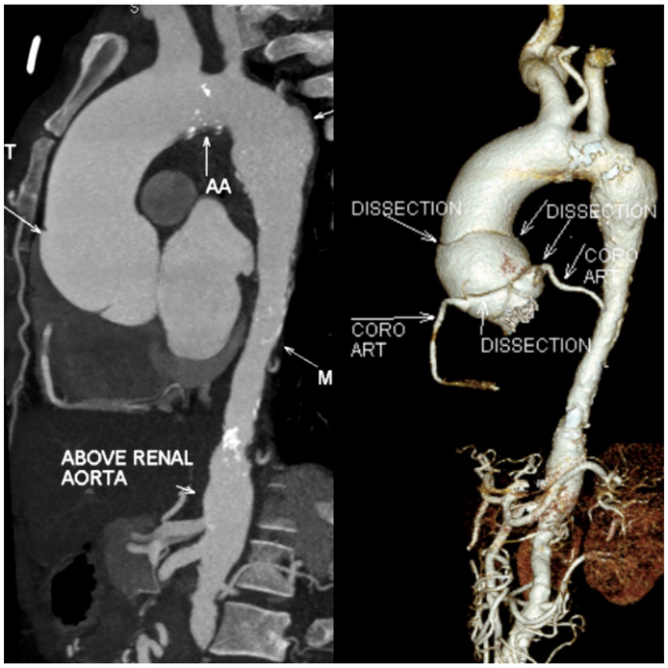


Fig. 2. Pre-Op CT s/o Stanford type A aortic dissection.

femoral pressures were the same as opposed to the preoperative condition. The chest was kept open in view of diffuse oozing. Next day, chest closure was done and the patient was extubated 72 h after surgery. The patient did not require any ionotropic support. The Patient was discharged on 10th post-operative day. Post-operative trans-thoracic echo showed no flap in aorta with trivial AR and normal LV Function.

Case II

A 35 years old female patient with no known medical disease presented with complains of acute onset of chest pain, palpitation and dyspnea for five days. She was admitted in intensive care unit where she was found to be hypertensive (150/90 mmHg) and tachycardia (110/min) and tachypnoic. She was awake, alert and exhibited no neurologic deficits. She had adequate urine output.

Investigations

Her electrocardiogram showed normal sinus rhythm without ST-T wave changes, and her troponin T was normal with normal renal function test. Chest radiography revealed dilated ascending aorta. Transthoracic echocardiography revealed moderate dilatation of aortic root, dissection flap in ascending aorta and severe aortic regurgitation with normal LV dimension and LVEF of 55%. A computed tomography (CT) scan with contrast was obtained. The CT scan revealed acute Stanford type A dissection arising from the aortic root and extending 2 cm proximal to the origin of brachiocephalic artery (Fig. 2). She was scheduled for emergency aortic reconstruction with aortic valve replacement.

Surgery

The patient underwent induction of general anesthesia with tracheal intubation. Two arterial invasive monitoring were placed; one each in right radial artery and left femoral artery. Transesophageal echocardiography (TOE) was subsequently performed, which confirmed the presence of a dissection flap in the ascending aorta with severe AR, the LV wall motion was normal and there was no hemopericardium. CPB was established with axillary and right atrial cannulation and the patient was cooled to 28 °C. The aortic root with ascending aorta was excised and Bentall's procedure was done. The patient had an uneventful recovery. Post-operative trans-thoracic echo show no flap in aorta (Confirmed on Post-Op CT scan Fig. 3) with normal LV Function.

Discussion

The evolution in the management of patients with acute Stanford type A aortic dissection has been one driven by inertia and oscillating whims, rather than progressive change. Throughout this process, the debate over the extent of the operation both proximally and distally has recurred.[1] The primary event was a tear in the aortic intima. Its incidence is estimated at 2.6–3.5/100,000 patient years.[3] Systemic hypertension is the most important predisposing factor for acute aortic dissection.[4,5] If untreated, acute dissection of the ascending aorta

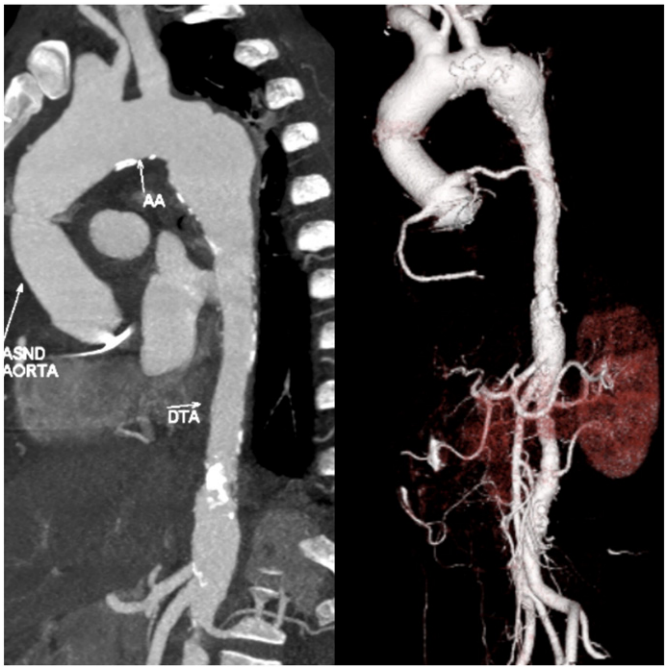


Fig. 3. Post-operative CT scan.

(Stanford type A) is associated with a mortality rate of 60% in first 24 h., 80% within 15 days and 90% within 3 months. Early diagnosis and surgical treatment improves survival preventing a number of sequelae (hemorrhagic shock, cardiac tamponade, aortic rupture or acute occlusion of major aortic branches and cardiogenic shock due to aortic regurgitation).[6,7] Successful surgery of circumferential dissection of ascending aorta is rarely reported.[8] However, we replaced ascending aorta only in first case and the root was spared due to the normal morphology of aortic valve while ascending aorta with aortic valved conduit been replaced in second. Continuity of the true lumen was maintained in both cases so that false remains will subsequently get thrombosed preventing extension of dissection in either direction. First patient is in follow up in NYHA class I with echocardiography suggestive of trivial AR, second patient had first follow up and was in NYHA class I. The rarity of the condition demands a long term follow up of surgical correction and a technical challenge to surgeons.

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Case report

Acute coronary syndrome after coronary subclavian steal syndrome treatment☆,☆☆

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ABSTRACT

Left subclavian artery stenosis can be a cause of recurrent angina after coronary artery bypass grafting in patients with a coronary steal. These patients may suffer claudication in the left arm and angina pectoris when using their left arm. The subclavian artery shows retrograde filling with use of a left internal mammary artery graft, which can cause coronary ischemia depending on blood flow. This is a report of a patient who presented with coronary subclavian steal syndrome and underwent percutaneous implantation of a stent in the left subclavian artery. However, the patient was readmitted with acute coronary syndrome 1 year later. An angiogram showed an unexpected decrease in anterograde flow in the left internal mammary artery graft after stenting the subclavian artery.
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Introduction

Left subclavian artery stenosis is a cause of recurrent angina after coronary artery bypass grafting (CABG) in patients with coronary steal syndrome. The patients can suffer claudication in the left arm and angina pectoris when using their left arm. The subclavian artery shows retrograde filling when a left internal mammary artery (LIMA) graft is used, which can cause coronary ischemia depending on blood flow. Herein, we report a case of coronary subclavian steal syndrome (CSSS) in a male patient who underwent percutaneous implantation of a stent into the left subclavian artery. However, the patient was re-admitted with acute coronary syndrome 1 year later. Angiography showed an unexpected decrease in anterograde flow in the LIMA graft after stenting the subclavian artery.

Recurrent angina can occur as a result of atherosclerotic progression of coronary lesions after CABG.[1] Left subclavian artery stenosis proximal to the LIMA graft is a rare and critical cause of angina following CABG. Therefore, CSSS should be considered in patients with claudication in the left arm and angina pectoris when using their left arm. Retrograde filling may result from circulation from the LIMA to the left subclavian artery depending on the coronary blood flow and may cause myocardial ischemia and thereby CSSS eventually.

Case report

A 57-year-old male smoker with hypertension and a history of coronary artery disease was admitted to our clinic. He had undergone CABG 7 years previously. He was being re-admitted for angina pectoris and a painful left arm when in use, particularly when carrying bags. The angina had progressively worsened, occurring during mild efforts and posing some limitations on his daily activities, but it resolved with rest. The patient was not suspected of having arm paresthesia. Physical examination findings were normal except a distinct difference in blood pressure (i.e., 20 mmHg) between the two arms. Electrocardiography, echocardiography, and laboratory studies were unremarkable on admission. Coronary angiography and bypass graft imaging revealed mild to moderate coronary stenosis in the mid-region of the left anterior descending (LAD) artery and retrograde filling of the LIMA bypass. Retrograde flow in the LIMA graft was revealed by angiography obtained from the LAD artery to the left subclavian and left vertebral artery (Fig. 1). The saphenous vein graft to the left circumflex artery and right coronary artery

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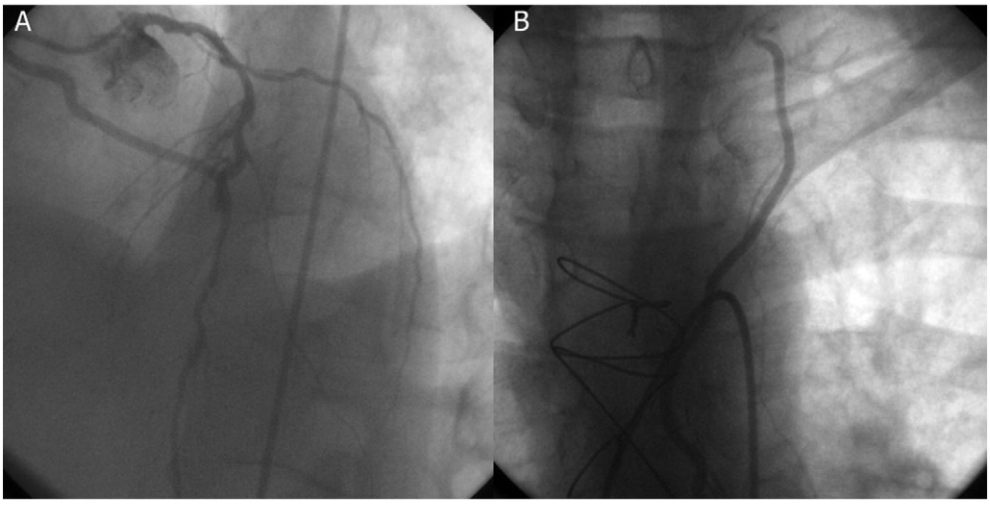


Fig. 1. Left coronary coronarography reveals stenosis in the left anterior descending (LAD) artery and retrograde filling of the left internal mammary artery (LIMA) graft (A). Retrograde flow from the LAD to left subclavian and left vertebral arteries (B).

was nearly normal. Significant stenosis of the left subclavian artery proximal to the origin of the LIMA was detected by angiography (Fig. 2). Based on these findings, we recommended percutaneous treatment. However, the patient rejected treatment. A percutaneous stent was implanted into the left subclavian artery 1 month later at another clinic. Clopidogrel was added to his treatment regimen. The patient had an uneventful course following stent implantation but was admitted to the emergency department 1 year later with unstable angina. Coronary angiography revealed reduced retrograde flow in the LIMA from the LAD artery (Fig. 3A). Anterograde flow in the LIMA was also reduced unexpectedly as a result of flow competition between the LAD artery and LIMA (Fig. 3B). Subclavian angiography revealed a patent subclavian artery stent. As a result, the patient was scheduled for follow-up visits under pharmacological therapy.

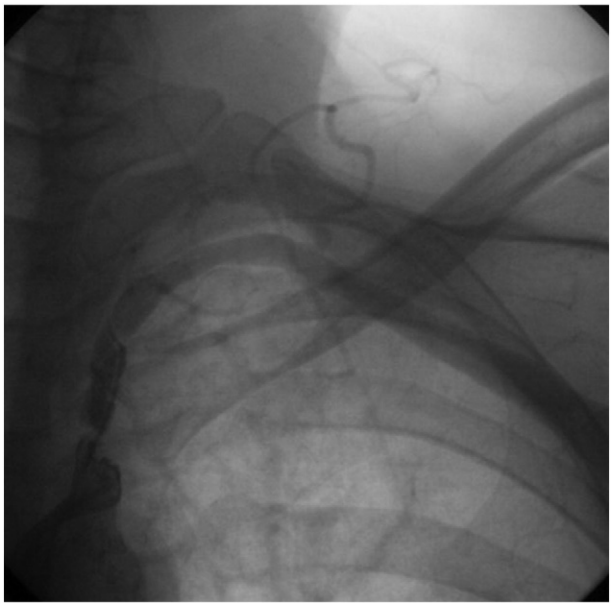


Fig. 2. Severe subclavian artery stenosis before stenting.

Discussion

CSSS is defined by a reversal of coronary flow in the LIMA caused by proximal subclavian artery stenosis.[2,3] The incidence of CSSS is about 0.2% to 0.68% of patients referred for CABG with LIMA. Its diagnosis is determined easily by angiography.[3] Symptoms have been reported to occur between 2 and 31 years following CABG surgery.[4] The clinical spectrum of CSSS is broad and includes stable angina, silent ischemia, or acute coronary syndrome[6,7].

Reversed flow in the subclavian artery is usually part of the cause of significant stenosis in the LAD artery. However, flow may rarely reach the subclavian artery because of the blood flow from the native vessel and the peripheral vascular resistance of the subclavian artery.[6] As a result, myocardial ischemia may develop upon use of the left arm as a result of a blood flow shift from the LAD artery to the subclavian artery. These patients are often admitted with stable angina. However, they may also present with acute coronary syndrome[7,8].

The treatment for CSSS is revascularization of the subclavian artery. The subclavian artery and anterograde LIMA flow may increase following a successful subclavian angioplasty.[3] However the restenosis rate for angioplasty is reported to be as high as 40.7% over 5 years in patients with CSSS.[5] Besides the anterograde flow in the LIMA graft may decrease following percutaneous treatment of the competing flow.

Acute coronary syndrome was diagnosed for multiple reasons in our case. One possible mechanism is plaque destabilization in the native coronary, subclavian artery, or coronary grafts with distal microembolization. Endothelial dysfunction or microvascular disease may also cause unstable angina.[2] Another mechanism is increased peripheral vascular resistance of the subclavian artery following angioplasty, as in our case.

In conclusion, CSSS can be treated successfully by subclavian angioplasty. However, these patients may encounter recurrent cardiac events due to diffuse atherosclerotic plaques. They may be admitted with acute coronary syndrome following percutaneous treatment. The culprit lesion was a challenge to identify in the present case.

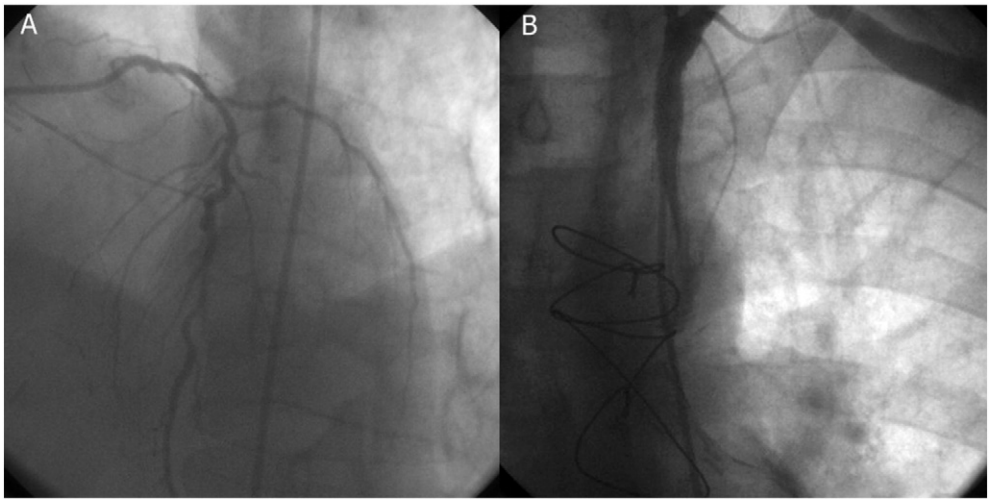


Fig. 3. Subclavian artery (A) and decreased anterograde blood flow in the left internal mammary artery (LIMA) after stenting the subclavian artery (B).

Although the subclavian stent was patent, the LIMA graft was recognized as the culprit lesion.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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Case report

Incidentally detected aorto-right atrial fistula in asymptomatic patient with operated aortic dissection☆

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ABSTRACT

There are many complications that can occur after aortic dissection surgery. Also these complications arise in unstable clinical conditions. Development of fistula between aorta and right atrium is rare. These patients often present with cardiogenic shock. Our purpose in presenting this case, unlike other cases in the literature, the patient was found to be completely asymptomatic and incidental rather an urgent clinic.

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Introduction

Aorta-right atrial fistulas are abnormal connections between the aorta and the right atrium. Fistulas can be congenital or passed as to cardiac and aortic surgery or may be secondary to infective endocarditis.[1–4]

Case report

A forty years male patient without any complaint admitted to our clinic for testing purpose. It was learned that he was operated for aortic dissection 3 years ago. Physical examination was unremarkable. Electrocardiogram was normal sinus rhythm. He was evaluated with transthoracic echocardiography during routine outpatient clinic follow up. Moderate aortic regurgitation and fistula development between aorta and right atrium were seen (Video 1). Transesophageal echocardiography was performed and fistula was clearly seen (Fig. 1, Video 2). The patient was asymptomatic and hemodynamically stable, thus, he was followed clinically without further interventions.

Discussion

Common complications occurring in the setting of acute aortic dissection include occlusion of the vessels originating from aorta, hemopericardium, pleural effusion and aortic regurgitation. A potential rare complication is the development of fistula between aorta and right atrium as a result of rupture of a hematoma in interatrial septum into right atrium.[5,6]

Development of a fistula is more common in patients who had a prior cardiac surgery or sinus valsalva aneurysm.[6] It is usually presents with cardiogenic shock.[3] Although computed tomography (CT) is the gold standard for diagnosis, lack of hemodynamic stability in these patients at diagnosis limits its use as a diagnostic tool. Therefore, transthoracic and transesophageal echocardiography are used for diagnosis.[7]

Fistulas between aorta and right atrium which are usually detected during follow up of patients with aortic dissection, cause left to right shunts and cardiogenic shocks. Therefore, rapid detection and surgical treatment are necessary.[8]

Conclusion

Development of fistula between aorta and right atrium is a rare complication after aortic dissection operation. These patients are usually present with cardiogenic shock, as maybe presented with asymptomatic. Careful echocardiographic diagnosis is vital in these cases. In asymptomatic patient, we should clinically monitor the patient for the development of heart failure symptoms. When these symptoms occur it will be life threatening. And urgent surgery is necessary.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ijcac.2016.12.002.

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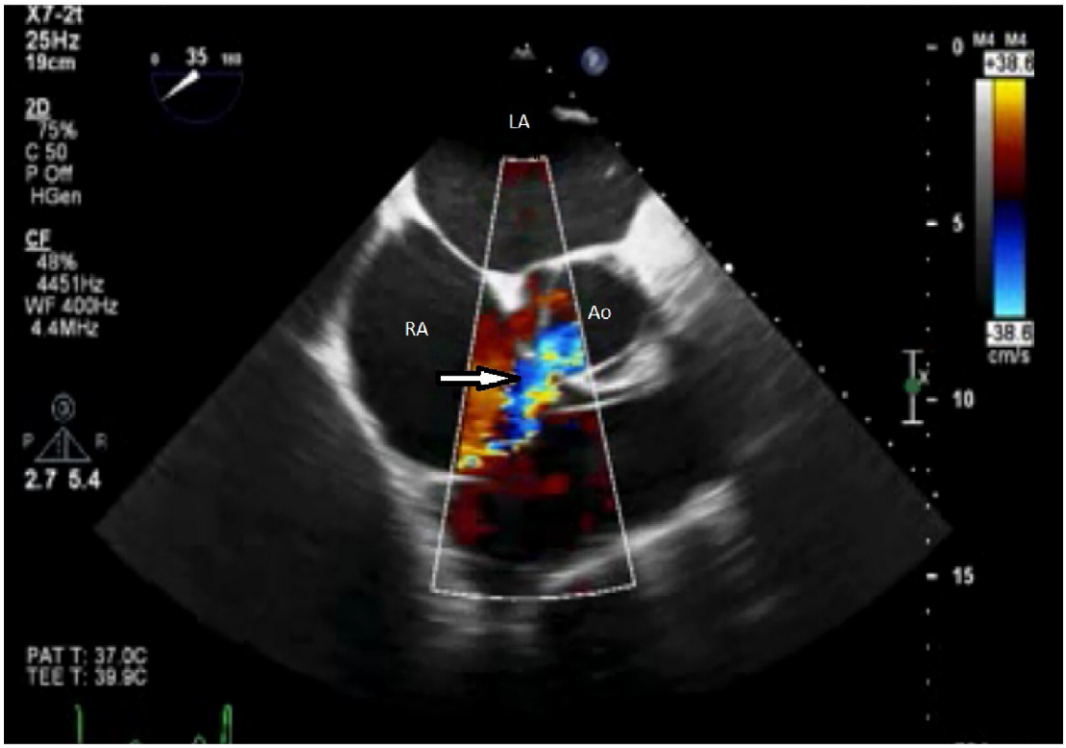


Fig. 1. A fistula was observed between aortic noncoronary cusp and right atrium in transesophageal echocardiography.

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