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Atypically mitral valve originated giant myxoma presenting with acute ST-segment elevation myocardial infarction and acute pulmonary edema

Akut ST elevasyonlu miyokard infarktüsü ve pulmoner ödem ile presente olan mitral kapakta atipik yerleşimli dev miksoma

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Abstract

Cardiac myxoma is a rare disease with an incidence of 0.0017-0.03%, and most frequently are seen between the third and sixth decades. The 65% of cases are female. They originate from left atrium in 75% of the cases, from right atrium in 18% and from ventricles in 4%. The most rarely seen cardiac origins are valvular tissues and respectively origin from tricuspid valve, mitral valve, pulmonary valve and aortic valve. The exact incidence of myxomas originating from the mitral valve is not clear. Clinical signs are classified in three groups such as systemic symptoms, symptoms related to thromboembolisms and symptoms related to intracardiac obstructions. Weakness, fever, weight loss, arthralgia are some of the systemic symptoms. Thromboembolisms are caused by either the tumoral tissue or the clot locating on the mass. Dyspnea, orthopnea, dizziness, syncope and pulmonary edema are examples of symptoms resulting from intracardiac obstructions, depending on the size, mobility and localization of the tumor. We aim to present a 77-year-old female presenting with dyspnea, angina pectoris and tachycardia and getting a diagnosis of a giant myxoma originating from the mitral annulus and posterior leaflet causing myocardial infarction with ST elevation, acute pulmonary edema, pulmonary hypertension, paroxysmal atrial fibrillation.

Keywords: Myxoma, Mitral valve, Acute coronary syndrome, Pulmonary edema

Öz

Kardiyak miksomalar %0,0017-0,03 insidansına sahip oldukça nadir olan bir hastalık olup en sık üçüncü ve altıncı dekadlar arasında görülür. Olguların %65'ini kadınlar oluşturur. Olguların %75'inde sol atriyumdan, %18'inde sağ atriyumdan, %4'ünde ise ventriküllerden köken alırlar. En nadir görüldükleri yer ise kapak dokuları olup sıklık sırası triküspit kapak, mitral kapak, pulmoner kapak ve aortik kapaktır. Kapak kökenli miksomaları gerçek insidansı belirsizdir. Klinik belirtileri sistemik semptomlar, emboliler ve intrakardiyak obstrüksiyonlardan kaynaklanır. Güçsüzlük, ateş, kilo kaybı, eklem ağrıları sistemik semptomlardan bazılarıdır. Tromboemboliler tümoral dokunun kendisinden kaynaklanabildiği gibi kitle üzerinde organize olmuş pıhtılardan da meydana gelebilmektedir. Dispne, ortopne, baş dönmesi, senkop ve pulmoner ödem intrakardiyak obstrüksiyonlardan kaynaklanan semptomlardır ve lokalizasyon, boyut ve hareketliliğe göre değişkenlik gösterebilir. Bu makalede dispne, göğüs ağrısı ve taşikardi ile prezente olan ve ST elevasyonlu miyokard enfarktüsü, akut pulmoner ödem, pulmoner hipertansiyon, paroksismal atriyal fibrilasyona yol açan mitral anuler ve posterior kapakçıktan köken alan dev miksoma tanısı alan 77 yaşında bir kadın hastayı sunmayı amaçladık.

Anahtar kelimeler: Miksoma, Mitral kapak, Akut koroner sendrom, Pulmoner ödem

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Introduction

The primary cardiac neoplasms are rarely seen in comparison to secondary cardiac neoplasms. The 80% of the primary cardiac neoplasms are benign and most of them are myxomas [1]. The remaining part of primary cardiac neoplasms is malignant, and often consists of angiosarcoma [2]. Besides, most of the cardiac malign neoplasms are formed by metastasis originating from pulmonary and are seen a hundred fold than the primary malign cardiac neoplasms [3]. Although the most of the primary cardiac neoplasms are benign, they may cause serious complications resulting with a remarkable increase in morbidity and mortality.

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Case presentation

A 77-year-old female patient applied to our hospital with unstable angina, dyspnea and tachycardia. In the telegraphy there were signs of acute pulmonary edema (Figure 1). In the lung examination bilateral basal crepitant rales were auscultated. ST segment elevation occurred in D2, D3, AVF and V6 derivations and reciprocal ST depression in V1-V3 derivations was observed, the rhythm was sinus (Figure 2). Troponin-I level was higher than 50000 pg/mL, and CKMB level was 101.7 U/L. The patient underwent emergent coronary angiographic intervention with a diagnosis of acute inferoposterior myocardial infarction. With percutaneous transluminal coronary angioplasty, the totally occluded middle circumflex artery lesion was extinguished (Figure 3). Following this process, atrial fibrillation occurred. In the medical history, the patient was complaining of palpitation attacks, which helped us in diagnosing paroxysmal atrial fibrillation. Hemodynamic findings were stable. Intravenous infusion of 1200 mg amiodarone was applied after the electrolyte replacement. Three hours later normal sinus rhythm was restored. On the transthoracic echocardiography a heterogeneous, smooth intracardiac mass with a size of 3.16x2.8 cm and originating from mitral annulus and posterior leaflet with a wide based pedicle. The mass was moving through the left atrium and left ventricle during the diastolic and systolic intervals (Figure 4). A third degree tricuspid regurgitation, normally sized tricuspid annulus, pulmonary arterial pressure of 90-95 mmHg, left atrial dimeter of 4.9 mm and an ejection fraction (EF) of 45% were other echocardiographic findings. Intravenous furosemide was applied due to pulmonary edema. The patient was observed in the intensive care unit and on the third day the patient underwent an elective open heart surgery.

Operational Technique

After median sternotomy bicaval venous cannulation of right atrial appendix and arterial cannulation of the ascending aorta were applied and cardiopulmonary bypass was started with mid-hypothermia (28°C). Cardiac arrest was provided with cold crystalloid cardioplegia and left atriotomy was applied. A smooth, jelly mass with the dimensions of 4x3.5 cm, originating from posterior mitral annulus and valve with a broad-based pedicle and prolapsing in to the left ventricle was observed (Figure 5). The mass was extracted with the pedicle without any harm on the valvular tissue. The remaining area was cauterized with 25W in order to annihilate possible residual myxoma cells. After the completion of left atriotomy closure, intraoperative transesophageal echocardiography was applied and reported as normal functional valvular tissue. The patient was transferred to the intensive care unit with dobutamin drug support with a dose of 5 mcg/kg/min.

In the early postoperative the transthoracic echocardiography, EF was 45-50%, PAB was 30 mmHg, and there was no valvular dysfunction. The patient was discharged on the postoperative seventh day.

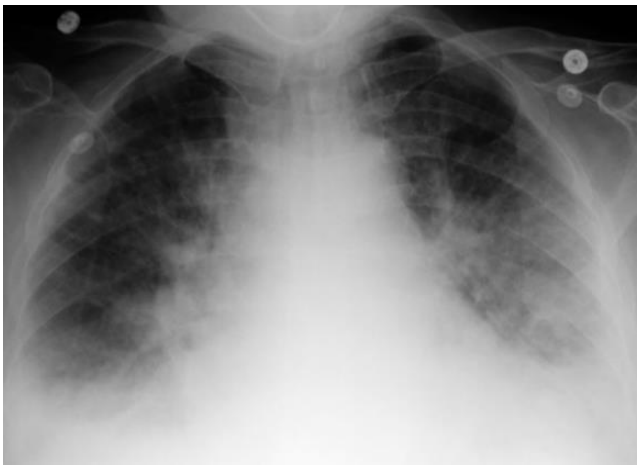

Figure 1: Posterior-anterior chest radiograph: Acute pulmonary edema

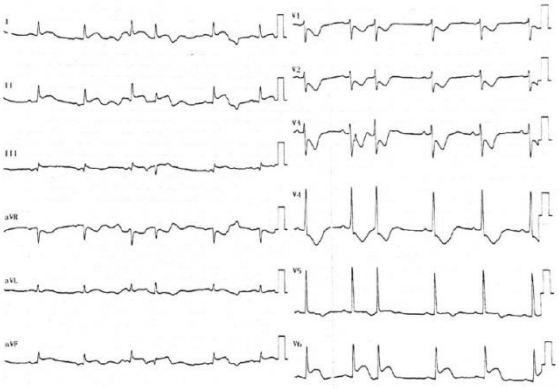

Figure 2: ECG of the patient, Acute Myocardial Infarction with ST-segment elevation in D1, D2, AVF, V6 derivations

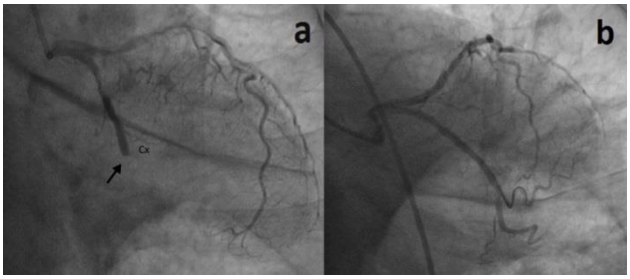

Figure 3: a. Angiographic image of acutely occluded Circumflex artery, b. Angiographic image of Circumflex artery after balloon angioplasty

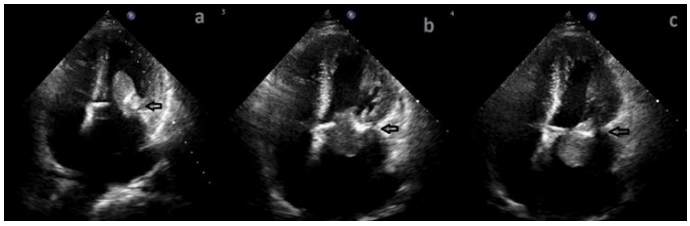

Figure 4: a. Transthoracic image of mobile giant myxoma in left ventricle, b. Myxoma moves through the mitral valve, c. Myxoma moves through left atrium

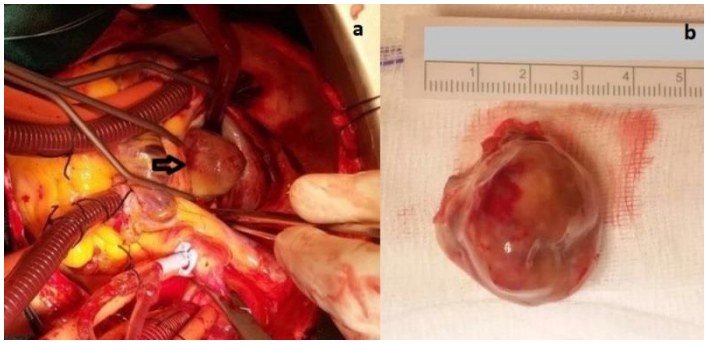

Figure 5: a. Intraoperative image of myxoma, b. Image of myxoma after resection

Discussion

Cardiac myxoma is a rare disease with an incidence of 0.0017-0.03%, and it is most frequently seen between the third and sixth decades. The 65% of cases are female [4]. They originate from left atrium in 75% of the cases, from right atrium in 18% and from ventricles in 4% [5]. The most rarely seen cardiac origins are valvular tissues and respectively origin from tricuspid valve, mitral valve, pulmonary valve and aortic valve [6]. The exact incidence of myxomas originating from the mitral valve is not clear.

Clinical signs are classified in three groups such as systemic symptoms, symptoms related to thromboembolisms and symptoms related to intracardiac obstructions. Weakness, fever, weight loss and arthralgia are some of the systemic symptoms. Anemia, high levels of CRP and erythrocyte sedimentation rate is examples for the laboratory findings.

Some of the symptoms of intracardiac obstructions are dyspnea, orthopnea, dizziness, syncope and pulmonary edema depending on the size, mobility and localization of the tumor [7]. There are two types of cardiac myxomas, polypoid and round-type. Polypoid myxomas are hemi-transparent and gelly-formed, and thus it accompanies a higher risk of embolization. On the other hand, round-type myxoma causes embolization less and the reason is mostly the cloth load on tumor in comparison with the polypoid myxomas' tumoral tissue embolism. Embolization rate is 45-60% for the left originated myxomas and 8-10% for the right originated myxomas, and mostly renal, cerebral and lower extremity arteries are affected [7,8].

Acute coronary syndrome (ACS) is a quite rare embolic complication of myxoma and caused mostly by the tumoral tissue or the cloth material due to the right angle between the aorta and coronary arteries [8]. Hence, in some cases the increased vascularization of myxoma and accompanying coronary artery anomalies may cause ACS, which is called coronary steal syndrome [9-11]. In the literature, it is possible to see only few cases reporting for myxomal coronary steal syndrome. Anyway, it is a significant point for a clinician to think about in a patient presenting with an ACS and myxoma.

Congestive heart failure prevalence among the myxoma cases is 60% but less seen in valvular myxomas in comparison to atrial ones. Because mobile valvular myxomas are diagnosed earlier than they become big enough to cause heart failure due to early embolic symptoms [12]. Myxomas originating from mitral valve are usually localized on the atrial surface and has an equal distribution on both anterior and posterior leaflets [12,13].

In this complicated case, we observed both acute and chronic complications of a giant myxoma as congestive heart failure, arrhythmia, acute pulmonary edema, pulmonary hypertension, tricuspid valve insufficiency and acute coronary syndrome caused by the thromboembolism of coronary arteries. Myxomas originating from valvular tissues are less seen than atrial ones and most of them are diagnosed before they reach giant dimensions due to becoming symptomatic earlier. In this case we observed a myxoma, which became symptomatic in late period and giant enough to cause chronic symptoms like pulmonary hypertension, congestive heart failure, arrhythmia [14,15].

Cardiac myxomas are the most frequently seen benign tumors of heart but sometimes they may have conclusions worse than the malign neoplasms depending on the localization, histology, dimensions and etc. With an early diagnosis and proper operational technique it is possible to cure the patient without any morbidity and mortality.

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