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Case report

A case of atypically located left atrial myxoma with concomitant acute myocardial infarction and severe pulmonary hypertension

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ABSTRACT

Myxomas are the most frequently seen benign cardiac tumor. They mostly originate from interatrial septum. They can lead variable signs and symptoms. Opposite to their benign structure, embolic and obstructive complications can be fatal. Myxomas can rarely lead acute myocardial infarction due to coronary embolism. In this article, we presented a left atrial myxoma case that originated from posterior mitral annulus with simultaneous acute inferoposterior myocardial infarction, severe pulmonary hypertension and dynamic mitral stenosis. The patient was managed with successful percutaneous transluminal coronary angioplasty of the left circumflex artery without stenting and surgical removal of the myxoma consecutively. Pulmonary hypertension dropped significantly in postoperative follow-up.

Introduction

Myxomas are the most frequent benign cardiac tumor but they are potentially hazardous. Seventy-five percent of myxomas are found in the left atrium (LA), mostly attached to interatrial septum (IAS). Mitral valve stenosis or systemic embolization are the most commonly seen clinical presentations of left atrial myxomas along with constitutional symptoms. Systemic embolism has been reported in 30%–50% of the case series.[1] A majority of the cases are sporadic but 7% of them have familial backgrounds. The left atrial myxomas have a significant potential for systemic embolization perioperatively to both peripheric and coronary vasculature.[2,3] Detection of this kind of pathology is very important in determining the treatment strategy since underlying pathology may lead to recurrence. Additionally, stenting with dual antiplatelet can increase bleeding risk perioperatively. In this article, we presented a case of atypically originated left atrial myxoma with simultaneous acute myocardial infarction and severe pulmonary hypertension. The patient was managed with successful percutaneous tranluminal angioplasty of the left circumflex artery (LCx) without stenting and surgical removal of the myxoma consecutively. In this article, we aimed to emphasize importance of preprocedural echocardiographic examination and the combined approach of percutaneous transluminal coronary angioplasty (PTCA) and surgery for the left atrial myxoma.

Case report

The patient, a 77-year-old woman, was admitted to the emergency room with clinical presentation of acute inferoposterior myocardial infarction with a squeezing chest pain of one-hour duration radiating to the shoulders. In the history, she had no relevant prior disease or any risk factor for cardiovascular disease and she did not describe any signs of heart failure or arrhythmias previously.

Blood pressure values were 130/70 mmHg, the pulse rate was 72 beats/min, the oxygen saturation was normal. Lung fields were clear of rales or ronchi except for a decrease in respiratory sound at the base of the right lung on auscultation. Cardiac examination revealed apical diastolic murmur of moderate intensity without opening snap at the left lateral decubitis position.

An initial ECG revealed sinus rhythm and premature atrial beats with significant ST segment elevation in leads II, III and aVF consistent with transmural ischemia.

A transthoracic echocardiographic examination showed the presence of an echogenic, mobile mass stemming from the corner of the lower IAS and the LA and protruding toward the posterior mitral leaflet and orifice, compatible with myxoma. The mass, 36 × 28 mm in size, was prolapsing toward the left ventricle and producing mitral gradient of 12 mmHg (Fig. 1). Along with these findings, there were inferoposterior wall motion hypokinesia, the left ventricule ejection fraction (LVEF) was 48%, moderate to severe tricuspid regurgitation, the estimated pulmonary artery systolic pressure (PASP) was

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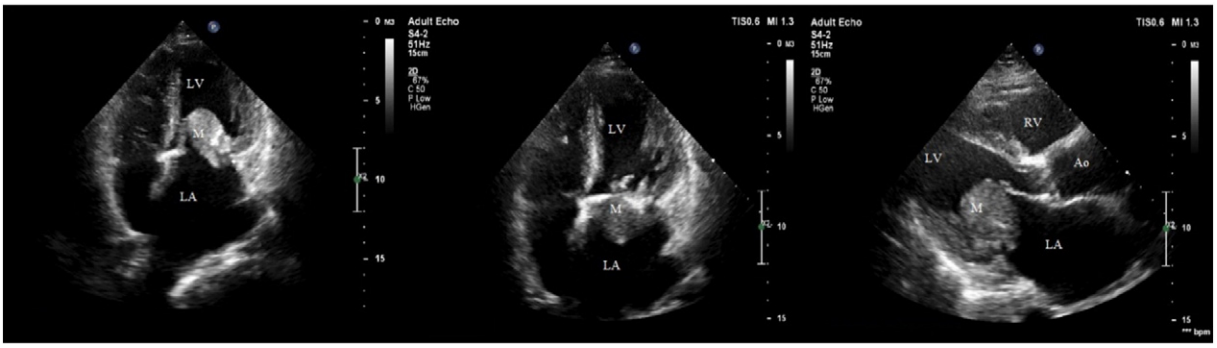

Fig. 1. Echocardiographic views of the myxoma.

90 mmHg and the LA diameter was 48 mm. Following prompt antiplatelet and anticoagulant treatment, she was transferred to the coronary angiography laboratory immediately. She had thrombotic total occlusion at the distal segment of the LCx along with non-significant, stable, atherosclerotic plaques at the left anterior descending and the right coronary arteries. Successful PTCA was performed without stenting for the LCx 30 min after her admission. Three days later, she underwent operation. A tumoral lesion of 45 × 38 × 46 mm in size with stalk stemming from posterior mitral annulus was excised via left atriotomy (Fig. 2). Under a light microscope, the tumor was composed of elongated, fusiform, stellate or polygonal cells with round to ovoid nuclei immersed in a myxoid stroma. No cytological atypia was present. Immunohistochemically, the tumor cells showed positivity for vimentin, CD34, factor VIII, calretinin and S100. They were negative for pancytokeratin and CD68. Histochemically, the myxoid stroma was positive for Alcain blue and PAS. In addition, focal calcification and excessive hemorrhage were detected in the tumor, mostly in the central area. Microscopic features were diagnosed as myxoma (Fig. 3). Postoperative ECG recordings were free of ST elevations on inferior leads. Additionally, postoperative first day echocardiography revealed significant decrease in PASP to 30 mmHg without any transmitral gradient and with no change in dimensions of the LV and the LA and mild mitral regurgitation. In the following days till discharge, the echocardiographic findings were similar to the previously described ones. The patient was discharged without complication. Postoperative 30th day echocardiographic examination showed mild mitral regurgitation and mild to moderate tricuspit regurgitation with estimated PASP of 40 mmHg with the LVEF of 59%.

Discussion

Myxoma is the most common benign cardiac tumor. Left atrial myxomas mostly present with changing levels of dyspnea imitating mitral stenosis and/or systemic embolization occurring in 30% to 50% of cases.[1] The incidence of coronary embolization is 0.06%.[4] Here, we presented a case of myxoma that originated from the posterior mitral annulus with concomitant acute inferoposterior myocardial infarction.

Atrial myxomas mostly stem from the fossa ovalis portion of the interatrial septum. The next most common location for myxomas is the posterior wall of the left atrium. However, valvular involvement of myxomas is more rare.[2] To the best of our knowledge, annular involvement has not been reported. In our case, the myxoma originated from the posterior mitral annulus with a pedicle. As in our case, females are more prone to have myxomas compared to males with a peak incidence at the third and sixth decades. Additionally, 67% of the subjects develop signs and symptoms of mitral stenosis in their life course;[5] however, our case was asymptomatic previously. It may be due to the development of severe pulmonary hypertension in the long term leading to reduced blood flow through the pulmonary artery and low cardiac output.

Compared to computed tomography and magnetic resonance imaging, transthoracic echocardiography is the method of choice in the emergency setting. It gives functional and anatomical data related to the myxoma. Myxomas present their clinical outcomes in accordance with location, size and mobility. Thus, symptomatology can vary respectively. Our case was asymptomatic with advanced age till the admission with acute myocardial infarction. As known, the incidence of coronary embolization is very rare.[4] It may be explained with the perpendicular alignment of the coronary ostia with respect to the aortic blood flow and protection of coronary ostia by the opening aortic valve leaflets. Approximately half of the cases involve the RCA. There is a concrete explanation for this tendency. There have been reports indicating that papillar or villous myxomas are more brittle and embolise more easily than myxomas with a smooth surface, it is not a must for embolization. Absence of previous coronary artery and risk factors for cardiovascular disease, absence of significant atherosclerotic coronary lesions, obtaining TIMI 3 flow just after guide wire passage and sustaining it

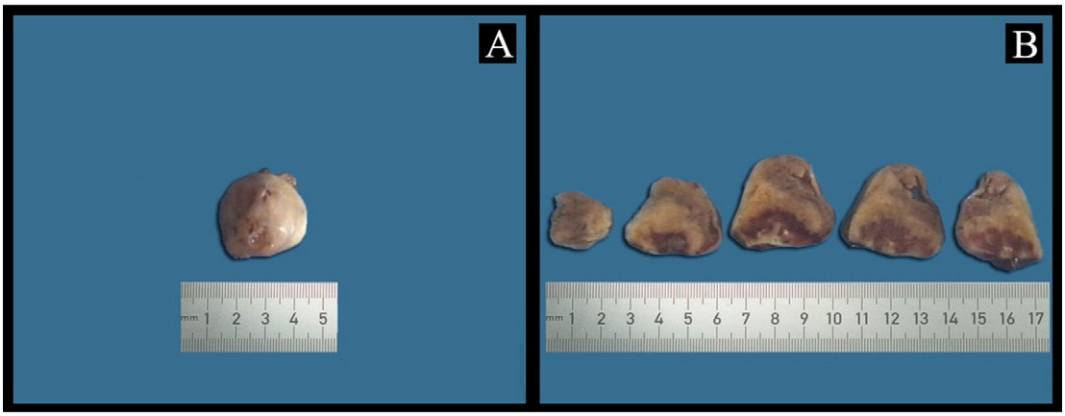

Fig. 2. Gross photographs of the lesion. (A) The rough surface of the lesion with a thin capsule. (B) The cut surfaces of the lesion containing hemorrhagic areas.