Case Report

Left Atrial Myxoma: A Rare Nonatherosclerotic Cause of Acute Myocardial Infarction

Rey Francisco Arcenas¹ and Mir Ishtiaque Ali²

¹ Third Year Resident Department of Internal Medicine, Indiana University Health Ball Memorial Hospital, Muncie, IN 47303, USA
² Indiana University Health Ball Memorial Hospital, Muncie, IN 47303, USA

Correspondence should be addressed to Rey Francisco Arcenas; reyfarcenas@yahoo.com

Received 30 April 2013; Accepted 5 June 2013

Myocardial infarction from coronary artery embolism is a very rare but potentially lethal sequel of left atrial myxoma. Most atrial myxomas causing myocardial infarction are diagnosed retrospectively after a 2D echocardiogram is performed for assessment of myocardial function after a myocardial infarction. We present a relatively healthy 53-year-old male with anterolateral wall myocardial infarction and 100% occlusion at the proximal part of the obtuse marginal branch of the circumflex coronary artery that was subsequently reperfused. A 2D echocardiogram performed two days later revealed a left atrial mass, which was successfully resected and proven to be a myxoma. No recurrence of the tumor was seen on follow-up after four months. An automatic implantable cardioverter defibrillator was placed for residual ischemic cardiomyopathy with clinical improvement.

1. Introduction

Primary cardiac neoplasms are rare, with an incidence of less than 0.2% in unselected patients at autopsy [1]. The majority of these tumors are benign myxomas located in the left atrium (80%) [2]. Rare coronary artery embolization from a cardiac myxoma usually involves either the right coronary artery (RCA) or the left anterior descending (LAD) coronary artery [3]. We report a case of a patient with 100% occlusion of the left circumflex (LCX) artery from a left atrial myxoma presenting as anterolateral wall myocardial infarction.

2. Case Report

A 53-year-old male with no significant past medical history presented with severe chest pain after exertion, associated with diaphoresis, dyspnea, cough, and three episodes of syncope. He denied smoking, use of alcohol, and illicit substances. He has no family history of coronary artery disease. On admission, he was in mild distress with blood pressure of 115/70 mmHg, heart rate of 100 beats per minute, respiratory rate of 20 breaths per minute, and temperature of 97.2°F. Body mass index was 30 kg/m². There was no jugular venous distention. He had bibasilar rales on lung exam. Cardiac examination revealed a normal heart rate, regular rhythm with no clicks, gallops, or murmur. The rest of the physical examination was normal.

The 12-lead electrocardiogram showed ST-segment elevation in the anterolateral leads with reciprocal changes in the inferior leads. Chest radiograph was normal. Initial set of cardiac enzymes were normal as he presented within 30 minutes from onset of chest pain. Cardiac catheterization revealed no evidence of coronary artery disease in the RCA, left main coronary artery, nor the LAD artery. However, the proximal obtuse marginal branch of the left circumflex (LCX) artery was noted to have a complete occlusion (Figure 1).

During percutaneous intervention, the occluded segment was opened with difficulty due to the significant amount of thrombus, employing multiple balloons, and aspiration with an Export catheter. After placement of two bare-metal stents, TIMI grade 3 (normal) flow with limited myocardial blush was observed (Figure 2). Left ventricular ejection fraction (LVEF) was approximately 30% measured by left ventriculography.

A transthoracic 2D echocardiogram performed two days after the intervention revealed a very large irregular mobile...
mass attached to the superior atrial septum freely prolapsing to the left ventricle (Figures 3 and 4). Severe hypokinesis of the apical, lateral, and midanterolateral walls were noted. LVEF is approximately 15–20%.

In the succeeding days, the patient developed various complications including cardiogenic shock that needed intra-aortic balloon pump placement, pneumonia, and an episode of ventricular tachycardia. These led to a delay of the definitive surgical intervention. Repeat coronary angiography showed patent stents with TIMI grade flow of 2 to 3 and limited myocardial blush. He subsequently underwent successful resection of the left atrial mass twelve days after his presentation. Histopathology confirmed the mass as an atrial myxoma.

Four months later, he was readmitted due to ventricular tachycardia; symptoms included mild dyspnea on ordinary activity. Repeat 2D echocardiogram revealed no recurrence of the myxoma. LVEF remained at 20% and still with significant anterior wall hypokinesia. An automatic implantable cardioverter defibrillator was subsequently placed. The patient has remained well since that time.

3. Discussion

From our review of the literature, we found two reported cases of combined left circumflex (LCX) artery and left anterior descending (LAD) artery embolism from an atrial myxoma causing myocardial infarction (MI) [4, 5], one case affecting both LCX and diagonal branches of the LAD artery [6] and only three cases affecting the LCX artery alone [7–9]. Embolization from an atrial myxoma is frequently seen clinically, occurring in 32% of cases as the presenting symptom in one series [10]. However, the incidence of coronary embolization from atrial myxoma is only 0.06% [11]. Possible explanations for this very low incidence include the perpendicular disposition of the coronary ostia in relation to the aortic blood flow and the protective effect of the aortic valve leaflets during systole [12].

Coronary artery embolism from a myxoma with resultant myocardial infarction is exceedingly rare [13–15]. Its true incidence is probably underestimated from the lack of systematic echocardiographic evaluation of patients after MI. It occurs over a wide age range, from 9 years [16] to 68 years of age [5, 17–19].
The lesion in the obtuse marginal branch in our patient cannot account for the electrocardiographic changes of anterolateral MI. The most likely explanation is that the emboli may have lodged temporarily in the left main coronary artery obstructing both the LAD and LCX arteries before it migrated down. It is also possible that he had a concomitant LAD embolization that broke off into smaller fragments or spontaneously lysed, accounting for a patent LAD artery on angiography [13, 20–22].

Several other characteristics of a myxoma are described in the literature. It is well documented that tumor emboli have malignant potential [6, 23–25], with the ability to persist [9], invade, and even grow inside the coronary artery. Conversely, there are cases of left atrial myxoma presenting as acute MI but with normal coronary arteries. Braun et al. [26] noted that a normal coronary artery despite a clinical diagnosis of acute embolic MI is still possible due to the myxomatous histology of the tumor, allowing it to embolize and resolve spontaneously. This makes the timing of symptom onset and the actual presentation critical for accurate diagnosis. Isobe et al. [27] cited the ability of myxomas to secrete interleukins 6 and 8 that may cause systemic manifestations like fever and cachexia. Our patient did not have any systemic manifestation prior to presentation.

The diagnosis of atrial myxoma causing MI is seldom established prior to cardiac catheterization, with rare exceptions [10]. Most cases have unrevealing cardiac examination findings which would prompt an echocardiogram. In other cases, a loud S1, a short presystolic murmur, and an apical middiastolic sound prompted echocardiographic evaluation [11, 28]. The diastolic sound is produced as the tumor strikes the ventricular wall and is characteristically intermittent [29]. The myxoma may obstruct the mitral valve and cause syncope, as in this patient. Rarely, neovascularization of the tumor mass may give a clue to its presence during cardiac catheterization [13].

Most atrial myxomas associated with myocardial infarction are discovered incidentally by 2D echocardiogram done to assess myocardial function after an event. Early echocardiography in all patients with a first episode of pulmonary edema [30] or acute coronary syndrome, especially in a relatively young patient with few risk factors, will expedite the diagnoses in most if not all cases. It is important to diagnose this condition because surgical intervention is the only means to improve the prognosis [18].

Additional concerns with an undiagnosed left atrial myxoma causing MI are the risk of extensive hemorrhagic MI with tamponade [10] and tumor fragmentation with further systemic embolization [28] when thrombolysis is performed.

Almost all cases previously reported had relatively good outcome after surgical intervention. However, some cases have complicated courses before the surgery, such as what happened to our patient. Tomita et al. [10] reported a case of a patient who died from multiorgan failure despite the use of intra-aortic balloon pump, due to extensive hemorrhagic myocardial infarction from a myxoma. Surgical resection is usually sufficient and curative with 0–3% operative mortality rate [1]. Resection of the tumor with percutaneous coronary intervention or coronary artery bypass surgery is needed when there is subtotal or total occlusion of a coronary artery [31]. And because there is a 2–5% recurrence rate for myxomas, [1] follow-up with 2D echocardiography is necessary.

4. Conclusion

Myocardial infarction from embolization by an atrial myxoma, although very rare, highlights the need to consider myxoma as one of the rare nonatherosclerotic causes of MI. This condition is lethal if not diagnosed and treated in a timely manner. 2D echocardiography after a myocardial infarction is the most efficient way to diagnose atrial myxoma even among those without symptoms. Surgical resection of the myxoma with percutaneous coronary intervention or coronary artery bypass grafting for patients with significant coronary occlusion is essential to improve the prognosis of the patient.

References


