A case report of left atrial myxoma-induced acute myocardial infarction and successive stroke

Qiushuang Wang, MD[a, *], Feifei Yang, MD[a], Fei Zhu, BD[a], Cunshan Yao, MD[b]

Abstract

Rationale: Left atrial myxoma is a common primary cardiac tumor, however, due to poor image quality or atypical myxoma images, it is often misdiagnosed by echocardiography. A case of left atrial myxoma being misdiagnosed as a thrombus, which successively caused acute myocardial infarction (AMI) and stroke, is very rare. Contrast-enhanced echocardiography can play an important role in definitive diagnosis.

Patient concerns: A 44-year-old woman was diagnosed AMI because of chest pain with no significant stenosis in the coronary arteries. One month later, the patient was suddenly found unconscious, magnetic resonance imaging (MRI) showed acute multiple cerebral infarctions in the left cerebral hemisphere.

Diagnoses: Left atrial myxoma, acute myocardial infarction, and stroke.

Interventions: The patient was given a cardiac surgery for tumor resection, the mass was surgically removed and histopathologic findings showed myxoma.

Outcomes: After several weeks of rehabilitation, the patient was able to resume daily activities without chest discomfort or dyspnea. One year later, echocardiography showed no recurrence of left atrial myxoma. The patient generally was in good condition.

Lessons: Although myxoma is mostly benign, this patient occurred AMI and stroke because of misdiagnosis. Comprehensive assessments should be performed with multiple imaging methods for cardiac masses. If necessary, contrast-enhanced echocardiography should be used to clarify, so as not to delay the timing of surgery and bring potential risk of death to patients.

Abbreviations: AMI = acute myocardial infarction, CAG = coronary angiography, CHD = coronary heart disease, LCX = left circumflex artery, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging, TEE = transesophageal echocardiography.

Keywords: acute myocardial infarction, contrast echocardiography, left atrial myxoma, stroke

1. Introduction

Left atrial myxoma is a common primary cardiac tumor, which is mostly benign and accounts for 30% to 50% of cardiac benign tumors.[1] Its clinical manifestations are complex and occur mainly in 3 forms: hemodynamic disorders caused by mitral valve stenosis; systemic embolism due to emboli shedding; and Carney syndrome caused by inflammation.[2] The diagnosis of myxoma mainly depends on echocardiography, however, due to poor image quality or atypical myxoma images, it will be misdiagnosed. The use of ultrasound enhancing agents may characterize image quality or atypical myxoma images, it will be misdiagnosed. The use of ultrasound enhancing agents may characterize image quality or atypical myxoma images, it will be misdiagnosed.

Contrast-enhanced echocardiography has played a very important role in identifying the nature of cardiac masses.[4] System embolization is a common complication of myxoma, in which the incidence of stroke is between 10% and 30%.[5–7] and the incidence of myocardial infarction is relatively low, at approximately 0.06%.[8] The occurrence of concurrent stroke and acute myocardial infarction (AMI) is rare. In this case report, left atrial myxoma was misdiagnosed as thrombosis, which successively induced acute myocardial infarction and stroke, contrast-enhanced echocardiography played an important role in definitive diagnosis.

2. Case report

A 44-year-old woman suddenly felt chest pain that radiated to the back at 7:00 am on December 29, 2016. There were no risk factors for coronary heart disease (CHD), such as hypertension, hyperlipidemia, diabetes, smoking, or family history of CHD. She was admitted to the local hospital, her blood pressure was 95/62 mmHg, heart rate was 90 beats/min, and ECG showed elevated ST segment elevations in the leads of II, III, AVF, V5, and V6. Troponin I was 2.84ng/mL (normal range 0–0.04ng/mL), CK-MB was 64.4ng/mL (normal range 0.6–6.3ng/mL). CKMB was 64.4ng/mL (normal range 0.6–6.3ng/mL). CK-MB was 64.4ng/mL (normal range 0.6–6.3ng/mL), BNP was 70.7ng/mL (normal range 0–285ng/mL), AMI in inferior-posterior wall was suspected, so emergency coronary angiography (CAG) was performed immediately.

The CAG showed a normal right coronary artery and left anterior descending coronary artery, however, the distal branch of the left circumflex artery (LCX) was thrombo-occluded with thrombolysis (Fig. 1A). A BMW guidewire reached the distal end,
and a 2.0 × 15 mm balloon at 12 ATM was used to pre-expand the LCX lesion; then, nitroglycerin and 20 mg of recombinant human pro-urokinase were injected into the coronary artery. The CAG showed the blood flow recovery (Fig. 1B), and the patient’s symptoms were relieved. On the second day, echocardiography showed a significant decrease in left posterior wall motion, with EF 49%. A hyperechoic mass was seen in the left atrium, the size was approximately 34 mm × 24 mm, and thrombosis was suspected. An antiplatelet and anticoagulant therapy of aspirin, clopidogrel, and warfarin were given during hospitalization while metoprolol and atorvastatin were given at the same time. After 2 weeks, the patient was discharged from the hospital and continued to take these oral medications.

One month later, on February 1st, 2017, the patient suddenly appeared unconscious after breakfast. In the local hospital, MRI showed multiple fresh cerebral infarctions in the left frontal temporal lobe, basal ganglia, and radiation crown (Fig. 2A and B). Magnetic resonance angiography (MRA) showed the left middle cerebral artery was slender, and distal branches were reduced (Fig. 2C). Echocardiography showed a great mass in the left atrium, which had no change in size compared with the last examination. The patient was diagnosed with acute multiple cerebral emboli. After 20 days, she was transferred to the Department of Neurology of our hospital for further treatment. In our hospital, transthoracic echocardiography (TOE) revealed apical ventricular aneurysm of the left ventricle, markedly decreased posterior inferior wall motion, EF 37%, and left atrial hyperechoic mass (Fig. 3A). Further transesophageal echocardiography (TEE) revealed a gelatinous and pedicle-less mass in the left atrium, which was wide based and attached to the foramen ovale at the atrial septum. Its surface was not smooth or villous, its morphology was variable and the mass oscillated with the heart contraction (Fig. 3B). To further clarify that the mass was thrombosis or tumor (high possibility of myxoma), a 2 mL intravenous bolus injection of Sonovue contrast (Bracco Altana Inc., Milan, Italy) was initiated with left ventricular contrast-enhanced echocardiography. Pathological results confirmed left atrium myxoma (Fig. 4B). After several weeks of rehabilitation, the patient was able to resume daily activities without chest discomfort or dyspnea. One year later, echocardiography showed no recurrence of left atrial myxoma. The patient generally was in good condition.

3. Discussion

The most common pathophysiological mechanism of AMI is rupture of coronary atherosclerotic plaque, which induces thrombosis, but 3% of AMI are caused by coronary thrombosis.[9] Coronary embolism may be direct, paroxysmal, or iatrogenic. Direct coronary embolism may result from a thrombus originating from the left atrium, left ventricle, or pulmonary veins, endocarditis of the aortic or mitral valve, and more rarely, cardiac tumors.[10,11] In this case, when the patient experienced AMI, the myxoma was misdiagnosed as a thrombus, and she was given anticoagulant therapy without timely surgery. Unfortunately, 1 month later, the patient presented with multiple acute cerebral emboli. In our hospital, transesophageal echocardiography and contrast-enhanced echocardiography showed that the left atrial mass was gelatinous with a non-smooth surface and variable shape. In particular, the left ventricular contrast-enhanced echocardiography showed the visualization of punctate contrast media in the mass, and with the exception of thrombus, myxoma was highly suspected. This diagnosis was confirmed by surgical pathology.

Left atrial myxoma is a benign primary cardiac tumor. There are different clinical symptoms, from asymptomatic to fever, multiple arterial emboli, endocrine dysfunction, and syncope and even sudden death.[12,13] Thus, the early diagnosis of myxoma is
crucial. Echocardiography plays a key role in the diagnosis of myxoma. The sensitivity of transthoracic echocardiography in diagnosing myxoma is 95%, and the sensitivity in transesophageal echocardiography is 100%. However, when the patient is critical, the poor quality of the images caused by position-restricted bedside instrument examinations or the atypical ultrasound images of myxoma can still lead to misdiagnosis of myxoma, which delays treatment and leads to a life-threatening situation.

Echocardiography, in the identification of myxoma and thrombosis, mainly shows the following different features. Myxomas are mostly located in the left atrium and are round or oval in shape, are colloidal and have variable shapes, and often have pedicles attached to the foramen ovale. The activity of myxomas is regular, which can invade the mitral valve in diastole and return to the left atrium at systole. However, the thrombus in the echocardiography is mostly located at the apex but also in the atrium. It is elliptical and pedicle-less with little change in shape, and some thrombi can move. When transthoracic echocardiography cannot identify myxomas or thrombus, transesophageal echocardiography and left ventricular contrast-enhanced echocardiography can be helpful to confirm the diagnosis. The left ventricular contrast-enhanced echocardiography showed the thrombus was an intracardiac filling defect; there was no contrast agent filling in the thrombus, but scattered contrast agent filled in the myxoma. Although transesophageal echocardiography can clearly show the characteristics of myxoma, it requires anesthesia and intubation, and when the patient’s condition is critical, the examination is limited. Contrast-enhanced echocardiography is rapid and convenient, can be performed at the bedside without anesthesia and can be completed within minutes. This technique provides another rapid diagnostic method for differential diagnosis of cardiac thrombus and tumor.

Although myxoma is mostly benign, the patient has a risk of sudden death, and early surgery should be performed as soon as the diagnosis is confirmed. Tumor resection is the only effective method with a high cure rate, but there is a small recurrence rate; thus, it is important to perform echocardiography on regular review for early detection of recurrence.

The lesson of this case is that, for cardiac masses, comprehensive assessments should be performed with multiple imaging methods. If necessary, contrast-enhanced echocardiography should be used to clarify, so as not to delay the timing of surgery and bring potential risk of death to patients. For some unexplained causes of embolism, this case has important implications. Echocardiography plays an important role in definitive diagnosis.

4. Conclusion

Although it is very rare for myxoma to cause acute myocardial infarction complicated by stroke, the possibility of multiple arterial emboli due to myxoma should be considered when the clinical and vascular conditions do not match in young patients with no risk factors. Contrast-enhanced echocardiography has
Figure 3. (A) TOE revealed left atrial strong echoic mass (yellow arrow); (B) TEE X-plane showed the hyperechoic mass in the left atrium, which was wide based and pedicle-less and attached to the foramen ovale at the interatrial septum. (C) Contrast-enhanced echocardiography showed absence of contrast agent filling in the left atrial abnormal mass and exploration of scattered and dotted contrast media within the mass. TEE = transesophageal echocardiography, TOE = transthoracic echocardiography.

Figure 4. (A) Surgical specimen of the completely resected atrial myxoma. (B) Pathological results confirmed left atrium myxoma.
obvious advantages in the definite diagnosis of myxoma and a very large application prospect in clinical practice.

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The patient was informed about the research content and signed informed consent for publication of this case report.

Author contributions

Conceptualization: Qiushuang Wang, Feifei Yang.

Data curation: Qiushuang Wang, Fei Zhu.

Investigation: Feifei Yang, Qiushuang Wang, Cunshan Yao.

Methodology: Qiushuang Wang, Cunshan Yao.

Writing – original draft: Feifei Yang, Qiushuang Wang.

Writing – review & editing: Qiushuang Wang.

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