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Case Report

Assessment of embolic cardiomyopathy from atrial myxoma on magnetic resonance imaging: A case report [☆]

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ABSTRACT

Atrial myxoma is the most common benign cardiac tumor, which can present with diverse symptoms. Systemic embolism is a frequent complication, affecting up to one-third of cases and frequently involving cerebral arteries. However, cardiac myxoma-induced myocardial infarction (MI) is rare. We report a case of a 56-year-old man presenting with predominant neurological symptoms and an unexplained elevation of hs-Trop T without clinical signs of acute MI. Computerized tomography of the head showed no acute lesions, but subsequent magnetic resonance imaging (MRI) revealed multiple small ischemic lesions and old microhemorrhage foci. A comprehensive cardiovascular investigation was performed. Ultrasonography revealed a left atrial mass. Cardiac MRI confirmed the mass was an atrial myxoma, and showed many old infarctions and scarring lesions in the cardiac muscle. The patient underwent tumor resection, but residual motor-neurological deficits were observed. This case emphasizes the importance of cardiac MRI in the diagnosis of multiple focal infarctions attributed to coronary embolism.

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Introduction

Atrial myxoma is a cardiac tumor that arises from the endocardium and is the most common benign primary tumor of the heart. Although many patients with cardiac myxoma are asymptomatic, up to one-third of cases can result in systemic

embolism, frequently involving cerebral arteries in 12%–21% of patients[1]. Embolization can cause a wide range of clinical manifestations, including stroke, transient ischemic attacks, and peripheral arterial occlusion. However, atrial myxoma-induced myocardial infarction (MI) is a rare occurrence and not widely reported in the literature. The diagnosis of cardiac myxoma is typically established using noninvasive imaging

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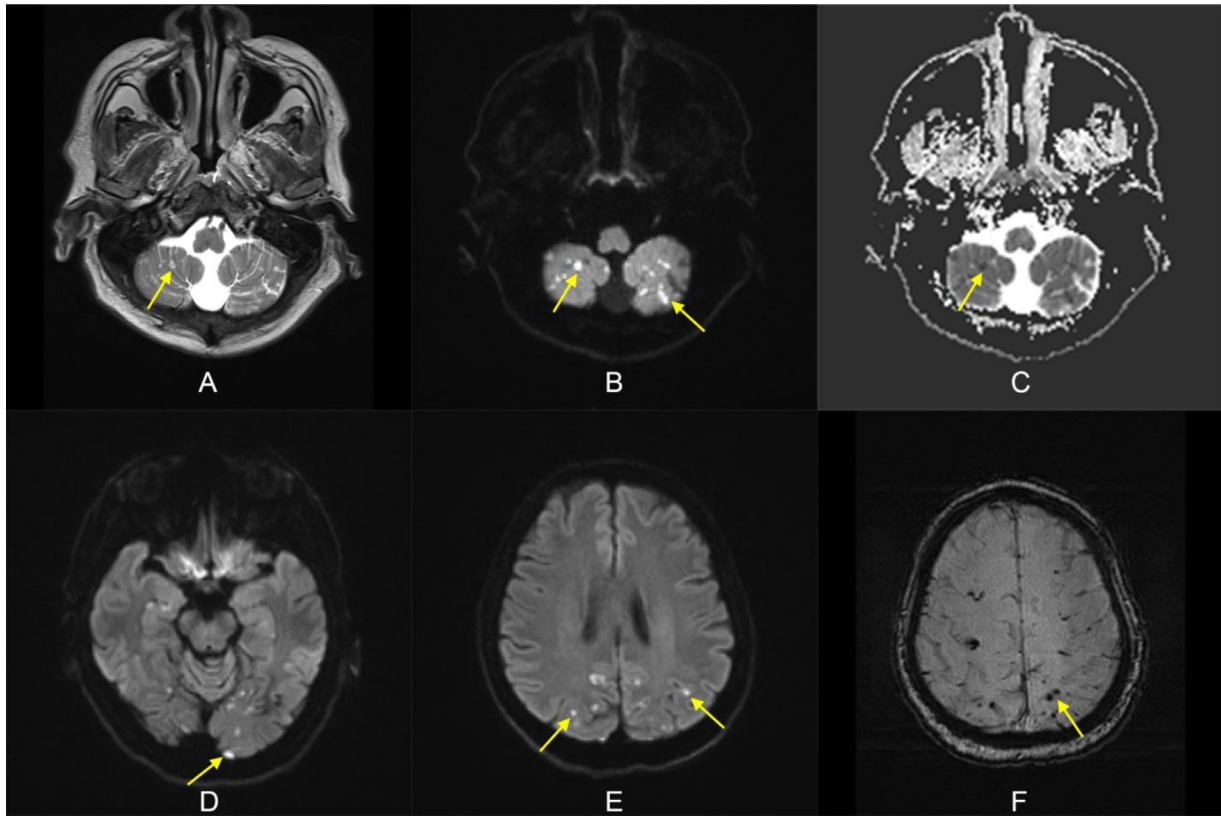


Fig. 1 – Initial magnetic resonance imaging of the brain: T2W, DWI, and ADC images (A–C) showing bilateral distribution of multiple small acute ischemic lesions and multilevel infarcts. The DWI images (B, D, E) showing restricted diffusion within the cerebellar hemispheres, as well as the temporal, parietal, and occipital cortex, primarily affecting the posterior circulation. The SWI images (F) reveal multiple old cerebral microbleeds, especially within the deep gray matter and posterior fossa."

modalities, such as transthoracic echocardiography and transesophageal echocardiography, both of which can demonstrate the characteristic appearance of a mobile mass attached to the endocardium by a stalk. However, cardiac magnetic resonance imaging (MRI) has emerged as a valuable tool in the assessment and management of patients with cardiac tumors, providing detailed information about the location, size, and characteristics of the tumor, as well as any associated complications, including embolic phenomena. Additionally, MRI is more sensitive in detecting small focal ischemic lesions and is particularly useful in patients with a normal electrocardiogram (ECG) or negative cardiac biomarkers, where the diagnosis of MI may be challenging[2,3].

Case presentation

A 56-year-old man was presented to the emergency department because of a sudden onset of severe dizziness and headaches (National Institutes of Health Stroke Scale =1), which began 2 hours before the admission, in conjunction with a mild chest pain without other significant cardiac symptoms. The patient was stable but too dizzy to stand up. His

blood pressure was 150/ 90 mm Hg. Neurological examination revealed vertical and horizontal nystagmus, ataxia of the right arm, and a mild decrease in strength (4/5) of the right upper and lower extremities. Investigations of speech and extraocular muscle functions were unremarkable. Computer tomography (CT) of the head did not show any acute brain lesions which could explain the patient's manifestation. The high-sensitivity troponin T (hs-Trop T) of the initial sample was 164.6 ng/L (normal: <14.0 ng/L) but it decreased to 153.3 and 120.2 ng/L after 2 and 6 hours, respectively. The routine ECG and other laboratory studies, including complete blood count, lipid profile, thyroid function test, rheumatoid factor, and antinuclear antibody, were unremarkable.

The patient reported several episodes of mild dizziness and headaches over the past 2 years but he had never been hospitalized. The patient drank alcohol and smoked occasionally. His past medical history included gastroesophageal reflux disease and mild dyslipidemia. No other risk factors nor family history of cardiovascular diseases were recorded.

The patient was managed symptomatically and started on a low dose of aspirin. After being transferred to the neurology department, he underwent a brain MRI scan, which showed a bilateral distribution of multiple small ischemic lesions and multilevel infarcts, mainly in the posterior circulation, pre-

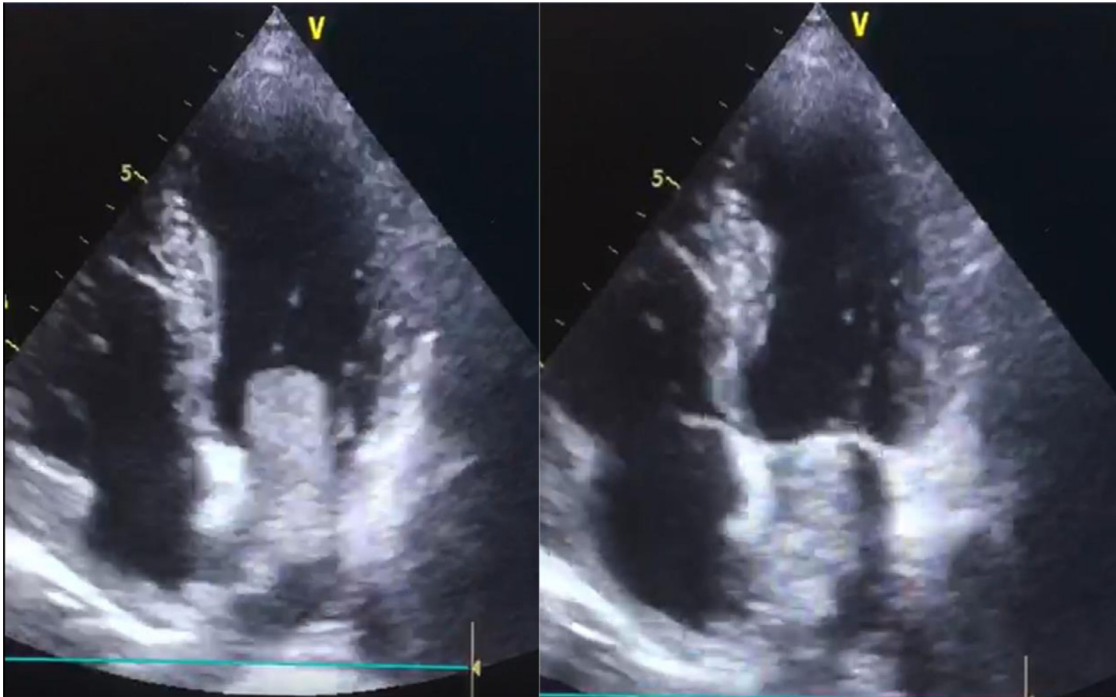


Fig. 2 – Transthoracic echocardiography during hospitalization showing a large, mobile, lobulated, heterogeneous echoic mass, attaching to the interatrial septal and prolapsing through the mitral valve in diastole, along with the reduction of wall motion in the apical region.

dominantly in gray-white matter junctions. Additionally, several old microhemorrhage foci were identified. These findings may relate to the intraluminal injuries of small-end arteries of brain parenchyma (Fig. 1) and suggested a proximal embolic source.

A comprehensive cardiovascular investigation was warranted. Duplex ultrasonography of carotid and vertebral arteries did not detect any significant stenosis. Transthoracic echocardiography revealed a pedunculated, highly mobile, echo-dense, and heterogeneous mass with irregular margins, attached to the interatrial septum and prolapsing through the mitral valve in diastole (Fig. 2). The ejection fraction was 63%, and there was reduced wall motion in the apex region. To determine whether the mass was a thrombus or a suspected myxoma tumor, further assessment with cardiac MRI was performed. The images demonstrated a spheroidal shape with diameters of $45 \times 21 \times 26$ mm that proved to be an enhanced tumor without a capsule. The signal intensity was intermediate on T1W, hyperintense on cine SSFP compared to the myocardium, and heterogeneous enhancement on late gadolinium enhancement (Fig. 3). The patient was diagnosed with left atrial myxoma. Additionally, multiple small focal subendocardial and transmural lesions were identified in the myocardium, with a random distribution observed across the territories of all coronary arteries. These findings highly suggested that the lesions were caused by embolism originating from the tumor. Furthermore, CT coronary angiography performed subsequently did not show any significant coronary stenosis.

The tumor was successfully resected without complications. Pathological results confirmed the diagnosis of left atrium myxoma, characterized by cells in a loose stroma, accompanied by some thrombi within the tumor (Fig. 4). The patient recovered well and was able to return to daily activities without any chest discomfort, dyspnea, or headache, although he did have mild residual motor-neurological deficits. Follow-up cardiac and brain MRI after 3 months was consistent with chronic cardiac and cerebral embolism lesions (Fig. 5).

Discussion

This case report highlights a rare etiology of embolic MI, namely myxoma. Myxomas are primarily known to cause embolic events in various organs, such as the brain and extremities, but their occurrence in the coronary system is relatively rare. While thrombosis is the leading cause of MI, and embolic events account for only a small percentage of cases. Thus, the occurrence of myxoma-induced embolic MI is a noteworthy phenomenon that warrants further investigation. Furthermore, the concurrent presence of both brain and cardiac lesions in this patient is an exceedingly uncommon occurrence, with limited previous reports in the literature. This rare occurrence of myxoma-induced embolic MI underscores the importance of maintaining a broad differential diagnosis and considering uncommon etiologies in patients with atypical clinical presentations.

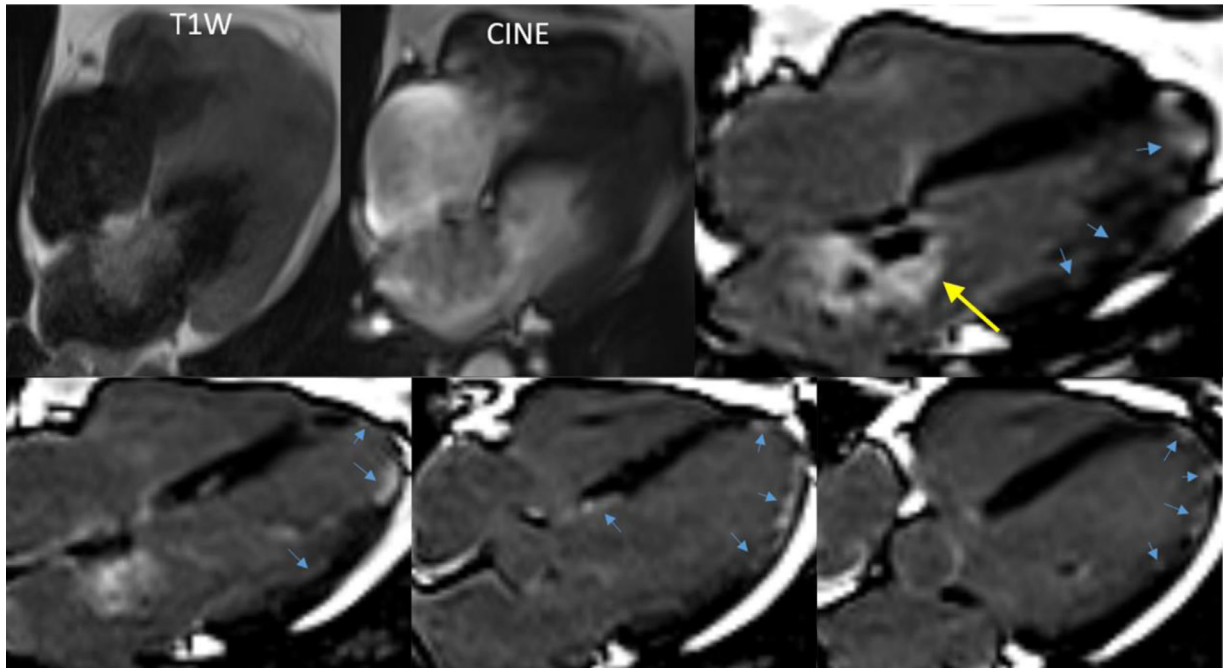


Fig. 3 – Contrast-enhanced cardiac MRI showing a mass with an intermediate signal on T1-weighted sequences, hyperintense on cine SSFP compared to normal myocardium, and exhibiting late gadolinium enhancement. The central areas that demonstrate no enhancement correlate with hemorrhagic breakdown products (yellow arrow). Late CMR images showing multiple small myocardial scars as separate and randomly distributed lesions (blue arrow).

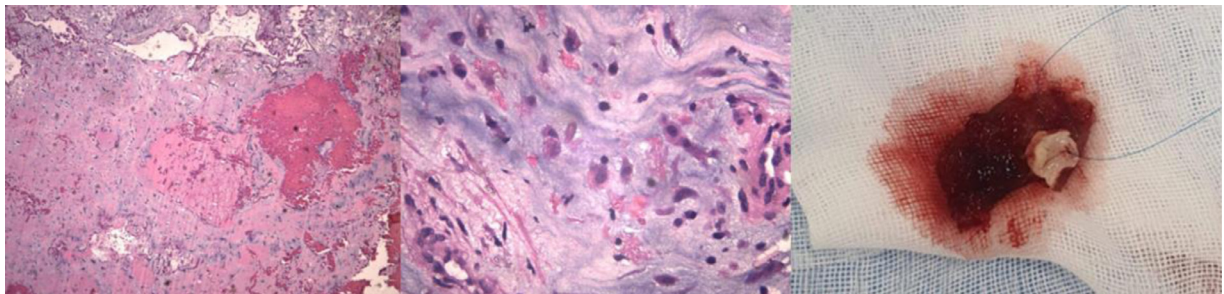


Fig. 4 – Pathological results confirmed a left atrial myxoma characterized by gelatinous tissue attached to the tumor stalk. The tumor cells included stellate or globular myxoma cells, surrounded by ground substance containing chondroitin sulfate and hyaluronic acid, with numerous red blood cells owing to hemorrhage.

The patient's cardiac presentation prior to hospitalization was asymptomatic, likely due to the small size of the injury, which was confirmed by CT coronary angiography showing no significant narrowing of the major coronary vessels. The history of recurrent episodes of mild dizziness and headaches suggested a progression of the illness, although the small size of the lesions may explain his tolerability and clinically silent period. A new cardiac embolism was hypothesized to be the cause of the worsening symptoms that led to hospitalization, as it reduced the patient's tolerance and stimulated the autonomic system. The injury was not large enough to cause a typical myocardial infarction, it was characterized by a transient elevation of cardiac enzymes.

However, it is not possible to conclude whether the mechanism of ischemia was attributed to myxomatous tissue emboli or occlusion by thrombus on the myxoma surface. The formation of thrombi on the tumor's surface often occurs on mobile tumors with stalks and irregular margins[4,1,3]. Thromboembolism may have been the primary cause of ischemia in our patient, as supported by the follow-up MRI findings of the cardiac ischemic lesions becoming more compact 3 months after the initial presentation of symptoms. Although myxomatous emboli may invade the vessel walls, and myxoma cells may produce interleukin-6, which promotes extracellular matrix degradation and can lead to cerebral aneurysm formation, no aneurysms were observed in our patient[5,2,6].

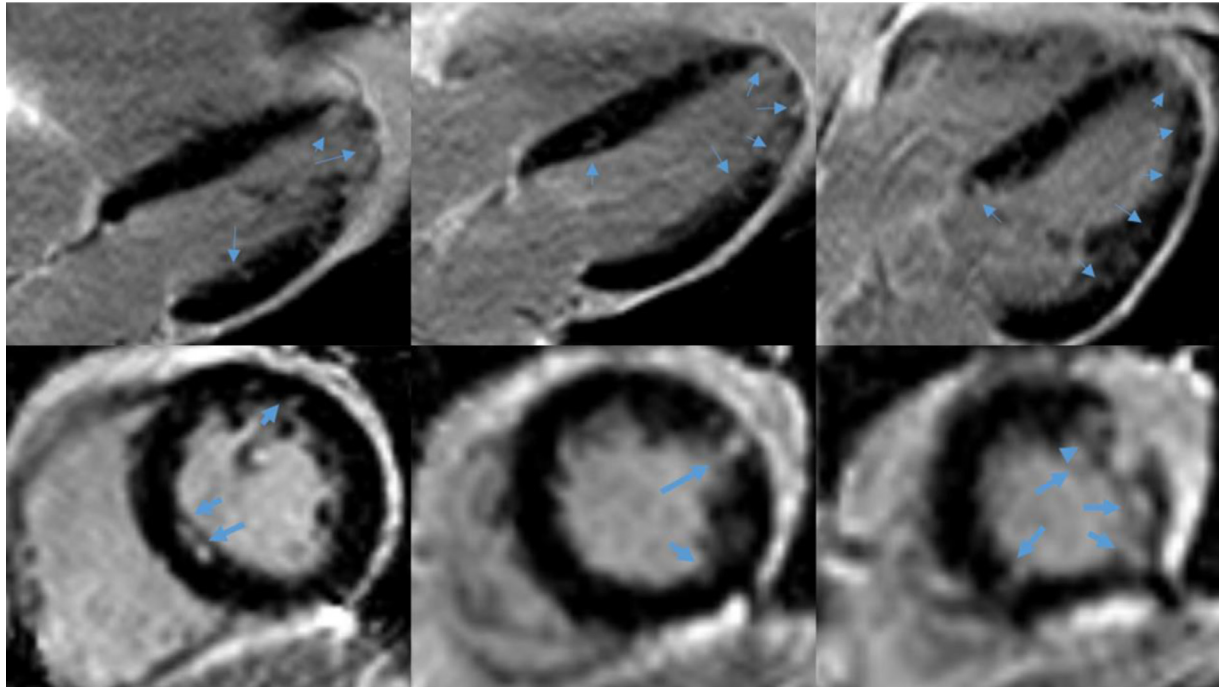


Fig. 5 – Repeated cardiac MRI after 3 months showing multiple scars on 4-chamber views and short axis views.

Magnetic resonance imaging has become a valuable tool in the diagnosis of cardioembolic cerebral infarction as well as embolic MI. In particular, diffusion-weighted MRI has demonstrated high sensitivity and specificity for detecting cerebral infarctions. Additionally, cardiac MRI is useful in assessing myocardial pathology and providing detailed information on cardiac function and structure, including the presence of cardiac tumors. Cardiac MRI serves as an excellent tool for differentiating between intracardiac thrombi and tumors, such as atrial myxomas. The use of cardiac MRI in diagnosing embolic MI has shown high diagnostic accuracy and can help identify patients who may benefit from further anticoagulation or surgical intervention. As a result, MRI has greatly improved the ability of clinicians to accurately diagnose and manage these conditions, ultimately leading to better patient outcomes[6,3].

This case report serves as a reminder of the potential variability in clinical manifestations and the need for a comprehensive diagnostic approach in patients with myxoma who have atypical clinical presentations such as those with transient elevations of cardiac enzymes or suspected embolic events. Further research is necessary to better understand the incidence, risk factors, and optimal management strategies for myxoma-induced embolic MI.

Conclusion

Atrial myxoma can present with various clinical manifestations, including uncommon complications such as embolic cardiac infarction and other embolic events. MRI has been demonstrated to be an effective tool in the diagnosis of acute

cardioembolic cerebral infarction and embolic myocardial infarction.

Data availability

All data generated or analyzed during this study are included in this article.

Patient consent

Written informed consent for publication of the patient's clinical details and images was obtained from the patient prior to submission. A copy of the signed consent form is available for review by the Editor of the journal.

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