A Large Left Ventricular Cystic Thrombus: Unusual Presentation of a Common Entity

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INTRODUCTION

Left ventricular (LV) thrombi are not uncommon following acute myocardial infarction (AMI). They are one of the most feared complications because of increased embolic risk.[1] We describe our case as the largest atypical cystic LV thrombus that has been reported, which mimicked a hydatid cyst.

CASE PRESENTATION

A 40-year-old man was brought to our emergency department from prison with chest tightness and dyspnea. He was in acute distress and demonstrated signs of acute heart failure. He was stabilized as per guideline protocols. Resting electrocardiography showed ventricular tachycardia (Figure 1) that was aborted successfully using emergency electrical defibrillation. Postdefibrillation electrocardiography showed normal sinus rhythm with pathologic Q waves and ST-segment elevation in leads V_1 to V_3, representing anteroseptal wall myocardial infarction (Figure 2).

Because of respiratory distress and ongoing chest pain along with the electrocardiographic findings, the patient was intubated and shifted immediately to the cardiac catheterization laboratory. Left heart catheterization and coronary angiography were performed via the right radial approach and showed a totally occluded mid left anterior descending coronary artery (Figure 3A). The left anterior descending coronary artery thrombus was aspirated, and the procedure was completed with the successful deployment of two drug-eluting stents (Figure 3B). An intra-aortic balloon pump was inserted through the right femoral artery, and an intravenous glycoprotein IIb/IIIa inhibitor (tirofiban) was initiated and continued for 24 hours. The patient was then transferred to the coronary care unit for further stabilization and management. The next day he was successfully extubated, and the intra-aortic balloon pump was removed.

Transthoracic echocardiography was done on the next day and showed moderately reduced LV systolic function with an ejection fraction of 40% (by the modified Simpson method) and anterior, septal, and apical wall akinesis with no hemodynamically significant valve disease. Surprisingly, a large hyperechoic mobile cystic mass measuring 4.3×2.3 cm was found attached to the apical septal and inferior walls of the left ventricle (Figure 4, Videos 1–4). The differential diagnosis included but was not limited to hydatid cyst, a large atypical thrombus with central lysis, bronchogenic cyst, blood cyst, and cardiac myxoma. *Echinococcus* titer was measured at 1:16. Complete blood count showed leukocytes of 7.8 k/μL (normal range, 4.0–11 k/μL) and eosinophils of 0.4 k/μL (normal range, 0.0–0.8 μL). Pan computed tomography with contrast was performed to asses for extracardiac cystic lesions; results were negative except for a nonenhancing LV filling defect (Figure 5). Cardiac magnetic resonance was not available.

Accordingly, in addition to routine anti-ischemic drug therapy, oral anticoagulation was started to cover the possibility of LV thrombus. Because routine international normalized ratio monitoring was not feasible in the prison, apixaban was preferred over warfarin.

One month later, the patient was brought for follow-up. He was asymptomatic, with no clinical evidence of heart failure or thromboembolism. Repeat follow-up transthoracic echocardiography showed almost complete disappearance of the cystic mass except for a small stalk (Figure 6, Videos 5–8), suggesting the diagnosis of a large atypical LV cystic thrombus.

DISCUSSION

The incidence of LV thrombus has been markedly reduced with advances in AMI treatment, including primary percutaneous coronary intervention and anticoagulant therapy, reaching 2.5%–5% from 7%–46% during the previous prethrombolytic and thrombolytic eras.[2]

LV thrombus can occur within 24 hours up to 2 weeks after AMI. The main underlying pathogenesis is based on Virchow's triad, which comprises blood stasis, endothelial injury, and hypercoagulable state. Anterior infarction, larger infarct size, akinetic or dyskinetic wall segments, and reduced LV systolic dysfunction (ejection fraction $\leq 40\%$) are believed to predict LV thrombus formation.[3] Thrombi mostly tend to be located apically, but they can be attached to the septal wall and to the inferoposterior wall.[4] On the basis of a literature review (PubMed, Simmon), our case appears to be the largest reported. The appearance of the thrombus in our case mimicked hydatid cyst.[5] Multiple cases with atypical cystic thrombi

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Keywords: Acute myocardial infarction, Cystic thrombus, Hydatid cyst, Anticoagulation

Conflicts of interest: The authors reported no actual or potential conflicts of interest relative to this document.

2468-6441

https://doi.org/10.1016/j.case.2017.09.009

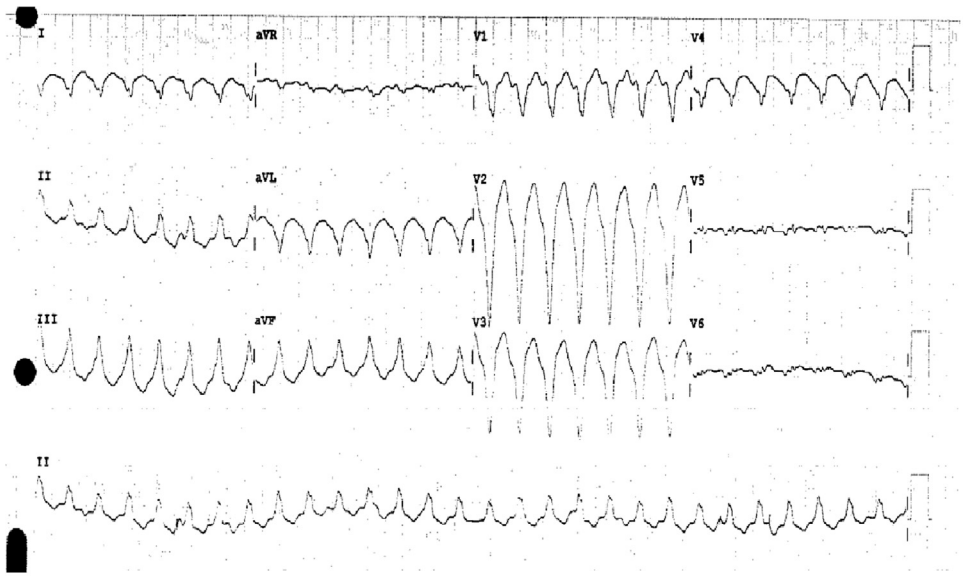

Figure 1 Electrocardiogram on presentation showed ventricular tachycardia.

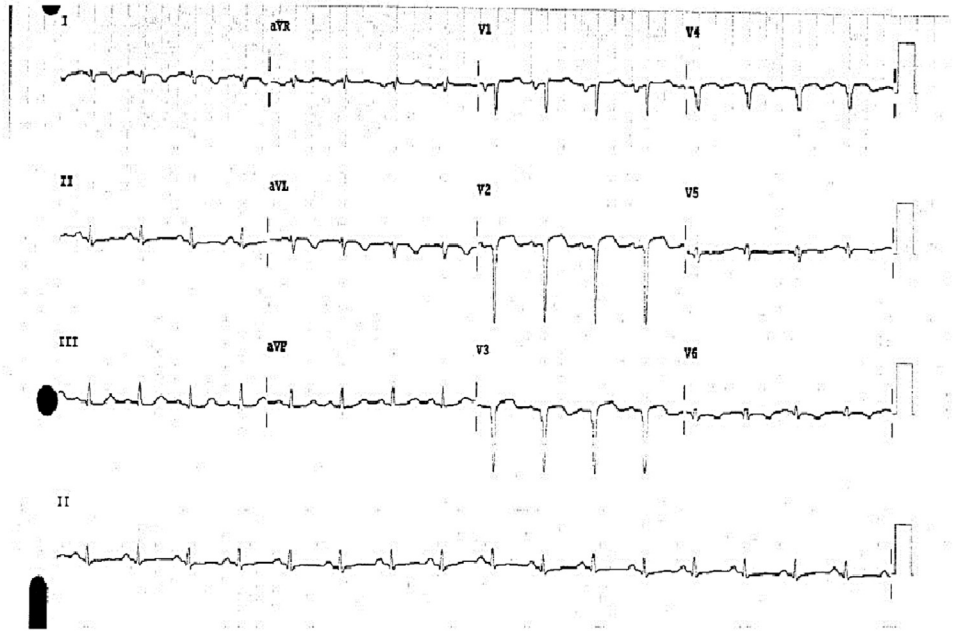

Figure 2 Electrocardiogram after defibrillation showed normal sinus rhythm with pathologic Q waves and ST-segment elevation in leads V_1–V_3, representing anteroseptal wall myocardial infarction.

have been reported in the literature.[6,7] Therefore, it is becoming necessary to consider this type of thrombus as part of the differential diagnosis of LV cystic masses. Early thrombus identification permits early treatment and hopefully avoidance of possibly devastating sequelae.[8]

Absence of liver and lung lesions on computed tomography and the low *Echinococcus* titer, along with absence of leukocytosis and eosinophilia, made hydatidosis less likely, especially given that hydatid cyst occurs in the liver or lungs 90% of the time.[9] Pan computed tomography is considered a reliable test for the diagnosis of hydatid cyst, with accuracy of 98%.[10,11]

Few cases of intracardiac bronchogenic cyst, blood cyst, and atypical cardiac myxoma have been reported in the