Massive Mobile Left Ventricular Thrombus in a Patient With Normal Left Ventricular Systolic Function

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INTRODUCTION

Left ventricular thrombi are a severe pathologic phenomenon commonly associated with conditions related to impaired left ventricular systolic function, most commonly following acute myocardial infarction. However, in the setting of preserved systolic function, left ventricular thrombi are extremely rare and may be mistaken for cardiac tumors. Their early and prompt recognition is therefore vital in patients' clinical outcomes. We present a case with normal systolic function and thrombus formation. The patient presented to the emergency department with abdominal pain and was diagnosed with multiple renal infarcts. Subsequently, she was found to have a large, mobile, cone-shaped mass in the left ventricle. The mass was surgically excised and was found to be a thrombus on histopathologic examination. This case highlights the importance of echocardiography in the diagnosis of left ventricular thrombi.

CASE DESCRIPTION

The patient was a 69-year-old woman with a medical history significant for hyperlipidemia and tobacco use. There was no significant history of bleeding, easy bruising, or complicated pregnancies. Her family history was not significant for thrombophilia. She presented to the emergency department reporting severe abdominal pain that woke her from sleep. The pain was associated with nausea, vomiting, and diarrhea. Physical examination revealed epigastric and suprapubic tenderness to palpation but was otherwise unremarkable. Laboratory tests showed a white blood cell count of 13,000 cells/μL, a prothrombin time of 12.7 sec, an activated partial thromboplastin time of 23 sec, and a platelet count of 309,000/μL. Electrocardiography displayed normal sinus rhythm, with no ST- or T-wave changes. Computed tomography of the abdomen and pelvis with contrast revealed multiple areas of decreased enhancement in the kidneys suggestive of infarcts (Figure 1). The computed tomographic findings raised concern for a cardioembolic source. The patient's only home medication was hydrochlorothiazide 25 mg/d. Transthoracic echocardiography revealed normal overall left ventricular systolic function without any segmental wall motion abnormality and a large (2 $\times$ 4 cm), cone-shaped, highly mobile mass in the left ventricle (Videos 1-4). The mass had its base anchored to the distal anteroseptal wall, with its apex directed toward the mitral valve. Echocardiographic findings were concerning for a left ventricular tumor, and given the patient's recurrent embolisms, she was referred for emergent surgical excision.

Intraprocedural transesophageal echocardiography confirmed the presence of the mass (Figures 2A–2C). Surgical exploration revealed a friable, broad-based mass attached to ventricular trabeculations in the distal anteroseptum (Figures 2D and 2E). The mass was completely excised, and the left ventricular cavity did not reveal any additional endocardial lesions. Immediate postoperative transesophageal echocardiography revealed complete excision of the mass (Figure 2F). Surgical pathology showed layers of collagen, fibrin, scattered inflammatory cells, erythrocytes, and macrophages, consistent with an organizing thrombus (Figure 3). After surgery, the patient's hospital course was uncomplicated. She was started on anticoagulation with warfarin. Given acute thrombosis and the use of warfarin, a hypercoagulable workup was not completed. However, limited testing revealed a positive lupus anticoagulant and negative anticardiolipin and B2 macroglobulin antibodies. This finding raises the possibility of an underlying hypercoagulable state, perhaps antiphospholipid syndrome, but this was not confirmed on subsequent testing.

Follow-up transthoracic echocardiography showed normal left ventricular size and systolic function, with an ejection fraction $>$ 55% and no evidence of apical stasis or reduction in Doppler velocities in the apical region. The patient received an age-appropriate malignancy workup that included mammography and colonoscopy, and both were unremarkable.

DISCUSSION

Left ventricular thrombi occur most often as a complication of ST-segment elevation myocardial infarction. Other risk factors are consistent with Virchow's triad and include blood stasis, local myocardial injury, and hypercoagulability. Moreover, left ventricular thrombus has also been reported in cases with transient systolic dysfunction, such as takotsubo cardiomyopathy.[1] Anterior wall ST-segment elevation myocardial infarction creates ideal circumstance for thrombus formation, as it usually leads to impaired systolic function, in which blood stasis and endothelial injury may occur. The incidence of left ventricular thrombus in the prereperfusion era was 34% following an anterior wall myocardial infarction.[2] In the postreperfusion era, however, the incidence has decreased to 4%.[3]

In the presence of normal left ventricular systolic dysfunction, left ventricular thrombus formation has rarely been reported in the setting

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VIDEO HIGHLIGHTS

Video 1: Four-chamber view reveals highly mobile echodensity and preserved systolic function.
Video 2: Parasternal short-axis view reveals highly mobile echodensity and preserved systolic function.
Video 3: Two-chamber view reveals a highly mobile echodensity and preserved systolic function.
Video 4: Apical three-chamber view reveals a highly mobile echodensity and preserved systolic function.

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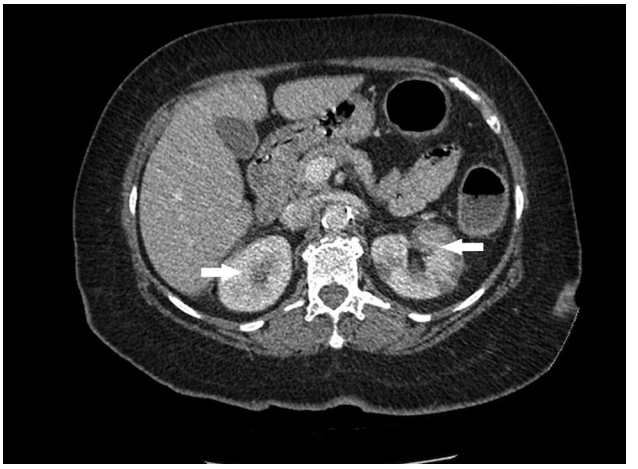

Figure 1 *Arrows* indicate areas of decreased enhancement consistent with renal infarcts.

of hypercoagulable states such as protein C and S deficiency,[4] antiphospholipid syndrome, essential thrombocytopenia, and malignancies.[5,6] Endothelial injury can also give rise to thrombus formation and it has been reported in patients with endomyocardial fibrosis.[7]

Cardiac magnetic resonance is considered the gold standard in the diagnosis of left ventricular thrombus,[8] but given its wide availability and low cost, echocardiography remains the initial test of choice. On echocardiography, left ventricular thrombus generally appears as a mural or pedunculated echodensity, often of similar acoustic properties to normal myocardium. With contrast injection, thrombi or tumors appear as filling defects, while echocardiographic artifacts are obscured. Near-field artifact may also give the appearance of apical thrombus.

In the presented case, given the size, heterogeneous appearance, and mobility of the mass, the working diagnosis was a cardiac tumor,

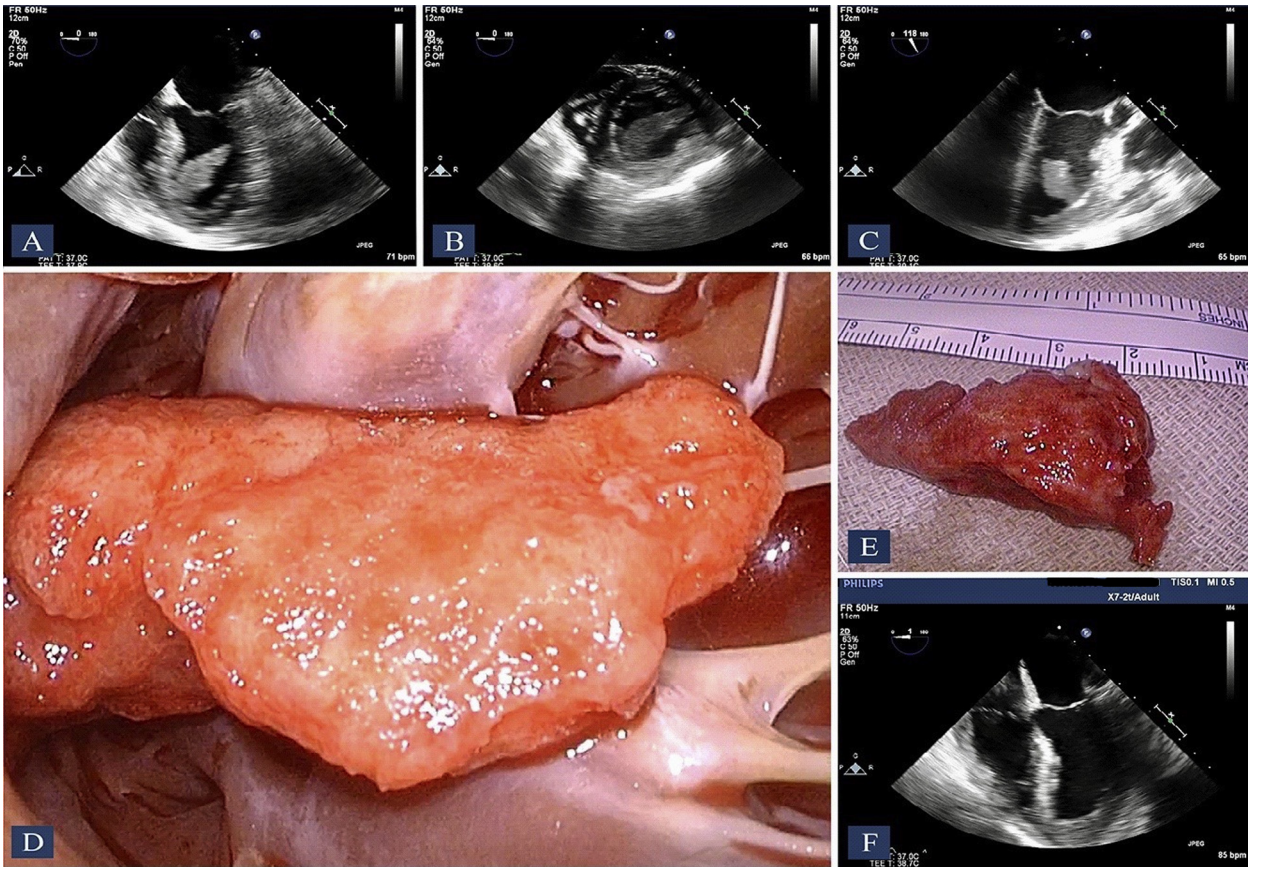

Figure 2 **(A–C)** Intraoperative transesophageal echocardiogram showing a left ventricular thrombus originating from the lower interventricular septum. **(D)** Intraoperative picture of the mass seen from the outflow tract. **(E)** Specimen. **(F)** Immediate postoperative transesophageal echocardiogram showing successful removal of the mass.

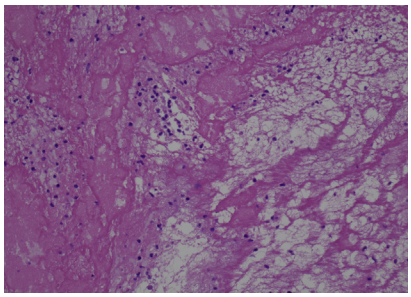

Figure 3 Layers of deposition of collagen and fibrin and scattered inflammatory cells, erythrocytes, and macrophages, consistent with organizing thrombus.

possibly a myxoma. The literature contains multiple reports of misdiagnosed intracavitary thrombus as a myxoma.[9] Given the presumed embolization, the patient underwent emergent surgical resection, which confirmed the diagnosis. This highlights the limitations of current noninvasive imaging modalities to confirm the pathologic diagnosis.

In the evaluation of clinical features, laboratory results, imaging, and age-appropriate malignancy workup, we could not detect any underlying abnormality in our patient that would give rise to the development of left ventricular thrombosis. However, a full hypercoagulable workup was not completed. The presence of positive lupus anticoagulant antibodies raises the possibility of underlying antiphospholipid syndrome, but this was not confirmed with subsequent testing. The hypothesis of microvascular ischemia causing aggregation of platelets by means of inducing patchy areas of endocardial fibrosis with formation of thrombi on these endomyocardial areas has previously been described.[5] Because our patient had no evidence of acute coronary syndrome, with normal findings on electrocardiography, normal biomarker levels, and no history of chest pain, we did not evaluate her coronary anatomy. Foresight into the possibility of entertaining other diagnosis might have led us to perform an endomyocardial biopsy during surgical excision to evaluate for an underlying pathophysiology. In a previous case report of a woman presenting with multiple arterial emboli, left ventricular thrombus, and no identifiable cause for heart disease or thrombus formation, a normal coronary arterial tree was seen, with no atherosclerotic disease and a negative hypercoagulable panel.[6]

As previously reported in studies of normal left ventricular systolic function and left ventricular thrombus, systemic embolism is a recurrent clinical presentation.[5,9-11] Although many patients have complete resolution with anticoagulant therapy, recurrent embolic phenomena are a common complication, as seen in our patient. Having discussed the relative success of thrombus resolution with anticoagulation,[12] the mere presence of an embolic source in the ventricle poses an immediate threat to a patient's life, and emergent surgical intervention to remove the thrombus and to definitively diagnose the mass seems appropriate.

CONCLUSION

Left ventricular thrombi can occur in patients with normal systolic function. In many of these patients, the presenting symptom is systemic embolization, and prompt echocardiography must be undertaken. Echocardiographic findings in left ventricular thrombi may be indistinguishable from those in cardiac tumors such as myxoma.[9] It is important that clinicians retain thrombus in the differential diagnosis of any cardiac mass visualized on echocardiography.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2019.07.007.

REFERENCES

1. Sasaki N, Kinugawa T, Yamawaki M, Furuse Y, Shimoyama M, Ogino K, et al. Transient left ventricular apical ballooning in a patient with bicuspid aortic valve created a left ventricular thrombus leading to acute renal infarction. Circ J 2004;68:1081-3.
2. Weinreich DJ, Burke JF, Pauletto FJ. Left ventricular mural thrombi complicating acute myocardial infarction. Long-term follow-up with serial echocardiography. Ann Intern Med 1984;100:789-94.
3. Gianstefani S, Douiri A, Delithanasis I, Rogers T, Sen A, Kalra S, et al. Incidence and predictors of early left ventricular thrombus after ST-elevation myocardial infarction in the contemporary era of primary percutaneous coronary intervention. Am J Cardiol 2014;113:1111-6.
4. Pahuja M, Ainapurapu B, Abidov A. Large Left ventricular thrombus in a patient with systemic and venous thromboembolism secondary to protein C and S deficiency. Case Rep Cardiol 2017;2017:7576801.
5. Verma AK, Alam M, Rosman HS, Brymer J, Keith F. Systemic embolization from thrombus in normal left ventricles. Chest 1988;93:441-2.
6. Vaganos S, Fox K, Kitchen J. Left ventricular thrombus in the absence of detectable heart disease. Chest 1989;96:426-7.
7. Malani S, Chadha D, Banerji A. Biventricular thrombosis in a structurally normal heart at high altitude. BMJ Case Rep 2014;2014:bcr2014204520.
8. Weinsaft JW, Kim J, Medicherla CB, Ma CL, Codella NC, Kukar N, et al. Echocardiographic algorithm for post-myocardial infarction LV thrombus: a gatekeeper for thrombus evaluation by delayed enhancement CMR. JACC Cardiovasc Imaging 2016;9:505-15.
9. Allende NG, Sokn F, Borracci R, Milani A, Kusselevski A, Camilletti J, et al. Giant pedunculated thrombus with normal left ventricular systolic function mimicking myxoma. Echocardiography 2011;28:E31-3.
10. Matitiau A, Tabachnik E, Sthoeger D, Birk E. Thrombus in the left ventricle of a child with systemic emboli: an unusual presentation of hereditary protein C deficiency. Pediatrics 2001;107:421-2.
11. Wiyono SA, Vletter WB, Soliman OI, ten Cate FJ, Geleijnse ML. Thrombus in a normal left ventricle: a cardiac manifestation of pheochromocytoma. Echocardiography 2010;27:195-7.
12. Butman SM. Rapid resolution of a massive left ventricular thrombus by usual systemic anticoagulation. Am Heart J 1991;122:864-6.