Case Report

Boxer Thrombus: A Large Highly Mobile Thrombus in a Normal Functioning Left Ventricle

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A 45-year-old woman with complaint of left side weakness admitted to neurology ward. Trans-esophageal echocardiography showed a large, highly mobile mass in left ventricle with its tip looking like a fist, punching the aortic valve cusps. Emergency operation was done and the mass was confirmed to be a large thrombus and was removed completely. During the follow up, patient was free of symptoms.

Introduction

Pathologic arterial or venus thromboembolism results from a complex interplay of inherited and acquired risk factors. The most common inherited thrombophilia is factor V leiden.[1] Immobilization, surgery, and bed rest are well documented risk factors for thrombosis. Antiphospholipid antibody syndrome is also associated with an increased risk of thrombosis.[2] Among cardiac diseases associated with ventricular thrombus formation and subsequent thromboembolism, patients with ischemic cardiomyopathy and apical aneurysm appear to be at particularly high risk.[3] It is extremely rare for a thrombus to form in a normal functioning left ventricle.[4]

Case report

A 45-year-old woman with chief complaint of left side weakness was admitted to neurology ward. Medical history regarding any predisposing condition was unrevealing. CT scan showed non-hemorrhagic brain infarction. The patient underwent trans-esophageal echocardiography (TEE) which showed a large, homogenous, very elongated, and highly mobile left ventricular mass originating from anterior wall near the apex and extending into left ventricular outflow tract. Interestingly, its tip was looking like a fist, punching the aortic valve cusps (Fig. 1). No additional cardiac disease was found and left ventricular contractility was normal without any segmental wall motion abnormality. The patient was immediately transferred to operation room. At surgery a large thrombus was removed from the left ventricle. Patient was discharged with minimal residual neurological deficit and was symptom free during the follow up.

Discussion

The incidence of left ventricle thrombus in patient with end stage cardiomyopathy is 11 to 44% [5] and the annual risk of systemic embolization in patients with dilated cardiomyopathy is 1.4 to 12% [6]. A poorly contracting ventricle

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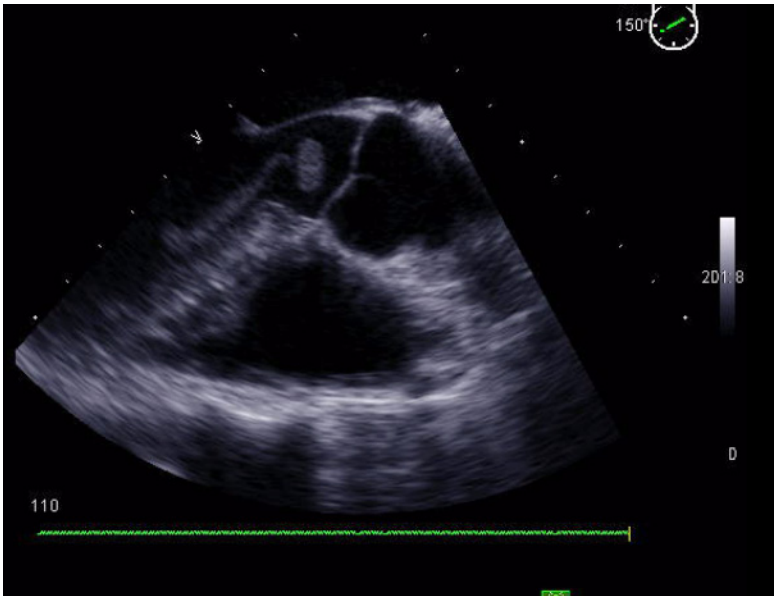

Figure 1. Trans-esophageal echocardiography in the long axis view showing a large, highly mobile thrombus resembling a fist, punching the aortic valves.

allows blood stasis, which can lead to thrombus formation and subsequent embolization. Mobile and protruding thrombi are thought to carry the highest risk. Ventricular thrombus is differentiated from a tumor, because the former is associated with abnormal regional wall motion, except apical thrombus noted in hypereosinophilic syndrome and occasionally, in lymphoma. Contrast echocardiography can be helpful in differentiating an apical mass from a thrombus. Thrombus is visualized as dark structure on contrast echocardiography, because it is not vascular. Thrombus formation in a normal functioning left ventricle has been reported previously,[7] but it is extremely rare.[4] In the present case, the patient had suffered from a large thrombus in left ventricle in the absence of any detectable heart disease. Also laboratory data such as antiphospholipid antibody, factor V leiden , and collagen vascular disease markers were normal. However, we were unable to check for some other rare coagulation disorders (antithrombin deficiency, plasminogen activator inhibitors, etc).

Acknowledgements

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