

Left ventricular mass: Myxoma or thrombus?

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ABSTRACT

Patient with embolic episode should always be evaluated for cardiac mass. Mass in left ventricular can be a myxoma or thrombus even in a normal functioning heart. In either case, mobile mass with embolic potential should be surgically resected.

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INTRODUCTION

Patient with embolic episode should always be evaluated for cardiac masses. Mass in the left ventricle (LV) can be a myxoma or thrombus even in a normal functioning heart. In either case, mobile mass with embolic potential should be surgically resected.^[1-3]

CASE REPORT

A 29-year-old male patient presented with left hemiparesis and dysarthria. Computed tomography scan revealed the embolic infarct in right middle cerebral artery territory. He was managed conservatively with an anticoagulant warfarin keeping international normalized ratio value between 2 and 3. Patient was evaluated and he was found to have pedunculated mass in LV on transthoracic echocardiography. He recovered gradually and was referred to our center for further management after 15 days of the diagnosis. Echocardiographic examination showed 0.9×1.7 cm homogenous mass present in LV toward apex attached to the interventricular septum (IVS). No LV regional wall motion abnormality was detected with LV ejection fraction of 65%. Patient had normal sinus rhythm without any arrhythmia or ischemia

changes in electrocardiogram. The mass was presumed to be myxoma and considering the potential for embolization, patient was scheduled for open heart surgery next day to remove the mass. After smooth induction and intubation, intraoperative transesophageal echocardiography was done. Mobile LV mass (size 1.1×1.8 cm) attached to IVS was seen [Figures 1 and 2, Video Clips 1 and 2]. Another LV mass attached to left ventricular outflow tract (LVOT) was noticed, but there was no LVOT obstruction as evident on insignificant LVOT gradient [Figure 3 and Video Clip 3]. After going on cardiopulmonary bypass, left ventriculotomy on arrested heart was performed on the anterior wall distally parallel to left anterior descending artery. Intraoperatively a pedunculated mass of size 2×1.5 cm attached to the IVS was seen. Another calcific fibrotic sessile mass of size 2×2 cm was observed on LV wall in outflow tract. Endocardium appeared to be thickened and fibrotic. After the surgery, patient was weaned off bypass smoothly. Resected mass was sent for histopathological examination. Microscopic examination revealed organized and calcified thrombus. No classical or diagnostic features of cardiac myxoma were observed. Patient was started on oral anticoagulants; however thrombophilia profile could not be evaluated due to noncompliance of the patient.

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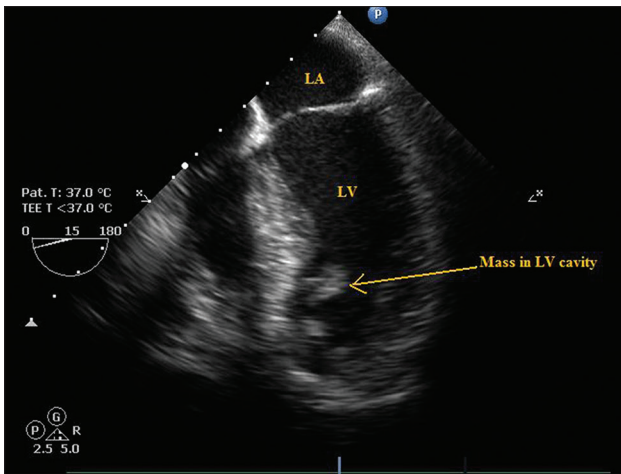


Figure 1: Midesophageal four chamber view of transesophageal echocardiography showing left ventricular mass

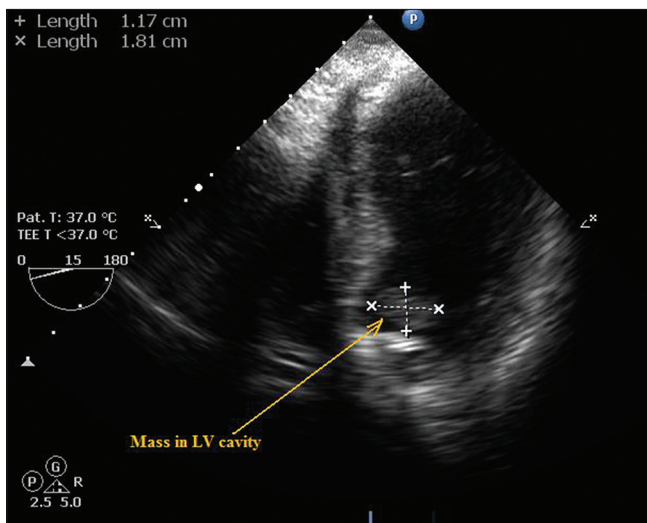


Figure 2: Transgastric short axis view of transesophageal echocardiography view lunar module mass

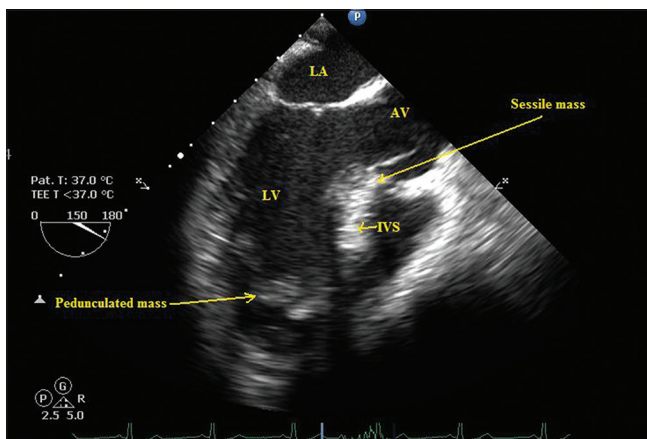


Figure 3: Midesophageal long axis view of transesophageal echocardiography showing two left ventricular masses

DISCUSSION

Primary cardiac tumors are uncommon with incidence

of 0.02%.^[1] Benign cardiac myxomas constitute 88% of cardiac tumor cases.^[2] LV myxomas account for only 2.5% of cases.^[2,3] The clinical features of LV myxoma are mostly caused by embolization and obstruction to LVOT. Arrhythmias, conduction disturbances, and LV dysfunction can also be seen.^[4-6] Embolic phenomena in LV myxoma are more common than LA myxomas, occurring in 64% of patients with LV myxoma.^[7] Considering the risk for embolization, myxomas should be surgically resected as early as possible.^[8]

Thrombus formation in LV is well-known complication in systolic heart failure (incidence 10–30%) and after acute myocardial infarction (incidence 5–15%).^[9,10] Hypercoagulable state or undetectable disorder of the endocardium can lead to ventricular thrombus formation even in the normal heart.^[11] Main causes of Inherited thrombophilia are G1691A mutation of factor V gene, G20210A mutation of the prothrombin gene, antithrombin deficiency, protein C and protein S deficiency.^[12] LV thrombus formation is also associated with antiphospholipid antibody syndrome and hypereosinophilic syndrome.^[13,14] Autoimmune disorders like Adamantiadis-Behcet's disease and lupus erythematosus, have been suggested to cause left ventricular thrombus formation.^[15,16] Large doses of alpha epoetin administration causing spontaneous LV thrombus formation has been reported. The rapid increase of hematocrit due to alpha epoetin has been suggested as the reason. Author has cautioned about the use of erythropoietin and hematocrit should not rise >30% above baseline within 10 days.^[17] Studies have reported that iron deficiency anemia has been implicated as a cause of systemic thromboembolism.^[18-20] Akins *et al.*^[21] suggested iron deficiency anemia induces turbulence by decreasing viscosity and increasing velocity and thrombosis may be caused by endothelial injury due to turbulent blood flow.

A pedunculated thrombus moving throughout the cardiac cycle has a high tendency to embolize despite adequate anticoagulation.^[22,23] Treatment for such thrombi has included thrombectomy, anticoagulation, or thrombolysis. Surgical removal by ventriculotomy may cause deterioration of LV function and potentially induce ventricular arrhythmia.^[24] Transaortic video-assisted removal of an LV thrombus, and also the trans-left atrial appendage and mitral valve approach, can be alternatives and can provide good LV visualization. However, video-assisted cardioscopy can be associated with serious potential complications.^[24]

Anticoagulation (high-dose intravenous heparin or low-molecular-weight heparin) has variable resolution rates 13–59%.^[25] Rester *et al.*^[26] have successfully used recombinant tissue plasminogen activator for lysis of a mobile, pedunculated LV thrombus in a patient with peripartum cardiomyopathy and evidence of systemic embolization. However, thrombolysis carries high risk of hemorrhagic or embolic complications.

In the present case, LV mass that was supposed to be myxoma preoperatively was found out to be a thrombus on histopathological examination. Outcome of patients with recurrent emboli from mobile, pedunculated thrombi who are treated conservatively is generally very poor compared with that of a limited number who undergo surgery.^[27,28] Pedunculated globular thrombi connected to the endocardium by a very narrow stalk and moving freely within the LV lumen carries 60–80% risk for embolization.^[29] Hence, such patients with a history of embolization should be treated surgically as early as possible. Causes for spontaneous LV thrombus formation in a normal heart should be evaluated thoroughly. Smooth anesthesia induction in patients with pedunculated cardiac masses will lessen the possibility of embolization. Real-time transesophageal echocardiography monitoring would help the surgeon in deciding the approach.

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