Case Report

Successful Excision of the Left Ventricular Thrombus in a Young Stroke Patient

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Abstract:
In this case report, a 29-year young male patient who presented with a history of right sided hemiparesis for 12 hours with no apparent cause. He showed no cough, palpitations, breathlessness, chest pain, syncope, or loss of vision. He had no relevant family history but had a history of two times admission in a National Neuroscience hospital for stroke. Echocardiography showed a freely mobile LV apical mass with hypokinetic apical septum and apex. He underwent successful removal of the mass via the LA roof. The postoperative TEE was normal. Histopathological examination of the resected mass revealed organized thrombus and patient had a satisfactory outcome.

Keywords: Left ventricle; Left ventricular mass; Transthoracic Echocardiography; Cardiopulmonary bypass

Introduction:
A patient with repeated history of stroke 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. [1-4]. Here we report a case of a left ventricular thrombus that was supposed to be myxoma preoperatively undergoing successful resection presenting as right sided hemiparesis.

Case Presentation
A 29-year-old young man was admitted to our hospital on February 21, 2023. He had a history of right sided hemiparesis for 12 hours with no apparent cause. He showed no cough, palpitations, breathlessness, chest pain, syncope, or loss of vision. He had no relevant family history but had a history of two times admission in a National Neuroscience hospital for stroke. Physical examination revealed right sided limb weakness. Computed tomography scan revealed the embolic infarct in left 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 LV mass on echocardiography. He recovered gradually and was referred to our center for further management after 15 days of the diagnosis. Echocardiographic evaluation showed freely mobile LV apical mass. Hypokinetic apical septum and apex. Normal LV systolic function with LV ejection fraction of 55-60%. Patient had normal sinus rhythm without any arrhythmia or ischemia. MDCT coronary angiography revealed unremarkable study of coronary arteries with thrombus in left ventricle. The mass was presumed to be myxoma and considering the clinical presentation of the patient and the potential for further embolization the patient was scheduled for an

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emergency. The pre-operative transthoracic echocardiography (TEE) showed freely mobile mass present in the left ventricle towards the apex (Figure 2). A median sternotomy was performed. The ascending aorta and both venae cavae were cannulated and standard cardiopulmonary bypass (CPB) was performed. The heart was stopped by cross-clamping the ascending aorta. Myocardial protection was achieved by means of intermittent anterograde administration of cold blood cardioplegia. The venae cavae were snared with tourniquets and via the LA roof. Then LV thrombus was identified, which was attached with LV wall by two connecting stalks. With care not to injure the other structure of the heart, the mass was completely removed and the tumor’s pedicle was completely shaved from the endocardium. The patient was weaned from CPB easily, without inotropic support. The postoperative TEE was normal. Resected mass was sent for histopathological examination (Figure 6). Microscopic examination
revealed organized thrombus. No classical or diagnostic features of cardiac myxoma were observed. The postoperative course was uneventful, the patient was put on anticoagulation treatment and on postoperative day 8 he was discharged home. However, thrombophilia profile could not be evaluated due to noncompliance of the patient. An echocardiography, performed at follow-up examination 7 days postoperatively and revealed no evidence of residual mass.

Discussion:
Cardiac tumors may be primary or secondary tumors. Primary cardiac tumors are rare, with a incidence of 0.02% (1). Benign cardiac myxomas constitute 88% of cardiac tumor cases and 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 left ventricular outflow tract (LVOT). Arrhythmias, conduction disturbances, and LV dysfunction can also be seen. [4-6] Embolic phenomenon 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 a known complication of heart failure and acute myocardial infarction (9, 11). Left ventricular thrombus formation in individuals with normal ejection fraction is a rarely seen phenomenon.

Very rarely, in a normal functioning LV has been reported in the presence of a hypercoagulable state (10). Main causes of inherited thrombophilia such as, factor V gene mutation, prothrombin gene mutation, antithrombin and protein C and S (12), and the presence of autoimmune disorders, such as antiphospholipid antibody syndrome (13), Behcet disease, lupus erythematosus (14, 15) and myeloproliferative disease, are all associated with LV thrombus formation in normal LV. In addition, other causes that lead to the formation of an LV thrombus in a normal LV are: idiopathic hypereosinophilic syndrome (toxic eosinophilic granules directly traumatize the endocardium), inflammatory bowel disease (platelet aggregation), pheochromocytoma (transient LV dysfunction) and Takatsubo cardiomyopathy (reversible LV dysfunction) (16).

The differential diagnosis of an LV mass between thrombus and myxoma can be challenging. However, there is no diagnostic feature, either by 2D-echocardiography or by direct inspection in which the diagnosis can be confirmed and either pathology may masquerade as the other (18). Histopathology is the final court of appeal and must always be performed, as in our report.

The surgical approach we chose to remove the mass in this location was through the LA roof, but can be carried also by other ways: (i) through the left atrium and mitral valve (18), (ii) through the ascending aorta with video assistance (19), and (iii) through a small longitudinal incision in the left ventricle (6). We chose the LA roof approach because it avoids ventriculotomy and its potential complications like deterioration of the LV function and bleeding, despite the fact that ventriculotomy provides good visibility and the possibility for complete resection. A potential disadvantage of the approach through the mitral valve would be limited room for maneuvering during the mass resection. Recently, endoscopy and minimally invasive techniques have also been applied.

In evaluation of clinical features, and laboratory results and in the absence of cardiac rhythm abnormality, left
ventricular dysfunction, malignancy or hypercoagulable state, it is not clear why the patient developed an LV thrombus. In these patients’ hypothesis of microvascular ischemia has been proposed as a triggering mechanism for the aggregation of platelets by means of inducing patchy areas of endocardial fibrosis and formation of thrombi in these endomyocardial areas (12). ECG in this patient was unremarkable, and he did not describe any episode of chest pain.

First line treatment for a LV thrombus is anticoagulation, although a mobile thrombus presented as an embolic complication, as in this case often requires urgent surgical resection.

In conclusion, we report an unusual case of LV thrombus leading to stroke. Early detection such as TEE and 2D echocardiography can be used and patients could acquire complete recovery after total thrombus resection.

References