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LEFT VENTRICULAR THROMBUS IN THE PRESENCE OF NORMAL LEFT VENTRICULAR FUNCTION

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ABSTRACT

Left ventricular (LV) thrombi in the presence of normal Ventricular function are uncommon. We report a 7-year old boy who presented with an LV thrombus in the setting of normal LV function identified by echocardiography, to highlight the rarity of this entity and its clinical significance. The thrombus disappeared after a few days of anticoagulant and steroid therapy without symptoms of embolization.

Key Words: Thrombus, Echocardiography , Embolization , Anticoagulant.

INTRODUCTION

Normal LV contractility is a major factor in preventing the formation of thrombus. But in case of impaired LV function the contractility is disturbed ,so chances of LV thrombi is common.

CASE REPORT

A 7-year-old boy was admitted with a transient ischemic attack (left hemiparesis). Echocardiogram showed a mobile LV mass 10×20 mm in size (Fig. 1).Initially it was reported as LV mass in periphry and was referred to Lady Reading Hospital for management. He was admitted in Paeds A unit and plan was made to review echo before MR Scan and cardiovascular surgeon consultation. His echo was reviewed and it was suspected that it is LV clot not LV mass . His differential blood count revealed 30% eosinophils with an absolute eosinophil count of 3000/cmm. The ESR was 25 mm in the first hour. Blood cultures, Antinuclear antibody (ANA), anticardiolipin antibody and VDRL tests were negative.He showed no evidence of parasitic, allergic or neoplastic illnsses. He was put on oral anticoagulants preceded by heparin and oral steroids. Echocardiogram repeated on day 6 of admission showed that the mass had disappeared. At 6-month follow-up, his eosinophil count was normal and repeat echocardiogram was absolutely normal. He has no residual neurological deficits.

DISCUSSION

Left ventricular thrombi is rare in the presence of preserved LV function, LV thrombi usually occurs in patients having impaired LV function. The most

common causes are dilated cardiomyopathy, LV aneurysm and following a myocardial infarction. Rarely, other causes which produce a thrombogenic milieu such as antiphospholipid antibody syndrome (APS)[1–4] and protein C deficiency[5] can lead to LV thrombus formation. Other conditions such as cardiac trauma, Salmonella septicemia, myeloproliferative disorders[6] and eosinophilic endocarditis[7-11] can give rise to LV thrombi. Reports of LV thrombi occurring without any obvious heart disease are also mentioned in the literature.[12]

Major thrombi can have irregular translucencies due to malacia. Furthermore, most thrombi are located near the apex as was seen in our patient. Thrombi can be of 3 types: (1) perimural, (2) protruding and (3) mobile; and thrombi may even be pedunculated. However, there is no diagnostic feature, either by 2-D echocardiography or by direct inspection, in which the diagnosis can be confirmed, and either pathology may masquerade the other .

Intracavitary echodense masses in the LV are considered to be thrombotic in nature. Full dissolution of the masses indicate that they are likely to be thrombotic. The size of the thrombus is likely to be more than the size measured on echocardiography, as only the central core is echogenic and can be measured.

Idiopathic hypereosinophilic syndrome is an entity where the eosinophils count is increased with tissue toxicity manifesting as multisystem involvement. The tissue damage is produced by a major basic protein, eosinophilic

Figure 1-A: Echocardiogram Showing LV Mass

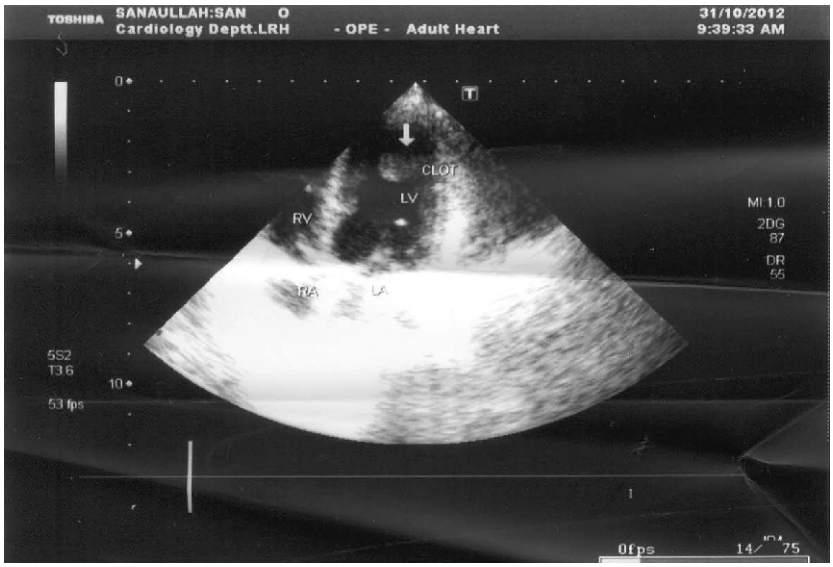


Figure 1-B

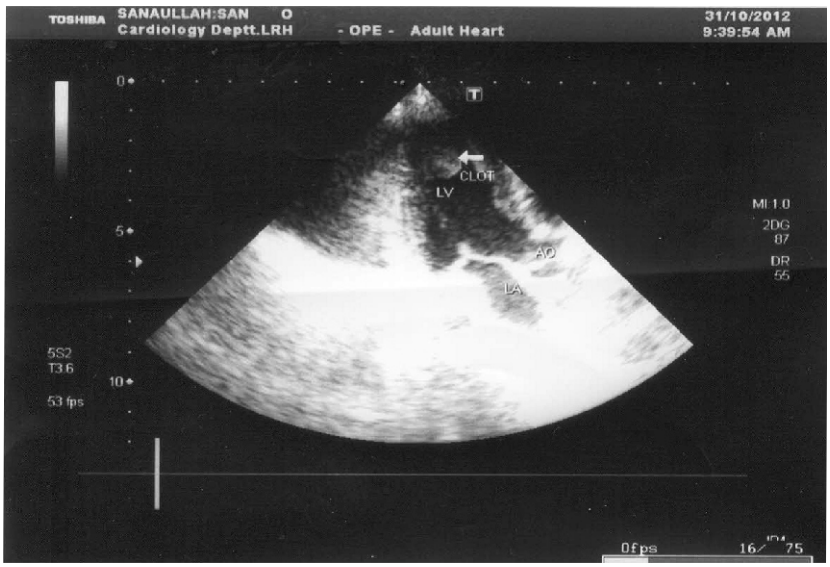


Figure 1-C

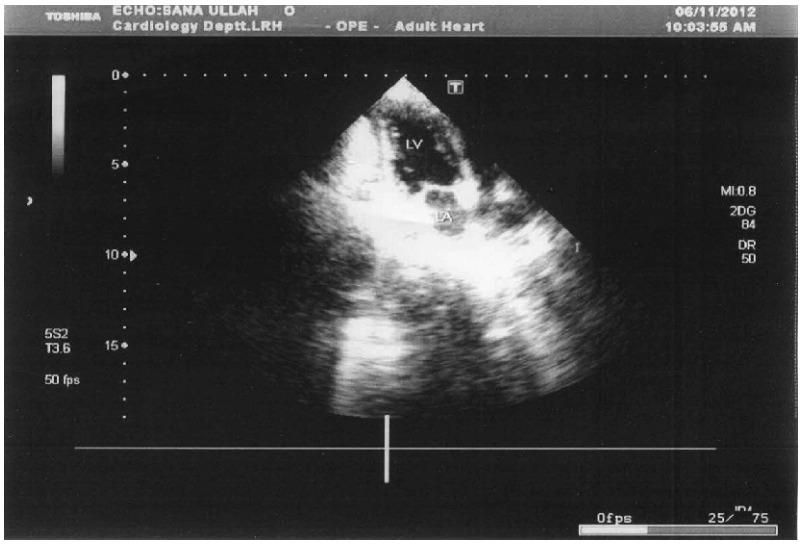


Figure 1-D

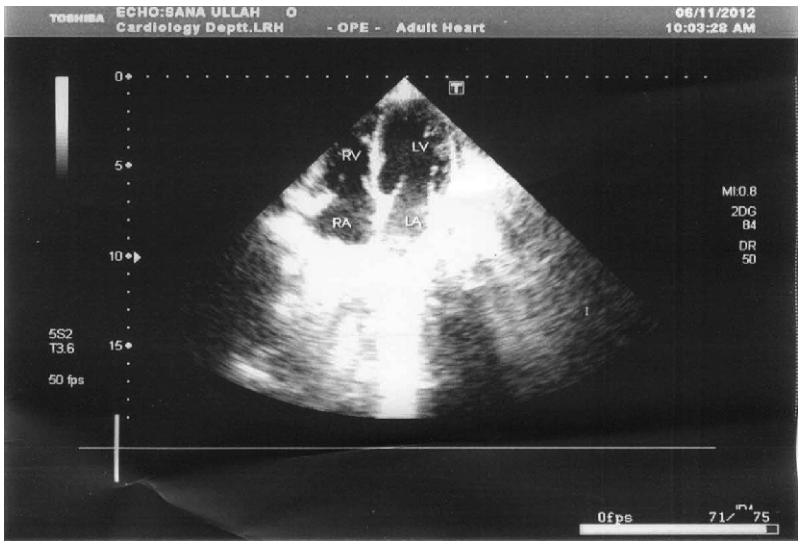
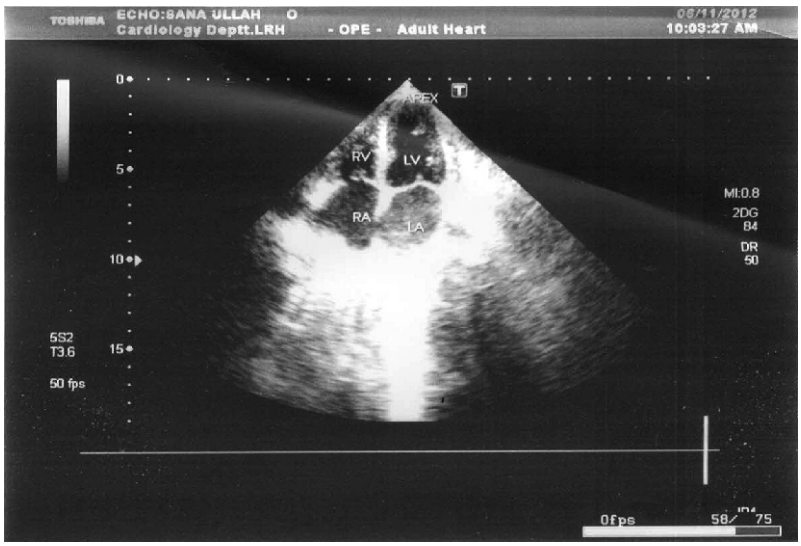


Figure 1-E



cationic protein, eosinophil peroxidase and eosinophil-derived neurotoxin present in the eosinophils.[7,8,10] A major cause of the morbidity and mortality due to this syndrome is the associated cardiac involvement. Typical cardiac findings include endocardial fibrosis and mural thrombus, which is most frequent in the apices of both ventricles. Valvular involvement is reported and cavity obliteration is seen in the later stages of the disease. The thrombus may extend up to the inflow tract of the atrioventricular valves, impede normal leaflet function and produce valvular regurgitation.[11]

Hypereosinophilic syndrome is usually treated with steroids which give good remission rates.[14] In patients who do not respond to steroids , immunosuppressives such as hydroxyurea and vincristine are the most commonly used drugs.[7–9] Our patient also responded well to steroids with regression of endocardial thickening and reduction in eosinophil count. In both the above cases, the thrombi disappeared after 3–4 days of anticoagulant therapy. Though early dissolution of thrombus has been reported,[15] it is more likely that fragments of the thrombi had embolized downstream without any clinical consequences.

There are no established protocols for management of these cases. Though dissolution with anticoagulant therapy is reported in the literature[16], risk of embolization is ever lurking. The rate of embolic episodes in mobile pedunculated thrombi is reported as high as 60%.[17]

CONCLUSION

LV thrombus with normal LV function can lead to thromboembolism and responds to anti coagulation as was reported in our case.

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