

Case Report

Left Ventricular Thrombus as seen on Echocardiography: From Evolution to Resolution

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INTRODUCTION

The association between acute myocardial infarction and thrombus formation is well known. We present one such case, which was followed up from the evolution (formation) to the near complete resolution and include a short discussion and literature review.

CASE REPORT

In September 1997, a 50-year-old deaf and mute male patient was admitted to the medical ward of our hospital with a right-sided hemiplegia. A computerized tomographic scanning of the brain revealed a cerebral infarction in the temporo-parietal region on the left side. The patient was a poorly controlled insulin dependent diabetic for the past 20 years. He had a recent extensive anterior myocardial infarction.

A transthoracic echocardiography (TTE) was done on the third day of admission and revealed a large highly mobile "gelatinous" echogenic mass in the left ventricle (LV). The mass was multi-lobulated and seemed to be attached with a pedicle to another thin, highly mobile, ribbon-like filamentous structure. This thin ribbon-like structure appeared, in the 2-dimension 4-chamber view, to be attached on one end to the interventricular septum (IVS) and the other end to the apical segment of the lateral wall of the LV. This echo image gave a very peculiar appearance of an encysted mass, not often seen on routine echocardiography. The shape and mobility of the lobulated mass was described as looking like a "uterine sac with a fetus" (Fig. 1). There was a large area of akinesia of the left ventricular wall involving the mid and apical segments of the IVS and the apical segment of the lateral wall. The LV ejection fraction was calculated as 35%. A diagnosis of a left ventricular thrombus (LVT) in early evolution was considered and immediate anticoagulation with intravenous heparin was started.

A planned transesophageal echocardiography (TEE) could not be carried out as the patient's condition did not permit it and, instead, a TTE was performed nearly a week after the first echo. This echo revealed a very different picture from the first one and the large mobile mass described above had changed itself into a well-formed thrombus attached, not mobile, to the mid and the apical segments of the IVS. (Fig. 2). Even the lateral wall of the LV had also taken up a part of the initial highly mobile gelatinous structure. The patient was continued on intravenous heparin for approximately 10 days followed by an adjusted dose of warfarin to maintain an international normalized ratio of 2.5 to 3.0.

The patient improved clinically with additional anti-ischemic treatment along with physiotherapy for the neurological deficit. Another TTE was done nearly a month from the first echo and, interestingly, it showed a significant resolution of the LV thrombus and only a very small, round, fixed, echogenic shadow was seen at the apex of the LV (Fig. 3). The patient was discharged and he did not come for a further follow-up.

DISCUSSION

Inflammation of the endocardium resulting from myocardial necrosis in any location may produce layered mural thrombus^[1,2]. More extensive thrombi with protruding appearances are at increased risk for systemic embolization^[2,3]. Echocardiography has been instrumental in refining our understanding of the pathophysiology of LVT and is a useful tool in the identification of patients who would benefit from continued anticoagulation and assessing the status of the LVT by serial echocardiographies^[4].

Left ventricular mural thrombus is a well-recognized consequence of acute anterior myocardial infarction (AMI). LVT formation occurs often and early after AMI, even when a thrombolytic agent has been given and delayed



Fig. 1: 2-D Four-chamber view shows a large, highly mobile, multiculated, gelatinous mass at the LV apex, occupying nearly a third of LV. A filamentous ribbon-like structure is also seen as if attached from the lateral wall to the IVS

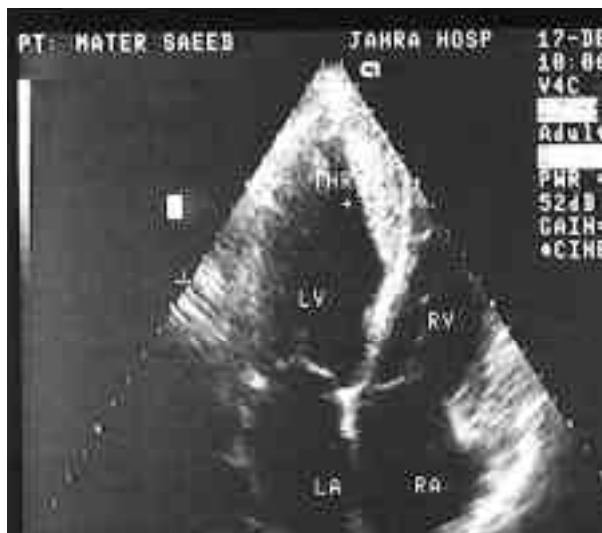


Fig. 2: 2-D echo repeated a week later shows the mass seen in Fig. 1, becoming well-formed and attached to the mid and apical segments of the IVS and also to the apical segment of the lateral wall.

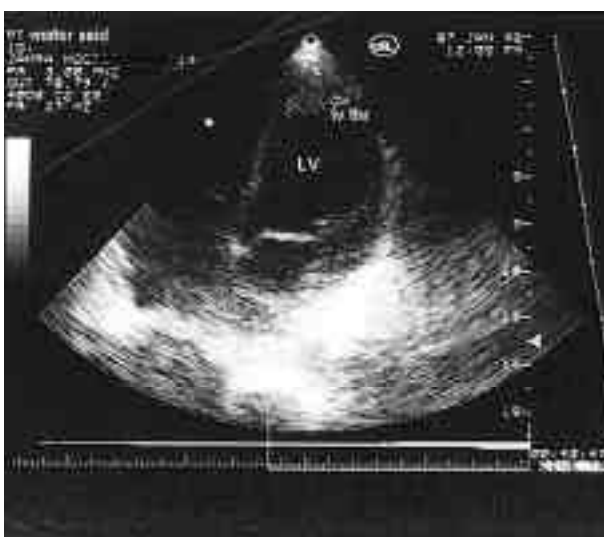


Fig. 3: 2-D echo repeated nearly one month later shows a significant resolution of the LV thrombus. A small, round, non-mobile echogenic shadow is seen in the LV apex.

LVT formation is associated with wall motion deterioration^[5]. The use of thrombolytic agents has, however, reduced the risk of embolic complications^[5,6].

The highest rate of occurrence of LVT was found among patients with anterior MI and an LV ejection fraction of less than 40%. In a recent study, a Killip class of more than 1 and the early administration of intravenous beta-blockers were the only variables independently associated with a higher pre-discharge incidence of LVT after AMI^[7]. Pre-thrombolytic era reports on the incidence of LVT is reported between 20 and 56% but an ancillary study to the Healing and Early After-load Reducing Therapy (HEART) study assessed the prevalence of LVT as only 0.6% at day 1, 3.7% at day 14, and 2.5% at day 90^[8].

The role of activated protein C resistance (APC-R) is under study as a possible predictor of those who would develop LVT. In a recent study, smoking and APC-R were significantly greater in those with LVT than those without. Multivariate regression analysis showed APC-R as an independent risk factor for LVT, whereas protein S and antithrombin III concentrations were not significantly different in the two groups^[9]. The mainstay of treatment is anticoagulation and it leads to a successful resolution in a large number of cases. In a study carried out to follow the course of LVT, echocardiography was done serially at day three of admission, then before patient discharge, and again at one, three and twelve months after discharge. The results were that at discharge only 30% had the LVT still seen and at one, three and twelve months 81%, 84% and 90% of LVTs respectively had resolved completely^[10].

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