Original article

A CASE SERIES STUDY OF 26 SUBJECTS WITH LEFT VENTRICULAR THROMBUS - TO SEE ITS ETIOLOGY AND LEFT VENTRICULAR FUNCTION

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ABSTRACT:

Objective: To evaluate the left ventricular function and determine etiology of left ventricular thrombus in 26 cases.

Design: Prospective observational study.

Setting: Medical Unit of Ayub Teaching Hospital, Ayub Medical College, Abbottabad.

Patient and Method: Patients with left ventricular thrombi diagnosed with echocardiography from May 1999 to June 2001 were studied for its etiology and left ventricular function.

Results: Fifty-six percent of left ventricular thrombus cases were due to chronic ischemic heart disease while 36% had dilated cardiomyopathy. Left ventricular aneurysm was seen in 7.8% cases. Mean age was 54±3 years with 74% males and 26% females.

Mean ejection fraction was 22 ± 2.5% in dilated cardiomyopathy cases while it was 35 ± 17% in cases with underlying coronary artery disease.

Conclusion: Chronic ischemic heart disease and dilated cardiomyopathy are the main causes associated with left ventricular thrombi. Impaired left ventricular function is a common denominator in these cases.

KEY WORDS: Left ventricular thrombus, etiology, left ventricular function.

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INTRODUCTION

Left ventricular thrombi are known to occur in a variety of cardiovascular conditions. In the past cardiac thrombi were suspected in retrospect following clinical embolic events. With the development of angiography cardiac thrombi could be diagnosed antemortem but with low resolution.[1]

With the introduction of echocardiography it became clear that antemortem diagnosis of intracardiac thrombi has become a reality and it could be diagnosed as a routine and with confidence through non invasive means with sensitivity and specificity of nearly 90%.[2]

We commonly encounter cases with cerebral thromboembolism or sudden limb ischemia

secondary to arterial embolism from cardiac source. Left ventricular thrombi can be picked up even before arterial embolic event has occurred and has enabled preemptive anticoagulant therapy to forestall the embolic arterial event.

We studied 26 cases of left ventricular thrombi over a period of two years to see the underlying cause and study left ventricular systolic functions in these cases.

PATIENTS AND METHODS

Twenty-six patients having left ventricular thrombi picked up by echocardiography were included in this study. Patient with left atrial, right atrial and right ventricular thrombi were excluded. Age and gender was noted. Subjects were clinically evaluated to determine the underlying disease. The examination included history, physical check up, chest radiograph, ECG, and transthoracic dopplar echocardiography with Toshiba SSH-140A system. Left ventricular systolic and diastolic dimensions were noted. Left ventricular ejection fraction was determined in each case. In brief, the etiology and left ventricular functions were determined in cases with left ventricular thrombi.

RESULTS

Nineteen out of 26 cases with LV thrombi were males (74%) and 7 (27.3%) were females. Mean age was 54±3 years (Table-I).

Dilated cardiomyopathy was seen in nine patients (36.1%), while IHD was the cause in fifteen patients (56%) cases. Left ventricular aneurysm was the underlying cause in two patients (7.8%). Mean LV systolic and diastolic dimensions in dilated cardiomyopathy was 71±4 & 60±9 mm respectively. Mean ejection fraction was 22.7±2.5. Mean LV systolic & diastolic dimensions in cases with IHD were 67.4±5.5 and 55±11.5 mm respectively with ejection fraction of 35±17 (Table-II).

TABLE-I
Mean age 54±3 years according to etiology

Disease	*Mean Age (years)*
Dilated CMP	51.7±6.5
IHD	55.6±13.5
LV aneurysm	54±1

TABLE-II
LV function in 26 cases of LV thrombi

Disease	*Mean diastolic Dimension (mm)*	*Systolic Dimension (mm)*	*EF*
Dilated CMP	71±4	60±9	22.7±2.5
IHD	67.4±5.5	55±11.5	35±17
LV aneurysm	66.5±4	54±10	34.8±13

DISCUSSION

Left ventricular thrombi are seen in the setting of transmural myocardial infarction, left ventricular aneurysm and dilated cardiomyopathy. Identification of the presence of left ventricular thrombi is of major importance as it can predispose to catastrophic arterial events like cerebrovascular accidents and sudden limb ischemia so commonly encountered clinically.[3]

The incidence of systemic embolism was 12, 27 and 50% in cases of acute myocardial infarction, dilated cardiomyopathy and left ventricular aneurysm respectively.[2,6] Mean age of 54 years points to the fact that left ventricular thrombi are predominantly a complication of heart diseases in the aging population. The underlying chronic ischemic heart disease increases in incidence with increasing age.

High male to female ratio also points to the gender bias seen in coronary artery disease, which is the major predisposing factor for the left ventricular thrombi (Table-I).

Left ventricular thrombi are seen in 20 to 30% cases of acute myocardial infarction especially with large anterior infarcts, with left ventricular hypokinesia, atrial fibrillation and low ejection fraction[4]. It is seen in 60% cases of autopsies of dilated cardiomyopathies.[5]

Mean ejection fraction in our series in cases of chronic ischemic heart disease was 35 ± 17%, while it was 22.7 ± 2.5% in patients with dilated cardiomyopathy (Table-II). Impaired left ventricular systolic function appears to be a common denominator of left ventricular thrombus formation. Two dimensional echocardiography is the investigation of choice where cardiac emboli are suspected clinically and it has 90% sensitivity.

Low cardiac output and intracardiac stasis predispose to mural thrombus formation.[7] Left ventricular aneurysm meets both these conditions, hence it is a risk factor for persistence and recurrence of left ventricular thrombi during acute myocardial infarction. Systemic emboli are experienced in 10% of such cases within three months of acute Myocardial infarction.[4]

Left ventricular thrombi may be stationary or mobile. It has indistinct or clearly defined margins. It may appear solid or cystic. At times it is so laminated and marginated that it appears as localized thickening of left ventricular wall.[8]

Dilated cardiomyopathy is known for harbouring large, diffuse or multiple thrombi. Clot lysis leads to central lucency with cystic appearance. Protruding mobile thrombi are more likely to embolize. Contiguous areas of akinesia and hyperkinesias also encourage embolization. Textural analysis may identify thrombi prone to embolization.[9] The risk of embolization is more with non ischemic dilated cardiomyopathy as compared to chronic ischemic heart disease. It is also more with atrial fibrillation and with impaired left ventricular function, as is clearly evident in the present study.[10] The rate of embolism is low in chronic left ventricular aneurysm because of the clot organization and its sequestration from the main stream.[4]

Anticoagulation prevent and reduce the rate of systemic embolization and reduce the risk of death in cases of left ventricular thrombus.[11] Post myocardial infarction cases with left ventricular thrombi with left ventricular dysfunction or with history of thromboembolism need long term anticoagulation (3-6 months) with warfarin and aspirin with INR of 2-3.[12]

Because of low risk of embolism in chronic LV aneurysm anticoagulation is not advised beyond first three months of acute M.I.[12] Heparin therapy during the acute M.I. reduce the incidence of LV thrombus formation by 50%.[13]

Patients with dilated CMP pose greater risk of thromboembolism than patients with IHD. In the later, congestive heart failure may not be a sufficient indication for anti coagulation unless there is atrial fibrillation, previous thromboembolism or demonstration of LV thrombus on echocardiography.[14] Anticoagulation also alters the intracoronary thrombi favourably in cases of IHD.[15]

Thrombolysis reduce the rate of thrombosis but may precipitate arterial embolism by lysing the otherwise well anchored thrombus.[16] Anticoagulation can be discontinued after 3-6 months even after the residual thrombus persists since clot organization renders further fragmentation unlikely.[17] Protracted course of anticoagulation is indicated in case of LV thrombus enlargement or recurrence of thrombus formation.[18]

Presence of LV thrombus alters the clinical status of the patients and keeps the physician alert. Preemptive measures avert the catastrophic complications of arterial embolism seen in the form of stroke and limb ischemia. Two dimensional echocardiography plays unrivalled role in the management of patients harbouring LV thrombi.

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