LEFT VENTRICULAR THROMBUS IN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Objective: To determine Left ventricular thrombus (LVT) in acute Myocardial infarction (MI) and to find out the correlation of risk factors with development of LVT.

Study Design: Hospital based observational study.

Place and Duration: Department of Cardiology, Liaquat University Hospital Hyderabad between November 2005 to November 2006.

Patients and Methods: 280 consecutive patients presented with first episode of acute Myocardial infarction were included. Patients with previous history of Myocardial infarction, rheumatic heart disease, dilated cardiomyopathy and mural thrombus were excluded. Baseline characteristics were recorded on the proforma. Two dimensional echocardiography was performed on day 3, at the time of discharge, 3 and 6 months after infarction. Two echo cardiographers blinded to clinical details separately reviewed the echo images. Descriptive and inferential statistical analysis was performed using SPSS version 16.0.

Results: Two hundred and eighty patients with first episode of acute MI were studied; 214(76.4%) were male and 66(23.6%) were female. Mean age of patients was 54.08 ± 11.9 SD. Left Ventricular Thrombi (LVT) was found in 50/280 (17.86%) patients detected by 2-D echo method. 3(6%) patients died while in the coronary care unit and 7 (14%) with LVT failed to follow up. In remaining 14/40 (35%) patients' thrombus once detected, was present during the entire echocardiographic follow up and became organized. However in 26/40 (65%) patients thrombus disappearance was noted on follow up echocardiographic studies. Only 3/50 (6%) patients had complication of systemic embolization, all in the CNS. Among risk factors only smoking and Diabetes Mellitus were found to be statistically significant. LVT was seen in patients with decreased left ventricular wall motion especially anterioapical wall akinesia.

Conclusion: LVT is important complication of acute myocardial infarction. If diagnosed and anticoagulated earlier, further risk of complications and its potential to embolize can be minimized

Key Words: Left ventricular thrombus, acute myocardial infarction, Complications of LVT.

INTRODUCTION

Although there has been a dramatic decrease in age adjusted death rates due to ischemic heart disease(IHD) and Myocardial infarction(MI) since 1950, it still remains responsible for most years of life lost before age 65, regardless of gender or race[1]. This decrease in mortality probably reflects advances in management of risk factors and aggressive treatment modalities of thrombolysis and active interventional techniques. There remains a certain sub group of patients who are prone to have adverse outcome.[1, 3] The occurrence of left ventricular thrombus (LVT) after acute myocardial infarction (AMI) carries a significant prognosis implication. Left ventricular thrombus (LVT) occurs early after Acute Myocardial Infarction (AMI) even when thrombolysis has been done. Generally the location is in the left ventricle (LV), particularly at the apex but it can occur along

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the septum[2, 3] Left ventricle thrombus (LVT) occurs much more frequently (98%) in anterior infarcts as compared to infarcts present at other areas of myocardium(2%) and in larger infarcts as compared to smaller one.s[1,3,4]

A mural thrombus which adheres to the endocardium overlying the infarcted myocardium, a superficial part of it can become detached and produce fatal systemic arterial emboli.[1, 6]

Left ventricular thrombi carry certain risks due to its potential to embolize to various organs of the body; causing blockage of vessels (arterial system) of these organs especially brain and kidney are of great concern; because of the lethal consequences of the cerebrovascular accidents and hypertension respectively. The echocardiographic features suggesting most likely embolization of a thrombus are(1)Increased mobility, (2)Protrusion of thrombi into ventricular cavity, (3)When the thrombus is visualized in multiple views and continuous zones of akinesia and hypokinesia of left ventricular wall. The myocardial infarction is not the sole cause of left ventricular thrombus.

There are many other causes like dilated Cardiomyopathy, endomyocardial fibrosis, Loeffler's disease, eosinophilic leukemia's and myocarditis. The most concerning aspect regarding left ventricular thrombus is not its mere presence but its potential to systemic embolization. The aim of our study is to find out Left ventricular thrombus in acute Myocardial infarction and the correlation of risk factors with development of LVT.

PATIENTS AND METHODS

This was a hospital based observational study in which we examined a total no: of 301 consecutive patients of acute myocardial infarction admitted in the Cardiology Department, Liaquat Medical University Hospital Hyderabad during 6 months period November 2005 to November 2006. Patients with first Q-wave Acute MI, ECG evidence of ST-Elevation >2mm at least in two contagious chest leads, Chest pain lasting >30 minutes and Transient elevation of Creatine Kinase and/or MB isoenzyme were included in the study. Patient with Rheumatic Heart Disease and Dilated Cardiomyopathy, prior history of Mural Thrombus formation, histories of Systemic Embolism and with End stage or Chronic Debilitating Disease were excluded. Twenty- one patients were excluded from study, out of which thirteen patients died during hospital stay and eight patients failed to come for follow up. A detailed history of the current cardiac problem, time of onset, duration of illness, past and family history of coronary artery disease was taken from the patient. The history of common risk factors like smoking, job description, life style, eating habits, diabetes mellitus and hypertension was also recorded. A 2-D echocardiogram examination was performed on Toshiba Nemio 35 with color Doppler. Patients were serially evaluated by two-dimensional and Doppler echocardiography in the following sequence: on day 3, at the time of discharge, 3 and 6 months after infarction. The diagnosis of left ventricular thrombus was made when an echo dense mass with a margin distinct from the left ventricular wall was detected within the left ventricular cavity and was visible throughout the cardiac cycle in at least two different echocardiographic views and associated with asynergy (akinesis or dyskinesis) of the adjacent myocardium. The echo study was independently reviewed by two observers' one trained doctor and one consultant; both were blind about the clinical data. Serial ECG was done to look for the evidence of acute myocardial infarction as S-T segment elevation along with pathological Q-wave of 0.04 second wide and 2 mm in height.

DATA ANALYSIS PROCEDURE

The chi-square test of independence was applied to analyze the data for results. Normal P value of significance was considered as 0.05. All the data was analyzed using SPSS 16.0 (version).

RESULTS

Left Ventricular Thrombi (LVT) was found in 50/280 (17.86%) patients detected by 2-D echo method and 230/280 (82.14%) patients were without any evidence of LVT. Most of thrombi were located in the left ventricular apex and almost all were detected within the first week after infarction. The over all incidence of LVT was 17.86%, higher among patients of acute anterior myocardial infarction 45/138 (32.6%) as

compared to NSTEMI and Inferior + RV MI i.e. 4/66 (6%) and 1/33 (3.0%)respectively. In this study male out numbered females and mean age of 54.08 ± 11.9 SD. LVT was present in 40/214 (18.7%) in male and 10/66 (15.1%) female.

All patients received anticoagulant therapy on admission and throughout study period. Out of 50, 3(6%) patients died while in the coronary care unit (because of arrhythmias and left ventricular failure) and 7 (14%) with LVT failed the follow up after one month. In remaining 14/40 (35%) patients' thrombus once detected, was present during the entire echocardiographic follow up and became organized. However in 26/40 (65%) patients thrombus disappearance was noted on follow up echocardiographic studies. Only 3/50 (6%) patients had complication of systemic embolization, all in the CNS. Presence of LVT and systemic embolization was found to have a significant correlation. We also found that the patients who showed echocardiographic evidence of LVT were found to have significant reduction in ejection fraction and left ventricular wall akinesia and dyskinesia.

Of the other risk factors only smoking and Diabetes Mellitus were found to be statistically significant. Smoking history was positive in 40/50 (80%) with p value = 0.03 and Diabetes Mellitus in 33/50 (66%) patients with p value = < 0.001.

LVT was seen in patients with decreased left ventricular wall motion especially anterioapical wall akinesia. 33/50 (66%) were found with anterior and apical wall akinesia, 10/50 patients (20%) with apical and septal akinesia, 6/50 (12%) with akinetic septum, dyskinetic apex and hypokinetic anterior wall and 1/50 (2%) with akinetic septum, inferior and right ventricular wall.

Among complications, embolic phenomena occur only in patients with LVT, left ventricular failure and mitral regurgitation occur higher in patients with LVT as compared to those without, while other complication occur equally in both with and without LVT.

DISCUSSION:

Several issues should be considered regarding the clinical importance of left ventricular thrombus after acute myocardial infarction. First, higher mortality has been reported in patients with left ventricular thrombi after infarction[7], especially when these develop within the first 48 h after infarction. Second, left ventricular thrombi may have embolic potential[8, 9, 10, 11].

Left ventricular thrombus is a dreadful complication of myocardial infarction, especially of extensive acute anterior myocardial infarction. A vast study to highlight various aspects of LVT is going to be conducted all over the world but work in Pakistan is limited and needs to be further evaluated. Though LVT itself is dangerous but its clinical importance lies in its potential to embolize to various organs, resulting in stroke and limb ischemia. Various aspects of its embolization and its prevention are studied. Many researchers agree that although thrombolytic therapy does not prevent LVT formation but has significantly reduced the incidence of embolization. Although Illeri M[12] and many others also reported that a better earlier reperfusion had resulted in decreased incidence of LVT formation secondary to myocardial infarction. Nadeem M A[13] reported cardiac failure in 18.74% of patients who were thrombolysed, compared to 57.1% of patients who did not receive thrombolytic therapy. Carillo[5] have reported that dissolution of left ventricular mural thrombus with oral anticoagulants was significant with a p value of

Figure-I : Case Distribution according to type of Infarction (n = 280)

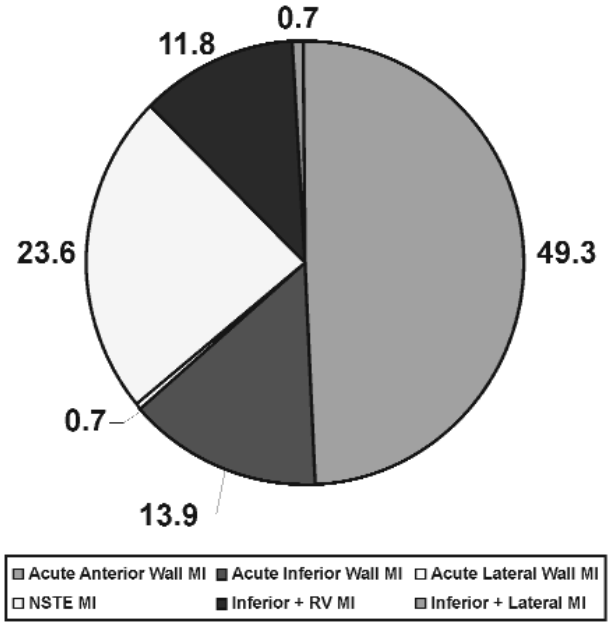

Table-I : Left Ventricular Clot (n = 40)

LEFT VENTRICULAR CLOT	PRESENT / ORGANIZED (n = 14)	ABSENT / RESOLVED (n = 26)	P VALUE
APICAL	12 (30%)	26 (65%)	0.04
SEPTAL	02(5%)	0	

my patients with LVT had systemic embolization, in all three patients emboli occurred into central nervous system; this may reflect the low overall embolic potential of left ventricular thrombi after infarction. However pooled data showed that arterial embolic events occurred in 18% patients with left ventricular thrombus and that the risk is especially high in those with mobile and protruding thrombi[14]. Of the

Table-II : Complications

COMPLICATIONS	WITH L.V.T (n = 50)	WITHOUT LVT (n = 230)	P VALUE
LVF	10 (20.0%)	23 (10.0%)	0.04
CARDIOGENIC SHOCK	5 (10.0%)	23 (10.0%)	1.0
MITRAL REGURGITATION	3 (6.0%)	9 (3.9%)	0.50
VENTRICULAR SEPTAL RUPTURE	1 (2.0%)	5 (2.2%)	0.93
POST INFARCTION ANGINA	14 (28.0%)	65 (28.3%)	0.97
PERICARDITIS	2 (4.0%)	9 (3.9%)	0.97
RIGHT VENTRICULAR FAILURE	1 (2.0%)	23 (10.0%)	0.06
EMBOLISM	3 (6.0%)	0	<0.001*
LEFT VENTRICULAR ANEURYSM	2 (4.0%)	5 (2.2%)	0.45
ARRHYTHMIAS	6 (12.0%)	23 (10.0%)	0.67
HEART BLOCK	7 (14.0%)	34 (14.8%)	0.88

** P value is highly significant*

Table-III : Relation of Ejection Fraction and LVT formation (n = 50)

%AGE OF LVEF	NUMBER	%AGE OF L.V.T FORMATION
<25%	24	48%
25% – 40%	26	52%

<0.05. Kotny[3] also agrees with Carillo that thrombus resolution occurs frequently with anticoagulation without any associated embolic risk. In our study LVT was found in 50 (17.86%) patients in all types of myocardial infarction. The frequency was high in acute anterior myocardial infarction. 14/40 (35%) patients on follow-up echocardiographic study showed constant presence of organized thrombus whereas in 26/40 (65%) patients disappearance of thrombus was noted on echocardiography. Three of

predictors for causing LVT formation the most frequent is apical and anterior wall akinesia/ dyskinesia. Smoking and Diabetes Mellitus were major risk factors which significantly affected the LVT formation.

CONCLUSION:

Our study has led to the conclusion that LVT is important complication of acute myocardial infarction. If diagnosed and anticoagulated earlier, further risk of complications and its potential to embolize can be minimized. The echocardiography is very sensitive and specific test in detection of LVT in acute MI patients. Echo should be done of every patient presenting with acute MI at the time of admission and discharge.

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