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INCIDENCE OF LEFT VENTRICULAR THROMBUS IN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

OBJECTIVE: *To determine the incidence of Left ventricular thrombus (LVT) following first episode of acute Myocardial infarction (MI).*
STUDY DESIGN: *Cross sectional observational study*
PLACE AND DURATION: *Department of Cardiology and department of Medicine, Liaquat University Hospital Hyderabad/Jamshoro and Isra University Hyderabad between January 2007 to June 2007.*
PATIENTS AND METHODS: *300 consecutive patients presented with first acute Myocardial infarction were included. 20 pts died/did not followed in the study. 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, 5 and 6. 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; 187(66.8%) were male and 93(33.2%) were female. Mean age of patients with LVT was 56.56 + 11.29 vs those without LVT 53.43 + 11.7. On echocardiographic examination, 50(17.86%) patients had LVT, of those who developed LVT, 45(90%) had anterior wall MI, 4(8%) had NSTEMI. 166(59.3%) patients were given intravenous streptokinase infusion; 19 of them developed LVT, among 114(40.6%) patients who were not thrombolyzed, 31 developed LVT (P value 0.001). Extensive antero apical asynergy with apical dyskinesia was present in 39/45(86.7%) with anterior wall MI and LVT vs 20/93(21.5%) with anterior wall MI and without LVT.*
CONCLUSION: *It is concluded that patients with acute MI should be given thrombolytic therapy as earlier as possible and proper measures should be taken to control the risk factors like Hypertension and DM, so that incidence of LVT and its associated complications can be minimized.*

KEY WORDS: Left ventricular thrombus, Acute myocardial infarction, Thrombolytic therapy.

INTRODUCTION:

Left ventricular thrombus (LVT) is common and well recognized complication of acute myocardial infarction (AMI). In survivors of infarction its incidence is influenced by the location and magnitude of infarction. Historically the incidence of LV thrombus complicating AMI had been reported to be 20-40 % and may reach 60% among patients with large anterior wall MI[1]. It occurs much frequently (98%) in large anterior Q-wave AMI as compared to infarcts present in other areas of myocardium[2-4].

Causes of LV thrombus include segmental dysfunction of infarcted myocardium (hpokinesia/ akinesia) causing status, endocardial tissue inflammation providing a thrombogenic surface and a hyper coaguable state[5-10]. The resultant LV thrombus is composed of fibrin, red blood cells and platelets. However, not all types and locations of MI have the same propensity to develop LVT. It is primarily a complication of acute anterior transmural MI, particularly those involving thee apex[11, 12, 13.] Apical stasis associated with specific abnormal flow pattern is in fact predictive of subsequent thrombus formation[14].

Serial echocardiographic studies have demonstrated that LVT develops primarily in the first week following MI, when the hypercoagulable state and endothelial injury are most intense[11, 12, 13]. Thrombi developing later in the course of infarction are most commonly associated with adverse remodeling, including left ventricular dilatation, reduced global

function and aneurysm formation[12]. Of all LV thrombus visualized echo cardio graphically, more than half seen within 48 hrs of the event, 80% within first week and almost 90% are seen within 2 weeks. Occasionally LV thrombus is unidentified until >1 month after AMI[15]. A mural thrombus adheres to the endocardium overlying the infracted myocardium and superficial portion of it can become detached and produce fatal systemic emboli[1, 2, 10]. The clinical importance of left ventricular thrombus (LVT) lies in the potential for systemic embolization particularly stroke. The reported incidence of ischemic stroke is 6% from mural thrombus originating from LVT [16].

PATIENTS AND METHODS:

This cross sectional observational study was conducted at department of Cardiology and department of Medicine, Liaquat University Hospital Hyderabad/Jamshoro and Isra University Hyderabad between January 2007 to June 2007. 300 consecutive patients presenting to our institute with first acute Myocardial infarction who met the following 1) Age < 70 years, 2) Chest pain lasting > 30 min, 3) ST segment elevation > 2 min in at least 2 electrocardiographic leads, 4) Transient elevation of creatine kinase or CK-MB isoenzyme were included. Patients with previous history of Myocardial infarction, rheumatic heart disease, dilated cardiomyopathy and previous history of mural thrombus / systemic embolization were excluded. Written informed consent was obtained from the all patients prior to enrolment in the study. Baseline characteristics were recorded on the proforma. Twenty pts died/did not followed in the study. Two dimensional echocardiography was performed using Toshiba Nemio machine by registered sonographer (echo technician) on each patient on day 3, 5 and 6. Two echo cardiographers blinded to clinical details separately reviewed the echo images of each patient. LV thrombus was defined as an echo dense mass with definite margin contagious but distinct from the endocardium adjacent to an area of hypo or akinetic myocardium and identifiable throughout the cardiac cycle. Asynergy was defined as hypokinesia, akinesia, or dyskinesia of the myocardium.

STATISTICAL ANALYSIS:

The data were analyzed in statistical program SPSS version 16.0. Fisher's exact test of chi-square was applied for categorical variables and Independent samples *t* test was performed for continuous variables to calculate mean and standard deviation. P value <0.05 was considered as statistically significant.

TABLE-I
BASIC CHARACTERISTICS OF PATIENTS AMONG GROUPS.

	Total (n=280)%	With LV clot (n=50)%	Without LV clot (n=230) %	P value
Age(Yrs) Mean ± SD	53.99 ± 11.67	56.56 ± 11.29	53.43 ± 11.7	0.08
Peak Ck-MB Level, Mean ± SD	111.2 ± 105	159.7 ± 127.7	100.69 ± 9.15	0.009
Age(Yrs) in Groups				
30-40	58 (20.7)	6 (12)	52(22.6)	
41-50	81(28.9)	10(20)	71(30.9)	
51-60	95(33.9)	21(42)	74(32.2)	0.02
61-70	28(10)	10(20)	18(7.8)	
70 and above	18(6.4)	03(06)	15 (6.5)	
Sex				
Male	187(66.8)	40(80)	147(63.9)	0.031
Female	93(33.2)	10(20)	83(36.1)	
Hypertension	109(38.9)	26(52)	83(36.1)	0.028
Diabetes Mellitus	122(43.6)	28(56)	94(40.9)	0.05
Smoking	188(67.1)	40(80)	148(64.3)	0.045
Obesity	76(27.1)	21(42)	55(23.9)	0.009
Family H/O CAD	54(19.3)	11(22)	43(18.7)	0.35

TABLE-II
RELATIONSHIP B/W INFARCT LOCATION AND THROMBUS FORMATION (N=280)

Infarct location	No of Patients (n=280)%	LV Thrombus (n=50)%	P value
Anterior Wall	138(49.3)	45(90)	
Inferior Wall	39(13.9)	0	
Inferior Wall +RV	33(11.8)	01(02)	<0.001
NSTEMI	66(23.6)	04(08)	
Lateral Wall	02(0.7)	0	
Inferior +Lateral wall	02(0.7)	0	

TABLE-III
COMPARISON OF OCCURRENCE OF LVT IN PATIENTS WITH RESPECT TO TREATMENT WITH THROMBOLYTICS. (N=280)

	LV Thrombus Present (n=50)%	LV Thrombus Absent (n=230)%	P value
SK Given	19(38)	147(63.9)	
SK not Given	31(62)	83(36.1)	0.001

RESULTS:

A total of two hundred and eighty patients with first episode of acute MI were studied; 187(66.8%) were male and 93(33.2%) were female. Mean age of patients with LVT was 56.56 + 11.29 vs those without LVT 53.43 + 11.7, majority of them were from 40 – 60 yrs age group. The presence of LVT was associated with a significantly higher peak value of CK-MB 159 + 127.7 vs 100.69 + 9.15 (P value < 0.009)

On echocardiographic examination, 50(17.86%) patients had LVT, 40(80%) were male and 10(20%) were females. Of those who developed LVT, 45(90%) patients had anterior wall MI, 4(8%) had NSTEMI and

1(2%) had inferior + right ventricle wall MI (Table-II). Majority of thrombi developed in acute phase of infarction (Fig. 1). In 33(66%) patients LVT was present on day 3 on echocardiogram, in 13(26%) patients on day 5 and in 4(08%) patients on day 6.

Among 280 patients 138(49.3%) had anterior wall MI, 39(13.9%) had inferior wall MI, 66(23.6%) had NSTEMI, 33(11.8%) had inferior + RV MI, 2(0.7%) had lateral wall and lateral + inferior wall MI. On admission 166(59.3%) patients were given intravenous streptokinase infusion; 19 of them developed LVT. Among 114(40.6%) patients who were not thrombolyzed with streptokinase, 31 developed LVT (P value 0.001).

Among the risk factors, Hypertension was present in 26(52%) patients having LVT compared to 83(36.1%) without LVT (p value 0.028). Diabetes mellitus was found in 28(56%) vs 94(40.9%) patients with and with out clot (p value 0.05). 40(80%) were smokers having LVT compared to 148(46.3%) smokers having no LVT (p value 0.045). 21(42%) obese patients developed LVT while 55(23.9%) obese patients did not developed LVT. Extensive antero apical asynergy with apical dyskinesia was present in 39/45(86.7%) with anterior wall MI and LVT vs 20/93(21.5%) with anterior wall MI and without LVT (Table-III). 3/4(75%) with NSTEMI and LVT had apical dyskinesia. None of the patients with inferior wall MI had segmental dyskinesia during the acute phase of MI.

DISCUSSION:

Ischemic heart disease (IHD) is one of the major cause of mortality and morbidity through out world[17]. LVT is a common complication in the clinical setting of acute anterior wall MI and very rarely after inferior wall MI[11, 12, 13]. It occurs early after acute MI[18, 19]. In patients of acute MI in whom in whom LV mural thrombus develops, the risk of systemic embolization is 10%[20] and increased risk is associated with (a) Advanced patients age. (b) A large anterior wall MI. (c) A protruding or mobile LV thrombus. (d) Hyperkinesis of the adjacent LV segments and (e) Central radiolucency of LV thrombus[21].

This present study indicates that LVT is common and early complication following acute MI, majority of cases of LVT developed in first few days after onset of acute MI. Two hundred and eighty patients with first episode of acute MI were echocaridograpically examined and LVT was found in 50(17.86%) patients. In majority of cases LVT developed in patients with anterior wall MI and were located in apical area. Wilbert S Arnow and his colleagues[22] also found LVT in 17% patients with anterior wall MI. A little high 26.66% incidence of LVT was found in study by Abdul Hafeez Chaudhry[23] and Waseem T[24]. We found that there was significant reduction in risk of LVT after thrombolytic therapy with streptokinase and found to be statistically significant (p value < 0.001). Similarly Domenicucci S[25] and Bhatnagar[9] have documented statistically significant decrease in incidence of LVT following thrombolytic therapy. Antero apical dyskinesia was far more frequent in patients with acute anterior wall MI with LVT than in patients without LVT (86.7% vs 21.5%). These findings suggest that there is an association between severe wall motion abnormality and LVT formation. A high incidence of apical dyskinesia in patients with LVT was also found by Asinger[26] and C.A Viser[27].

CONCLUSION:

LVT is a common complication in the clinical setting of acute MI especially acute anterior wall MI and usually develops within first few days in majority of patients and patients who develop LVT are at increased risk of systemic embolization. Early administration of thrombolytic therapy reduces the risk of development of LVT. It is concluded that patients with acute MI should be given thrombolytic therapy as earlier as possible and proper measures should be taken to control the risk factors like Hypertension and DM, so that incidence of LVT and its associated complications can be minimized.

CONFLICT OF INTEREST:

None declared.

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