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FREQUENCY OF LEFT VENTRICULAR THROMBUS FORMATION AFTER ACUTE ANTERIOR WALL ST ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

Objective: To determine the frequency of left ventricular thrombus (LVT) formation after Acute Anterior wall ST Elevation Myocardial Infarction (STEMI).

Methodology: This descriptive study was conducted at CCU department, Bahawal Victoria Hospital, Bahawalpur from January 15, 2012 to July 14, 2012. Patients age between 20 to 70 years and of any gender with diagnosis of acute anterior wall STEMI admitted through emergency department within 12 hours of chest pain were enrolled. Patients were treated according to standard ward protocol. Echocardiography was performed at 4th day of admission to diagnose for left ventricular thrombus.

Results: A total of 195 patients were studied. There were 146(74.9 %%) males and 49(25.1%) females. Mean age of patients with LVT was 55.43±7 and those without LVT were 52.48±10.2. On echocardiographic examination, 30(15.4%) patients had LVT, out of these 22(73.3%) were males and 8(26.7%) were females in total. Among 116 (59.5%) patients thrombolysed, 6 (5.2%) patients developed LVT while 79 (40.5%) patients who were not thrombolysed 24(30.4%) developed LVT (p value< 0.001).

Conclusion: The frequency of LVT is common in patients presenting with acute anterior STEMI particularly those patients who are not thrombolysed.

Key Words: Left ventricular Thrombus, acute myocardial infarction, Thrombolytic, streptokinase

INTRODUCTION

A vast number of patients presenting to Coronary care units(CCU) with ischemic chest pain are diagnosed as Acute ST Segment Elevation Myocardial Infarction (STEMI). It usually occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of coronary arteries previously affected by atherosclerosis. WHO criteria formulated in 1979 have classically been used to diagnose MI, if two of the following criteria are satisfied. Clinical history of ischemic type chest pain lasting for more than 20 minutes, changes in serial ECG tracings and rise and fall of serum cardiac biomarkers.[1]

Various complications can occur as a consequence of Acute ST Segment Elevation Myocardial Infarction like left ventricular failure, cardiogenic shock, acute mitral valve regurgitation, ventricular septal rupture, cardiac rupture, pericarditis, conduction disturbances, aneurysm formation, right ventricular infarction and left ventricular thrombus formation.

The development of left ventricular thrombi is one of the most common complications of STEMI.Thrombi are important clinically because they can lead to serious complications. The incidence of LVT as a complication of acute myocardial infarction (AMI) is approximately 23.5% and may reach higher in large anterior wall MI.[2-4]

Primary percutaneous coronary intervention (PCI) coupled with the use of potent antithrombotic and anti-platelet agents has reduced the incidence of left ventricular thrombus formation (4%for all MI, 11% for the anterior MI).[5,6] Stroke is the most common among these complications and a 44-fold increase in the incidence of stroke has been reported in patients with MI compared with the rate of stroke in the general population.[7,8] Transthoracic Echocardiography remains the imaging modality of choice and is 92% sensitive and 88% specific for detecting thrombus formation, dissolution and systolic function.[9,10]

All patients with Anterior wall ST elevation MI who seek medical care within the first 12 hours after the onset of symptoms should be considered for urgent reperfusion of infarct related artery.[11] The traditional reperfusion in our settings is in the form of thrombolysis by streptokinase infusion. Thrombolytic therapy reduces the rate of thrombus formation and the character of the thrombi so that they are less protuberant. Patients having left ventricular thrombus are treated with anticoagulants like warfarin for a period of 3 to 6 months. Although a mural thrombus adheres to the endocardium overlying the infarcted myocardium, superficial portions of it can bedetached and produce systemic arterial emboli. About 10 percent of mural thrombi result in systemic embolization.[12] Gender does not have any effect on the development of Left ventricular thrombus formation during in-hospital stay after first Anterior wall MI. LV Thrombus after Anterior wall MI occurs more frequently in patients older than 50 years of age who also have a Killip class >1 at presentation.[13]

Higher mortality has been reported in patients with left ventricular thrombi after infarction, especially when these develop within the first 48 hours after infarction. Left ventricular thrombosis is associated with increased embolism risk. If magnitude of LVT founds to be high then some practical recommendations regarding prevention of thrombus embolization could be made in the form of anti-coagulation like warfarin to prevent this morbidity. The purpose of this study was to look for the magnitude of left ventricular thrombus after anterior wall myocardial infarction.

METHODOLOGY

This descriptive study was conducted at CCU department, Bahawal Victoria Hospital, Bahawalpur from January 15, 2012 to July 14, 2012. Patients age between 20 to 70 years and of any gender with diagnosis of 1st acute anterior wall STEMI admitted through emergency department within 12 hours of chest pain were enrolled. Pregnant females, patients of CRF and cirrhosis of liver, hemodynamically unstable patients (Systolic blood pressure<90mm of Hg), patients having previous episode of myocardial infarction, patients having history of heart failure, patients having history of arterial or venous thrombosis and hematological disorders and patients not giving written consent were excluded from the study. Patients were treated according to standard ward protocol. Echocardiography was performed at 4th day of admission to diagnose for left ventricular thrombus. The images were recorded and analyzed by 2 independent cardiologists, who were blinded to each other's interpretation. The diagnosis of left ventricular thrombus was made when an echo dense mass was visible throughout the cardiac cycle in at least two echocardiographic views. Due respect was given to the patients and privacy was maintained. All investigations were carried out by the hospital.

All the collected information was entered and analyzed in SPSS (Statistical Package for Social Sciences) version 16. Clinical characteristics were summarized in terms of frequencies and percentages for categorical variables like gender and LVT. For numerical variables, mean±1SD were used like age. Statistical analysis was done by using Chi square test and p value of ⩽.05 was taken significant.

RESULTS

A total of one hundred and ninety five patients with first episode of acute MI were studied; 146(74.9%) were male and 49(25.1%) were female. Mean age of patients with LVT was 55.43±7.7 and those without LVT was 52.48±10.2,

Table 1: Baseline Characteristics in Different Groups of Patients

Characteristics	Patients with LVT (n=30)	Patients without LVT (n=165)	Total Patients (n=195)	P-value
Mean Age(yrs)±SD	55.43±7.7	52.48±10.2	52.94±9.9	.073
Age(yrs) in groups				
<35	0(0%)	7(4.2%)	7(3.6%)	.235
36-45	4(13.3%)	45(27.3%)	49(25.1%)	
46-55	13(43.3%)	53(32.1%)	66(33.8%)	
56-65	11(20.4%)	43(26.1%)	54(27.7%)	
>66	2(10.5%)	17(10.3%)	19(9.7%)	
Female	8(16.3%)	41(83.7%)	49(25.1%)	.822
Male	22(15.06%)	124(84.94%)	146(74.9%)	

majority of them were from 40-60 years age group (Table 1).

Echocardiographic examination was carried out on the 4th day of admission to look for the evidence of LVT. Thirty patients (15.4%) had LVT, out of these 22(73.3%) were male and 8(26.7%) were female (Table 1&2). None of the patients who developed LVT was having age less than 35 years, 4(13.3%) patients had age in between 36 and 45 years,13(43.3%) were having age in between 46 and 55 years, 11(20.4%) patients had between 56 and 65 and 2(10.5%) above 66 years(Table 1).

Among the risk factors, Hypertension was present in 76(39%) patients, Diabetes mellitus was found in 58(29.7%) patients, 117(60%) were smokers, 101(51.8%) patients were obese. There was a strong family history of ischemic heart disease in 57(29.2%) patients (Table 2).

On admission116 (59.5%) patients were given intravenous streptokinase infusion; 6(5.2%) out of them developed LVT. Among 79(40.5%) patients who were not thrombolyzed with streptokinase, 24(30.4%) developed LVT, p value <0.001. (Table 3).

Table 3: Comparison of Occurrence of LVT in Patients With Respect to Thrombolysis (Streptokinase)

Streptokinase	LV thrombus present, n=30	LV thrombus not present, n=165	P-value
Infused, n=116(%)	6 (5.2)	110 (94.8)	<.001
Not infused, n=79(%)	24(30.4)	55 (69.6)	

Extensive antero-apical asynergy with apical dyskinesia was present in 28(93.3%) with LVT vs 66(40%) without LVT (p value<0.001). Among the patients who developed LVT,12 (40%) patients were having ejection fraction(EF) of left ventricle below 35%, 14(46.7%) patients were having ejection fraction in between 36-45%, 3(10%) patients had EF in between 46-55% and only 1 patient of LVT(3.3%) had EF more than 55%(Table 4). Table 5 shows formation of LV clot in patients with anterior MI and its association with LV diastolic dimensions.

DISCUSSION

Acute myocardial infarction is one of the major cause of mortality and morbidity in the world. LVT is a common complication especially after large anterior wall MI due to the factors like left ventricular regional-wall akinesia or dyskinesia and resultant reduced left ventricular ejection fraction and/or left ventricular aneurysm formation.[2,5,14] The incidence of LVMT may be significant without the treatment and may be reduced with the proper management.[3-6] LV mural thrombi are important because they can get embolized and lead to serious embolic complications, including stroke, acute limb ischemia and mesenteric and renal infarction mostly in the first four months.[7,8,15] Transthoracic Echocardiography remains the imaging

Table 2: Frequency of Left Ventricular Thrombus Formation and conventional Risk Factors after Acute Anterior Wall STEMI

	Hypertension P-value = 0.9004		Diabetes Mellitus P-value = 0.049		Smoking P-value = 0.425		Obesity P-value = 0.427		Gender P-value = 0.822		Family History P-value = 0.081	
	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Male	Female	Present	Absent
Patients with LV clot n=30(%)	12 (15.8)	18 (15.1)	14 (24.2)	16 (11.7)	16 (13.7)	14 (17.9)	18 (17.8)	12 (12.8)	22 (15.1)	08 (16.3)	13 (22.8)	17 (12.3)
Patients without LV clot n=165(%)	64 (84.2)	101 (84.9)	44 (75.8)	121 (89.3)	101 (86.3)	64 (82.1)	83 (82.2)	82 (87.2)	124 (84.9)	41 (83.7)	44 (77.2)	121 (87.7)
Total	76 (39)	119 (61)	58 (29.7)	137 (70.3)	117 (60%)	78 (40%)	101 (51.8%)	94 (48.2%)	146 (74.9%)	49 (25.1%)	57 (29.2%)	138 (70.8%)

Table 4: Comparison of Occurrence of LVT in Patients With Respect to Ejection Fraction Groups

Ejection fraction (EF%) groups	LV thrombus present n=30(%)	LV thrombus not present, n=165	P-value
<35%	12(40.0)	11(6.7)	<.001
36-45%	14 (46.7)	41(24.80	
46-55%	3 (10)	48(29.1)	
56-65%	1(3.3)	60(36.4)	
>66%	0(0)	5(3)	

modality of choice.[8,9]

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.The mean age of the patients was 52.94 ± 9.9 years. There were 146(74.9%) male patients and 49 (25.1%) female patients and116 (59.48%) patients out of 195 were thrombolysed. In the distribution of patients by outcome, there were 30(15.4%) patients who developed LVMT with in the 72 hours of the acute anterior wall STEMI. Most common risk factor was smoking as it was present in 60% patients. HTN was present in 37.9% patients, DM in 29.7% of patients, family history of IHD in 29.2% patients and 51% patients were obese. Our results are in accordance with the previous studies.[4,16-19] Rathi et al, studied 280 consecutive patients at Liaqat University Hospital Hyderabad presenting with first acute myocardial infarction.[16] One eighty seven (66.8%) patients 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. 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. In our study, 146 (74.9%) patients were male and 49(25.1%) were female. Mean age of patients with LVT was 55.43 ± 7.7 vs. those without LVT 52.48 ± 10.2. On echocardiographic examination, 30(15.4%) patients had LVT. One hundred and sixteen (59.49%) patients were given intravenous streptokinase infusion; 6 of them developed LVT, among 79(40.51%) patients who were not thrombolyzed, 24 developed LVT. This study indicates majority of cases of LVT developed in first few days after onset of acute MI and the results are comparable with our study. However our study included patients only with anterior wall STEMI.

Table 5: Comparison of Occurrence of LVT in Patients With Respect to Left Ventricular Diastolic Dimensions Groups (LVIDD)(mm)

LVIDD(mm) groups	LV thrombus present n=30(%)	LV thrombus not present, n=165	P-value
<45	1(3.3)	39(23.6)	<.001
46-55	12(40)	101(61.2)	
56-65	15(50)	24(14.5)	
>66	2(6.7)	1(0.6)	

Aronow also found LVT in 17% patients with anterior wall MI which is comparable with our study.[17]

A little high 26.66% incidence of LVT was found in study by Waseem et al, and there was significant statistically reduction in risk of LVT after thrombolytic therapy with streptokinase.[13] Similarly Domenicucci et al, and Bhatnagar et al, had documented statistically significant decrease in incidence of LVT following thrombolytic therapy and gender was not a predisposing factor for the occurrence of LVT.[18,19] 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 comparable with our study but our study showed that the thrombus formation is more common in male patients as compared to the females.

Chaudhry et al, studied the incidence of left ventricular thrombus in first episode of acute MI.[4] In 30 patients, 24(70%) were males and 6(30%) were females. The mean age of males was 51.79±13.86 and that of females was 54.83±10.15 years. On echocardiography, 8 patients had LVT, 6(75%) were males and 2(25%) were females. On admission 16(53.33%) patients were thrombolized with streptokinase. Two of them developed LVT. Among LVT remaining 14(42.7%) patients, 6 were found to have LVT on the 6th day of admission. The occurrence of LVMT in this study was higher as compared to ours because their sample size was small and the echocardiography was done on the 6th day of admission.

Osherov et al, studied 642 patients at Rambam Medical centre, Israel in a 6-years period with anterior wall AMI and echocardiography treated with PPCI (n=297), thrombolysis (n = 128), or conservative treatment (n = 217).[14] The rate of LVT among anterior wall AMI was 6.2%. Predictors for LVT were reduced ejection fraction and severe mitral regurgitation. There was no statistical difference in LVT rate according to treatment: 21(7.1%) of 297 patients in the PPCI group, 10(7.8%) of 128 patients in the thrombolytic group, and 9(4.1%) of 217 patients in the conservative group. Those in the thrombolytic group were characterized by

shorter duration from symptom onset and were generally also treated with heparin/low-molecular weight heparin.

Porter et al, retrospectively analyzed patients (in the years 1997-2002) with a diagnosis of anterior ST-elevation AMI and no prior AMI, and who underwent a thorough echocardiographic assessment within 72 h of admission.[2] Of the 153 patients with complete data, LVMT was detected in 36 (23.5%). There were no significant differences in baseline demographic and clinical variables between LVMT and non-LVMT patients, or in treatments (all patients received reperfusion treatment). The mean wall motion score index was higher in LVMT than non-LVMT patients, indicating worse cardiac systolic function. The only independent predictor found for LVMT occurrence was worse regional wall motion of the apex. These results are little higher as compared with our study.

In a study by Mooe et al, the incidence of LVMT in patients admitted with anterior wall MI was observed to be 46% in those who received thrombolytic therapy (streptokinase).[20] The presence of LVMT was higher than observed in our study (15.4%) due to the fact that two echocardiograms were done during the hospital stay (3rd post MI day and predischarge) in contrast to our study where we did only one echocardiogram on 4th day of acute MI and secondly the patients with previous MIs were included in this study which were found strongly associated with the occurrence of LVMT, where as in our study we selected patients only with first acute MI.

Other important result of my study was the increased incidence of LVT in patients who had diabetes. It can be explained on the basis of the decreased formation of collaterals in diabetic patients which can cause LV dysfunction after STEMI more as compared with non-diabetic patients. The increased risk of LV dysfunction leads to cause a greater risk for the development of LVT.[21]

CONCLUSION

It is concluded that LVT is a common complication after anterior wall STEMI and it occurs within few days of MI. It can lead to various other thrombo embolic complications. The patients with acute anterior wall STEMI should be given the thrombolytic therapy as soon as possible and the proper measures should be taken to control the risk factors so that the incidence of LVMT and its complications can be minimized.

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