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FREQUENCY OF EARLY LEFT VENTRICULAR THROMBUS IN PATIENTS PRESENTING WITH VARIOUS TYPES OF ACUTE ST ELEVATION MYOCARDIAL INFARCTION USING ECHOCARDIOGRAPHY AS A MEASURING TOOL

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ABSTRACT

Objectives: To determine frequency of left ventricular thrombus (LVT) in acute ST elevation myocardial infarction (STEMI) and to find out the correlation of risk factors with development of left ventricular thrombus.

Study Design: Hospital-based observational cross sectional study.

Place and Duration of Study: AFIC / NIHD Rawalpindi, from Nov 2016 to Feb 2017.

Material and Methods: One hundred and fifty consecutive patients presented with first episode of acute STEMI were included. Patients with previous history of myocardial infarction, valvular heart disease, dilated Cardiomyopathy and mural thrombus were excluded. 2-D echocardiography was performed after 24 hours, 48 hours and 72 hours of admission. Descriptive and inferential statistical analysis was performed using SPSS version 23.0.

Results: Mean age of patients was 59.84 ± 11.16 years. Thrombus was seen in 35 (23.3%) patients out of which 21 (60%) were males and 14 (40%) were females. left ventricular thrombus occurred in 8 (22.8%) after 24 hours and 27 (77.2%) patients after 72 hours. LV thrombus was seen in 20 (57.1%) patients whose EF was less than 30% while it occurred in 10 (28.5%) patients having EF 30-45% and 5 (14.2%) patients with EF >45%. Among risk factor LV thrombus was seen in 17 (48.5%) diabetic patients, 4 (11.4%) patients had hypertension and 14 (40%) were those who had both diabetes and hypertension.

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

Keywords: Left ventricular thrombus (LVT), ST elevation myocardial infarction (STEMI), Two dimensional echocardiography (2D Echo).

INTRODUCTION

Acute myocardial infarction (AMI) is the leading cause of death all over the world and also among the most important reason for morbidity in hospitalized patients[1]. The most important initiating event in the development of AMI is coronary artery plaque fissuring or rupture that leads to the exposure of underlying subendothelial matrix to formed elements of blood. That further leads to cascade of events resulting in activation of platelets and thrombin generation leading to formation of thrombus. The development of occlusive thrombus within the lumen of coronary artery in the absence of collateral blood vessels most often results in the development of acute ST-segment elevation myocardial infarction (STEMI)[2]. Left ventricular mural thrombus is a well-recognized complication of acute myocardial infarction. In subjects dying of infarction, its overall incidence at postmortem is reported to be 30-40%[3].

The pathophysiologic mechanism for LV thrombus formation can be explained by so called "Virchow's triad" that is commonly found in patients suffering from AMI. The three components of this triad are stasis of blood, endocardial injury or dysfunction and a hypercoagulable state[4].

The likelihood of LV thrombus developing after an acute MI depends on the location as well

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on the size of infarct. LV thrombus is most often seen in patients with large anterior ST-elevation infarctions (STEMI) with antero-apical aneurysm formation[5]. One study has found increased age as a risk factor for LV thrombus[6].

The clinical significance of left ventricular thrombi lies on their potential risk of systemic embolization[7]. In fact, left ventricular embolism after myocardial infarction resulting in stroke, bowel, and limb ischemia has been reported in literature[8] In survivors of myocardial infarction with an LV mural thrombus, systemic embolization occurs in about 10%[9].

Factors that increase the risk of embolization are mobility, protrusion into the LV cavity and central echo-lucency of the clot[4]. The wall motion abnormalities, poor ejection fraction and mitral regurgitation have been linked with left ventricular thrombus formation.it has been seen that patients having low ejection fraction were more likely to develop left ventricular thrombus[10]. Another study done by glazier et al also supports the above finding[11]. Transthoracic echocardiography (TTE) is the most commonly used imaging modality to make a diagnosis of LV thrombus and has a sensitivity of 90%-95% and specificity of 85%-90%[4]. In recent years, timely therapy during the acute phase of AMI, such as anticoagulation, thrombolytic, and primary percutaneous coronary intervention (PPCI), has reduced the incidence of LVT[12].

This study is being done to highlight the magnitude of left ventricular thrombus in patients having acute ST elevation myocardial infarction[13,14]. The results will be helpful in assessing importance of diagnosis of this complication at early stage and will also guide in developing strategies regarding treatment in the post infarct patients, because significant frequency of left ventricular thrombus warrant early diagnosis and management of dangerous, yet silent complication of myocardial infarction.

Table-I: Left ventricular thrombus and gender of the patient.

Gender	Left Ventricular Thrombus	
	Present	**Absent**
Male	21 (22.8%)	71 (77.2%)
Female	14 (24.1%)	44 (75.8%)

Table-II: Demographic and clinical characteristics of patients presenting with STEMI.

Characteristics	Visible Thrombus			*p*-value
	24 hours n=4	**48 hours n=13**	**72 hours n=18**	
Left Ventricular EF				
< 30%	-	2 (15.3%)	15 (83.3%)	0.03
31 – 45%	1 (25.0%)	3 (23.0%)	3 (16.6%)	
>45%	3 (75.0.%)	8 (61.5%)	-	
Diagnosis				
Anterior wall MI	-	5 (22.7%)	16 (87.5%)	
Inferior wall MI	1 (25.2%)	8 (36.3%)	2 (12.5%)	0.05
Inferoposterior wall MI	1 (36.3%)	4 (18.1%)	-	
Anteroinferior wall MI	2 (36.3%)	5 (22.7%)	-	
Late for streptokinase	-	3 (23.0%)	16 (88.8%)	0.02

MATERIAL AND METHODS

This study was carried out in Armed Forces Institute of Cardiology & National Institute of Heart Diseases from November 2016 to February 2017. Approval was taken from institutions ethical and review committee prior to data collection. 150 consecutive patients presenting with first episode of acute STEMI were included. Patients with previous history of myocardial infarction, valvular heart disease, dilated Cardiomyopathy and mural thrombus were

excluded. 2-D echocardiography was performed at 24 hours, 48 hours and 72 hours after admission. Basis demographics and clinical data were collected on specified data collection forms. Basic Descriptive and inferential statistical analysis was performed using SPSS version 23.0.

RESULTS

Mean age of patients was 59.84 ± 11.16 years, males were more in number 92 (61%) as compared to females 58 (39%). 58 (12 %) patients were found to be diabetic while 40 (29%) were hypertensive. Anterior, inferior, infero-posterior and antero-inferior wall myocardial infarction was seen in 76 (50.67 %), 46 (30.67%), 17 (11.33 %) and 11 (7.33 %) patients respectively. Primary percutaneous intervention was done in 60 (41.3%) patients while 48 (30.8%) were given streptokinase, 42 (41.5%) patients were late for thrombolysis. Mean ejection fraction on 2D echo was 45.77 ± 7.34%. Thrombus was seen in 35 (23.3%) patients out of which 21 (60%) were males and 14 (40%) were females as shown in table-I. (11.4%) left ventricular thrombus occurred after 24 hours, 13 (37.1%) after 48 hours and 18 (51.4%) after 72 hours. LV thrombus was seen in 20 patients whose EF were less than 30% while it occurred in 10 patients having EF 30-45% and 5 in patients with EF >45% as shown in table-II & fig. Among risk factor LV thrombus figure was seen in 17 diabetic patients, 4 patients having hypertension and 14 patients have both diabetes and hypertension.

DISCUSSION

Formation of Left ventricular thrombus is a serious complication after acute ST elevation myocardial infarction. Incidence of early LV thrombus is around. 20-60% as reported in different studies. 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[15,16]. Higher mortality has been reported in patients with left ventricular thrombi after infarction, especially when these develop within the first 48 h after infarction[17]. The incidence of LVMT may be significant without the treatment and may be reduced with the proper management[18]. Transthoracic echocardiography remains the imaging modality of choice. Many researchers agree that although thrombolytic therapy does not prevent LVT formation but has significantly reduced the incidence of embolization.

Our study showed that early left ventricular

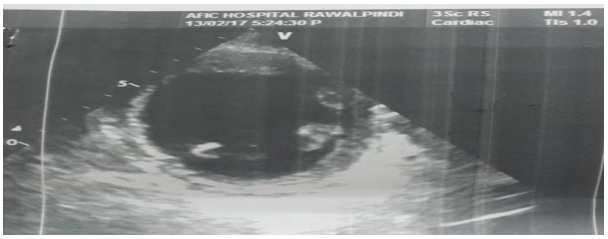

Figure: LV thrombus seen attached to anterolateral wall at mid papillary level after 72 hours of anterior wall myocardial infarction (late for SK).

thrombus occurred in 35 patients out of 150 (23.33%). Rathi and his colleagues found that 17.8% of the patient of all MI groups developed LV Thrombus which is close to our study[19]. Among the different types of myocardial infarction, the incidence of left ventricular thrombus was highest in anterior myocardial infarction 76 (50.67%) and 74 (49.33%) was seen in other myocardial infarctions. The mechanism of LVT after AMI is still not clear and it is thought to occur due to abnormalities of coagulation cascade and injury to myocardium[20]. An extensive area of myocardial injury stimulates a large quantity of fibrin, erythrocytes, and platelets to adhere to the exposed collagen, which impels a coagulation cascade and results in the formation of a thrombus. In our study, the percentage of extensive anterior wall myocardial infarction was much higher than that of other infarction locations among the LVT patients, possibly because extensive myocardial injury is related to a larger explosion of coagulation, which induces a greater amount of platelet adhesion. Additionally, severe regional wall motion abnormalities are usually accompanied by an extensive myocardial infarction, which

results in a higher incidence of LVT. In our study, Left ventricular thrombus was more common in males as compared to females. Regarding the occurrence of thrombus it was mostly seen in 72 hours as compared to 24 hours. LV clot were mostly seen in apical position which is comparable to Salehi et al who also found most left ventricular thrombi in apical position in their study[21].

This study also took into account two major risk factors i.e. diabetes and hypertension. In our study there were 30 (20.0%) hypertensive patients, out of which 4 (1.3%) had left ventricular thrombus. Out of 50 diabetic patients 17 (34%) developed left ventricular thrombus. This showed that LV thrombus was more common in diabetic patients. Patients managed with streptokinase and PPCI were also included in the study and it was seen that lv thrombus occurred more commonly in those who were late for thrombolysis as compared to those who underwent revascularization. This fact is supported by the Kambery et al study which concluded that in patients in whom PCI was administered as the primary therapy, the frequency of LVT was 5.6% in anterior MI while in the non PCI group the frequency was as high as 44.4%[22].

In our findings, who had low EF i.e. EF less than 30% LV thrombus were more as compared to those having EF more than 45%. Previous studies have also indicated LVEF ≤40% as an independent risk factor of LV thrombus[23].

CONCLUSION

Left ventricular thrombus is a common finding in patients of acute STelevation myocardial infarction (STEMI).

It commonly occurs after 72 hours, in anterior wall myocardial infarction and in those who present late for thrombolysis so echocardiography should be done routinely to rule out this complication.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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