

# Left Ventricular Thrombus in Acute Myocardial Infarction

Saulat Siddique, Naeem Sajjad, Zafar Iqbal

Department of Cardiology, Shaikh Zayed Hospital, Lahore

## SUMMARY

*Fifty consecutive patients with acute transmural anterior myocardial infarction were evaluated by transthoracic echocardiography, on average at day 5-7 of the acute infarction. Forty-two were males and 8 were females with an age range of 25-80 years. All received streptokinase and/or heparin. Sixteen (32%) of these patients had evidence of a left ventricular thrombus (LVT). Age, sex, duration of symptoms prior to presentation, prevalence of smoking, presence of other systemic illnesses, clinical status at admission, use of thrombolytic therapy, number of Q waves in the electrocardiogram (ECG), peak creatinine phosphokinase (CPK) level and presence of post-infarct angina, arrhythmias or cardiac failure were not significantly different in those developing LVT versus those who did not.*

*However, on echocardiography, significantly greater number of segments with abnormal kinetics and greater degree of abnormal movements were found in patients with thrombus ( $P < 0.01$ ). Also, these patients showed a tendency to have larger end-systolic and end-diastolic left ventricular dimensions and lower mean calculated ejection fraction though these did not achieve statistical significance. In summary, despite thrombolysis and anti-coagulation, about 1/3rd of the patients with acute anterior transmural myocardial infarction develop LVT. This is more common in patients with a greater degree of segmental wall motion abnormalities and a larger left ventricle. Hence, all these patients should have predischarge echocardiography.*

## INTRODUCTION

**M**yocardial infarction is one of the commonest medical emergencies. It affects about 1.5 million people in the USA each year and is responsible for 25% of the total annual mortality<sup>1</sup>.

Formation of a thrombus in the left ventricular cavity is a potentially disastrous complication of acute myocardial infarction. About one third of patients with an acute anterior Q-wave infarction develop left ventricular thrombus (range 16-46%)<sup>2-6</sup> while the incidence with infarction in other areas e.g. inferior and non-Q infarctions is less than 5% (on average 1.5%)<sup>3</sup>. Its presence is an ominous sign because of its embolic potential especially to the brain. 22-36% of all systemic emboli are associated with myocardial infarction<sup>7</sup> and upto 45% of patients with LVT dying after myocardial infarction have been found to have sustained an embolism on

autopsy<sup>8</sup>. Most of the embolic events occur in the immediate post-infarction period i.e. 67% by 3 weeks and upto 80% within 6 weeks of the infarction<sup>9</sup>. Majority of the emboli (60-80%) involve the cerebral circulation and about 30% block major limb arteries. Mesenteric emboli account for about 4% while other important sites include splenic and renal arteries which are silent in most cases<sup>5,9</sup>.

Many factors have been associated with the development of LVT. These include location<sup>2,10-13</sup> and extent of the infarct<sup>11</sup>, regional left ventricular dilatation<sup>11</sup> and presence of dyskinetic segments<sup>10,15-19</sup>, reduced ejection fraction<sup>1,12-14,20</sup>, presence of cardiac failure<sup>3,11,21-23</sup>, arrhythmias<sup>3,4</sup> and bundle branch block, history of previous infarction<sup>21</sup> and development of a left ventricular aneurysm<sup>2,25,26</sup>.

Our population has certain inherent differences concerning patterns of ischemic heart disease and thromboembolic events as compared to Western

communities. More and more patients with triple coronary vessel involvement without evident major risk factors are seen while incidence of deep vein thrombosis and pulmonary embolism remains low in our population. On these grounds, it is open to speculation that the frequency of LVT after acute myocardial infarction (AMI), factors contributing to its formation and its embolic complications may be different in our patients. This study intends to look into the incidence of LVT in acute anterior myocardial infarction, possible contributing factors and embolic complications. Moreover, most of the previous studies were in patients who did not receive thrombolysis. This study would also look for the effects of thrombolysis, if any, on LVT.

## PATIENT AND METHODS

Fifty consecutive patients with acute transmural anterior myocardial infarction admitted to the coronary care unit of Shaikh Zayed Hospital were included in the study.

### Inclusion criteria

Patients fulfilling following criteria were included in the study.

First, acute (duration of symptom  $\leq$  36 hours), transmural, anterior wall, myocardial infarction as evidenced by presence of either two of the three following criteria;

- i. Typical anginal pain lasting more than 30 minutes.
- ii. ST segment elevation of  $\geq$  2 mm in anterior chest leads or  $\geq$  1 mm in leads I, aVL followed by evolutionary changes and development of Q waves in at least two contiguous anterior chest leads.
- iii. Elevation of cardiac enzymes CPK and aspartate transaminase in proper sequence.

All adult patients belonging to any age group and both sexes fulfilling the above criteria were included in the study.

Patients with concomitant infarction of other regions e.g. inferior wall, were also included in the study if they had definite evidence of acute anterior myocardial infarction.

### Exclusion criteria

Following patients were excluded from the study;

- Patients with pure lateral wall, inferior or posterior wall myocardial infarction.
- Patients with history, ECG and enzyme changes suggestive of infarction more than 36 hours old.
- Patients found to have cardiac aneurysm suggestive of old anterior myocardial infarction.
- Patients with incomplete data (left against medical advice, discharged on request, unable to afford echocardiography).
- Patients dying due to complications of myocardial infarction other than documented thromboembolism e.g. due to arrhythmias, cardiogenic shock etc, before completion of data.
- Patients not able to receive anticoagulant or thrombolytic therapy.

### Treatment protocol

All patients were admitted to the coronary care unit of Shaikh Zayed Hospital. They were restricted to bed during the first 24-48 hours. All of them had their complete medical history and physical examination recorded. The things particularly stressed were major and minor risk factors for ischemic heart disease such as diabetes mellitus, hypertension, hyperlipidemia, family history, amount and duration of cigarette smoking, and clinical condition as assessed by pulse rate, Killip class at the time of admission etc. All patients were given intravenous isosorbide dinitrate infusion for the first 24 hours unless contraindicated (hypotension) in a dose adjusted to clinical needs and averaging 2 mg/hour. Patients were given one tablet (300 mg) of acetyl salicylic acid to chew and swallow at admission. Oxygen (4-6 l/min) was given for the first 24 hours, if tolerated, even if the patient was not in cardiac failure.

Thrombolysis was carried out with streptokinase 1.5 million units diluted in 100 cc of normal saline and infused over one hour as soon as available. Duration of symptoms of the patient and time interval between onset of symptoms and initiation of thrombolytic therapy was also recorded. All the patients able to afford streptokinase who did not have any contra-indication and came within twenty four hours or had clinical or electrocardiographic evidence of infarct extension even after twenty four hours of onset of symptoms

## *Left Ventricular Thrombus in Acute Myocardial Infarction*

were thrombolysed.

Results of activated partial thromboplastin time (APTT) were monitored every six hours and heparin infusion 1000 IU/hour was instituted at APTT 1.5-2 times the control.

Patients not qualifying for thrombolytic therapy were given 5000 IU of heparin as intravenous bolus for priming effect and then were started on continuous infusion at a rate of 1000 IU/hour. 24-48 hours after starting continuous infusion, patients were switched over to intermittent bolus therapy with heparin i.e. 5000 IU intravenously 6 hourly until echocardiography was done. Thereafter, patients with left ventricular thrombus were converted to warfarin therapy with 2-3 days of overlap period.

All patients had electrocardiographic monitoring for first 48 hours. Incidence of potentially dangerous arrhythmias (multifocal or early ventricular ectopics, couplets, atrial fibrillation, ventricular tachycardia or fibrillation), post myocardial infarction anginal pain and cardiac failure were recorded.

Patients received narcotic analgesics (pethidine or morphine), beta blockers, calcium channel blockers, angiotension converting enzyme inhibitors, diuretics or inotropic agents as indicated in individual cases.

Serial ECGs were done by trained technicians 6 hourly during the first 24 hours and then daily for 3-4 days and whenever specifically indicated. Cardiofax machine Model ECG-68511C was used in all patients. The number of leads showing changes of acute myocardial infarction (hyperacute T waves, ST segment elevation) at the time of admission was recorded. Evolutionary changes in ST segment and T wave morphology were followed in serial ECGs and the number of leads showing development of new Q waves was also noted.

Creatinine phosphokinase (except in special circumstances) was measured by CK-NAC-Activated Humazym UV test (Human manufacture) 6 hourly during first twenty four hours and then daily until it showed a downward trend. Our normal lab values of CPK are 24-195 U/L for males and 24-170 U/L for females.

All patients had their baseline platelet count, prothrombin time and activated partial thromboplastin time checked in the laboratory before starting thrombolytic or anticoagulant therapy. Thereafter, APTT was monitored 6 hourly

in patients receiving thrombolysis until value came to twice the control result and then during heparinization, it was checked daily. Prothrombin time was monitored daily in patients being converted to warfarin therapy. International Normalized Ratio of 2.0-3.0 was taken to be satisfactory (effective anticoagulation).

Transthoracic echocardiography was done in all patients with anterior wall myocardial infarction who agreed to have it done. One experienced person carried out all the studies on M-Mode and 2 dimensional echo machine with doppler facility (model SSH 40 A, Toshiba) on average at day 5-7 of the acute infarction. Standard views were used.

Wall motion abnormalities of the myocardial especially left ventricular apex were studied in detail. Symmetric systolic inward motion was defined as normal. Less than normal contractility was hypokinesis. Akinesia (absent systolic inward motion) and dyskinesia (paradoxical outward systolic motion) were taken as severe wall motion abnormalities.

End-systolic and end-diastolic volumes and ejection fraction were also recorded carefully. Doppler evidence of mitral regurgitation was also looked for.

Video recordings of the studies were observed independently by one of the consultant cardiologists working in Shaikh Zayed Hospital.

Following criteria were followed for diagnosis of thrombus;

- a. An intracavitary mass visible in two markedly different sector orientations in both systole and diastole.
- b. An intracavitary mass with definite margins.
- c. Clear delineation of underlying endocardium.

Cases with equivocal echocardiographic evidence of thrombus were taken as negative studies.

Symptoms suggestive of embolism were also inquired from all patients during hospital stay. Patients with definite echocardiographically documented thrombi were warfarinized.

### **Statistical analysis**

It was done with the help of SPSS package for different variables recorded in the study.

## RESULTS

Fifty consecutive patients with acute anterior myocardial infarction fulfilling the criteria of the study admitted in coronary care unit over a period of 13 months (mid-October 1993 to mid-November 1994) were included in the study. All the patients were assessed and treated by the residents under supervision of consultant cardiologists independent of the fact that the patient is being included or excluded from the study. Sixteen of the patients (32%) developed left ventricular thrombus (LVT). On this basis patients were divided into LVT and non-LVT groups for comparison.

### Age and sex distribution

Forty-two patients were male and 8 were female (Table 1). Age of the patients ranged from 25 to 80 years. Most of the patients were either middle aged or elderly. No age or sex predisposition was found in those developing left ventricular thrombus (Figs. 1 and 2) (P 0.723, not significant).

Sex	*LVT Group		**Non-LVT Group	
	No.	%	No.	%
Male	13	81.25	29	85
Female	3	18.75	5	15

\*Left ventricular thrombus; \*\*Non-left ventricular thrombus.

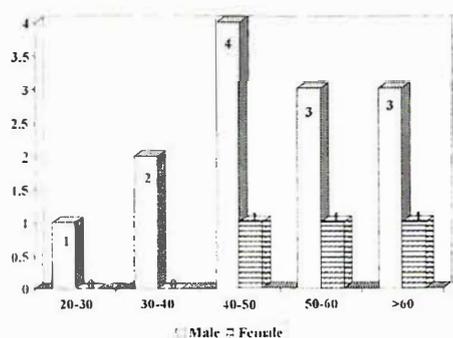


Fig. 1: Age and sex distribution of LVT group.

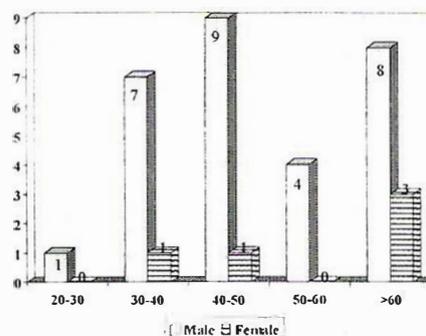


Fig. 2: Age and sex distribution of non-LVT group.

### Duration of symptoms

Majority of the patients (74%) presented to the emergency department within six hours of onset of symptoms. Those patients who later on developed left ventricular thrombus presented even earlier (Table 2).

Duration of symptoms	LVT Group		Non-LVT Group	
	No.	%	No.	%
< 6 hours	15	93.75	22	64.7
6-24 hours	1	6.25	6	17.6
> 24 hours	-	-	6	17.6

### Smoking

Fifty-four percent of the patients were smokers and 25% of these smoked more than 20 cigarettes per day. Prevalence of smoking was same in those with and without left ventricular thrombus (Table 3).

### Other systemic illnesses

Five patients suffered from ischemic heart disease before having this first anterior myocardial infarction. Eleven patients were non-insulin dependent diabetics and 8 patients were

## Left Ventricular Thrombus in Acute Myocardial Infarction

hypertensive. None of these conditions predisposed to left ventricular thrombus (Table 3). Asthma, chronic obstructive pulmonary disease, rheumatoid arthritis and hypothyroidism each affected one patient in non-developers of thrombus. One patient was previously operated for carcinoma of breast and one had chronic renal failure in those developing left ventricular thrombus.

Variables	LVT Group		Non-LVT Group	
	No.	%	No.	%
IHD	1	6.25	4	12
DM	2	12.5	9	26
HTN	1	6.25	7	21
Smoking	9	56.25	18	53

### Clinical status at admission

Majority of the patients (84%) were not in cardiac failure clinically at the time of admission. There was no increase in prevalence of cardiac failure at admission in those developing thrombus (Table 4). One patient in each group presented in severe cardiac failure. 79% of the patients had their heart rate between 60 and 100 beats per minutes. One patient in each group had heart rate less than 60 BPM. 21% of the patients without LVT and 13% of the patients with LVT had rate more than 100 beats per minutes (Table 5).

Killip class	LVT Group		Non-LVT Group	
	No.	%	No.	%
I	13	81.25	29	85
II	2	12.5	4	12
III	-	-	-	-
IV	1	6.25	1	3

### Thrombolytic therapy

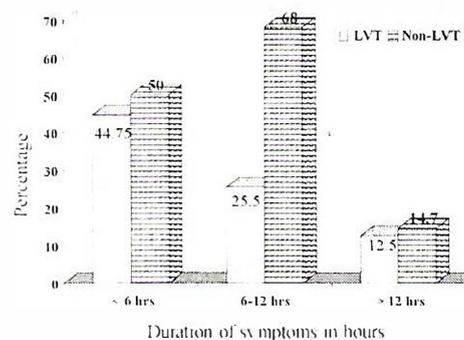
Seventy-two percent of the patients received thrombolytic therapy. Percentage of patients receiving streptokinase was higher in LVT group

(87.5%, 14/16 LVT vs 70%, 24/34 non-LVT) (Table 6), P 0.422, not significant). 50% of the patients in both groups received thrombolytic therapy within six hours of onset of symptoms. Two patients in LVT group and five patients in non-LVT group received streptokinase more than twelve hours after onset of symptoms (Fig. 3) (P 0.209, not significant).

All the patients included in the study received heparin therapy for about five days (Table 6).

Time	LVT Group		Non-LVT Group	
	No.	%	No.	%
< 60	1	6.25	1	3
60 - 100	13	81.25	26	76
> 100	2	12.5	7	21

Treatment	LVT Group		Non-LVT Group	
	No.	%	No.	%
Streptokinase	14	87.5	24	71
Heparin	16	100	34	100



**Fig. 3: Duration of symptoms before thrombolysis.**

**ECG**

Majority of patients had electrocardiographic changes of myocardial infarction in 3-5 anterior chest and/or lateral limb (I, AVL) leads. 66% of the patients had hyperacute T waves and ST segment elevation (of more than two millimeter in anterior chest and more than 1 mm in leads I, aVL) in 3-5 leads at the time of admission. 72% of the patients with LVT and 66% of the patients in non-LVT group had these changes. Five patients (31%) in the LVT group and 11 patients (32%) in non-LVT group had ST segment elevation in more than five leads (Table 7) (P 0.738, not significant).

Subsequently 50% patients in each group developed Q waves in 3-5 leads. Thirty percent of the patients in non-LVT group and 25% of patients in LVT group developed Q waves in more than 5 leads. Rest of the patients (25% with LVT and 21% without LVT) developed Q waves in less than three leads (Table 8) (P 0.667, not significant).

Three patients in each group showed persistently elevated ST segment and upright T waves (9% without LVT and 19.8% with LVT). Persistent ST segment elevation was found in 59.4% of the patients with LVT and 72% of the patients without LVT.

**Table 7: Number of ECG leads showing ST segment elevation at admission.**

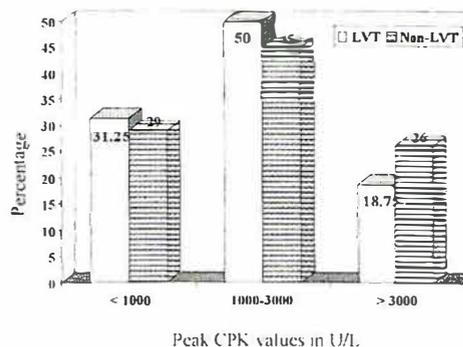
Number of leads	LVT Group		Non-LVT Group	
	No.	%	No.	%
< 3	-	-	1	3
3-5	11	68.75	22	65
> 5	5	31.25	11	32

**Table 8: Number of Q waves in ECG at day 3.**

Number of leads	LVT Group		Non-LVT Group	
	No.	%	No.	%
< 3	1	25	7	21
3-5	8	50	17	50
> 5	1	25	10	29

**Peak CPK level**

Fifty percent of the patients with LVT had peak CPK value between 1000-3000 U/L while 19.8% had CPK > 3000 U/L and value in 33% was < 1000 UL. In contrast, among patients without LVT, 30% had CPK less than 1000 U/L, 27% had more than 3000 U/L and 45% had between 1000 and 3000 U/L (Fig. 4) (P < 0.905, not significant).



**Fig. 4: Peak CPK values in patients.**

**Other complications**

Incidence of post-infarct angina, arrhythmias and cardiac failure was 6.6%, 19.8% and 25% respectively in patients with LVT and corresponding figures in non-LVT group were 18%, 27% and 18% respectively. Arrhythmias noted were frequent ventricular premature beats, ventricular bigeminy, ventricular fibrillation etc (Table 9) (P 0.924, not significant).

**Echocardiography**

Wall motion abnormalities involved more than two myocardial segments and were classified severe in all patients with LVT. Left ventricular apex, distal, middle and proximal 1/3rds of the septum, anterior and lateral wall each was taken as a single segment. In patients without LVT, majority (90%) of the patients had wall motion abnormalities of more than two segments. In 63% of these patients, wall motion abnormalities were severe. Rest of them had mild-to-moderate abnormalities. Involvement of more than 3 segments by dyskinesia was

## Left Ventricular Thrombus in Acute Myocardial Infarction

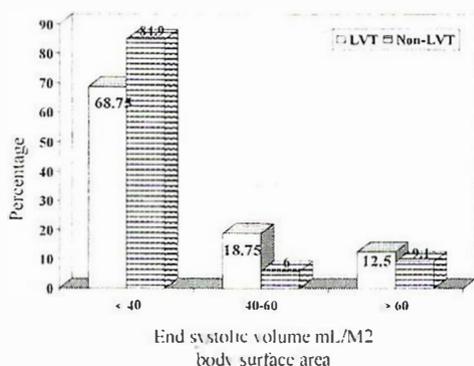
significantly higher in patients developing left ventricular thrombus than those who did not ( $P < .01$ , Two tail Fisher's exact test value .01).

**Table 9: Complications of myocardial infarction during admission.**

Complications	LVT Group		Non-LVT Group	
	No.	%	No.	%
Unstable angina	1	6.25	6	18
Arrhythmias	3	18.75	9	26
Failure	1	25	6	18

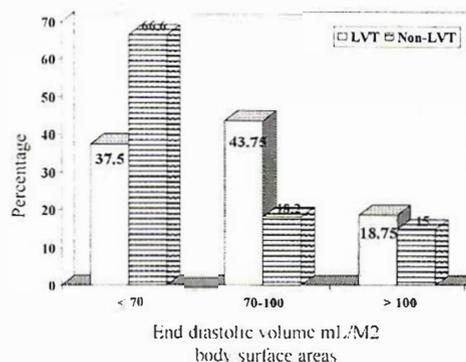
In patients not developing left ventricular thrombus during hospitalization, mean end-systolic volume was 28 ml/M<sup>2</sup>. Eighty five percent had mean ESV less than 40 ml/M<sup>2</sup>, in 6% value was between 40-60 ml/M<sup>2</sup> while 9.1% had ESV more than 60 ml/M<sup>2</sup>.

Calculated mean end-diastolic volume in this group was 69.12 ml/M<sup>2</sup>. Sixty seven percent of these patients had EDV less than 70 ml/M<sup>2</sup> while 18.2% and 15.1% had figures of between 70-100 ml/M<sup>2</sup> and >100 ml/M<sup>2</sup> respectively ( $P .113$ , not significant) (Fig. 6).



**Fig. 5: End systolic volume.**

Mean end-systolic and end-diastolic volumes in patients with LVT were 38.3 ml/M<sup>2</sup> and 81.8 ml/M<sup>2</sup> of body surface area respectively. The same values in those without LVT were 28 ml/M<sup>2</sup> and 69.1 ml/M<sup>2</sup> respectively. 68.7% of patients with LVT had end-systolic volume (ESV) less than 40 ml/M<sup>2</sup>. Three patients (18.7%) had ESV 40-60 ml/M<sup>2</sup> while two patients (12.5%) had ESV more than 60 ml/M<sup>2</sup> (Fig. 5). 37.5% patients with thrombus had end-diastolic volume (EDV) less than 70 ml/M<sup>2</sup>, 43.7% had value of 70-100 ml/M<sup>2</sup> and 18.7% had EDV more than 100 ml/M<sup>2</sup>.



**Fig. 6: End diastolic volume in patients.**

Mean calculated ejection fraction in the thrombus group was 55.6%. 18.75% had ejection fraction less than 40% while 37.5% and 43.75% had ejection fraction between 40-60% and more than 60% respectively (Fig. 7).

In non-LVT group, calculated mean ejection fraction (EF) was 63.7%. Majority of the patients (63.6%) had ejection fraction more than 60%, while 30.3% had EF between 40-60% and only 6% had ejection fraction less than 40%.

In one patient (in the non-LVT group), due to anatomical reasons, echocardiographic study was inadequate for the measurement of end-systolic and end-diastolic volumes and hence calculation of

ejection fraction. He has not been included in the data calculations (Figs. 5, 6, 7).

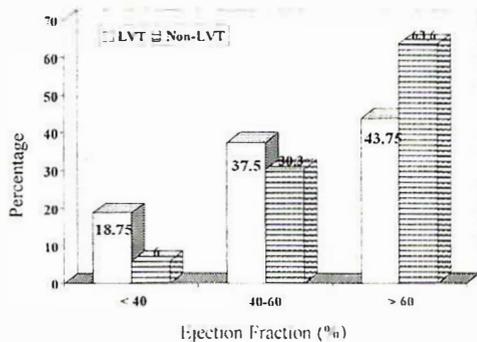


Fig. 7: Ejection fraction in patient population.

## DISCUSSION

Formation of a thrombus in left ventricular cavity after myocardial infarction is alarming. Its embolic potential especially to the brain is worrisome. Hence, it has received lot of attention from researchers. Although meta analysis of many small, short-term studies does give an idea of the etiological factors involved and interventions helpful in preventing this thromboembolic complication of myocardial infarction, so far there is no single large trial or universally accepted recommendations.

In my small study of fifty consecutive patients with anterior myocardial infarction, the incidence of left ventricular thrombus was 32% which is in agreement with results of most of the reported clinical studies<sup>5,22,27,29</sup>. Thrombus complicated infarction in both sexes and patients of all ages. There was no increase in incidence in the elderly. Lamas et al.<sup>14</sup> and Sagie et al.<sup>27</sup> also had similar observations.

About 75% of the patients sought medical advice within six hours of onset of their symptoms. Most of these patients received thrombolytic and

anticoagulant therapy immediately. No increase in the incidence of LVT was noticed in those who came late and the therapy was delayed. It is difficult to explain this on logical grounds because it has been observed in experimental and clinical situations that thrombus initiation occurs within few hours in most of the patients and earlier institution of thrombolysis and anticoagulation retards and reverses this process. It is possible that a high local concentration of pro-coagulants, stasis and large thrombogenic surface may offset the beneficial effects of thrombolytic and anti-coagulant therapy.

Smokers are also known to have relative hyper-coagulable state. No predisposition to thrombus formation, however, was noticed in smokers. Similarly, concomitant systemic diseases such as diabetes or hypertension, had no effect on thrombus occurrence.

Most of the patients presented in a stable condition with no clinical evidence of cardiac failure or bradycardia and hospital stay was also uneventful. However, occurrence of cardiac failure, excessive arrhythmias, bradycardia and post-infarct angina was not significantly different in the two groups. This is in contrast to the reports of Lamas et al.<sup>14</sup>, DeMaria et al.<sup>3</sup>; Spirito et al.<sup>21</sup>; Vecchio et al.<sup>22</sup> but consistent with results of Asinger et al.<sup>10</sup>.

Luckily very few patients in the study group were in cardiac failure or developed other complications during hospital course and it is not judicious to conclude that cardiac failure and dysrhythmias have no association with LVT. This could be due to exclusion of patients dying due to complications of myocardial infarction.

Conflicting results have been reported on the impact of thrombolytic and anticoagulant therapy on mural thrombus but general consensus is that they do reduce thromboembolic complications. There was no control group in my study and the incidence of mural thrombus without this mode of therapy is unknown in our population. It would have been unethical to deprive people from other benefits achieved by thrombolysis and anticoagulation and make a control group.

By ECG criteria, extent of myocardial damage was similar in both groups but persistently elevated ST segment with upright T waves was twice as common (18%) in those developing LVT as compared to those without LVT (9%). Similar ECG findings were reported by Sagie et al.<sup>27</sup> but they found this in a higher percentage of patients. Similarly Q

waves in leads I and aVL were not found to be sensitive or specific for LVT. Although ECG gives gross assessment of infarct size it is not very sensitive as many areas are not mapped by 12-lead ECG. It was observed echocardiographically that patients with LVT tended to have wall motion abnormalities of more myocardial segments and greater degree of functional impairment.

Peak CPK values were not markedly different in both groups. Actually, more patients without LVT had CPK values greater than 3000 U/L as compared to those with LVT. This also is not consistent with expected results and reports by DeMaria et al<sup>3</sup>, Johannessen et al<sup>28</sup>; Spirito et al<sup>21</sup>; Domenicucci et al<sup>30</sup>; Bhatnagar and Yusuf<sup>26</sup>; Penco et al<sup>23</sup>. It is possible that because most of the patients received thrombolytic therapy, which causes earlier and steeper rise in cardiac enzymes, peak CPK may not correlate with extent of myocardial damage closely. Another reason could be late reperfusion or non-reperfusion in those developing thrombus and hence, lower peak CPK values.

Echocardiographic findings were the only remarkable difference between the two groups of patients<sup>10</sup>. Higher number of segments with abnormal kinetics and greater degree of abnormal movements were found in patients with thrombus ( $P < .01$ ). Similarly, larger end-systolic and end-diastolic left ventricular dimensions and lower mean calculated ejection fraction were noted in patients developing thrombus although statistically not significant ( $P > .05$ ). Similar findings have been reported by Asinger et al<sup>10</sup>, Kupper et al<sup>16</sup>; Jugdutt and Sivaram<sup>17</sup>; Chamsi Pasha and Barnes<sup>18</sup>; Visser et al<sup>9</sup>; Lamas et al<sup>14</sup>; Friedman et al<sup>4</sup>; Sipula et al.<sup>13</sup>; Keren et al.<sup>12</sup>; Bhatnagar and Al-Yusuf<sup>20</sup>. Dilated, poorly contracting, left ventricle causes local stasis, favouring dominance of procoagulants and, hence, initiation and propagation of thrombus.

All of our patients were warfarinized after echocardiographic detection of left ventricular thrombus. Fortunately, none of them developed clinically evident embolism during hospital stay ranging from 1-2 weeks.

In summary, despite anticoagulation and thrombolysis, left ventricular thrombus is a common, silent and potentially disastrous complication of acute anterior transmural myocardial infarction. About one-third of the patients are likely to develop this complication. One should be on the lookout for it especially in those

with clinically large-sized infarction and big cardiac size. Echocardiographically, increased left ventricular dimensions and mechanical dysfunction predispose to thrombus formation. Preferably, all such patients should have predischarge echocardiography and those with thrombus should be anticoagulated.

## ACKNOWLEDGEMENTS

The authors wish to acknowledge the help and contribution of the following in the completion of this study; Prof. Tahir Shafi, Dr. Rashid Chaudhry, Mr. Tariq, Mr. Amjad Ali, Mr. M. Yaseen, Mr. M. Saleem and the staff of the Coronary Care Unit of Shaikh Zayed Hospital, Lahore.

## REFERENCES

1. American Heart Association: 1990. Heart Facts: Dallas American Heart Association National Center, p. 1.
2. Weinreich DJ, Burke JF, Pauletto FJ. Left ventricular thrombi complicating acute myocardial infarction. Long term follow up with serial echocardiography. *Ann Int Med* 1984; 100: 789-94.
3. DeMaria AN, Bommer W, Neuman A, Grehl T, Weinart L, DeNardo S, Amsterdam EA, Mason DT. Left ventricular thrombi identified by cross-sectional echocardiography. *Ann Int Med* 1979; 90: 11-8.
4. Friedman MJ, Carlson K, Marcus FI, Woolfenden JM. Clinical correlation in patients with acute myocardial infarction and left ventricular thrombus detected by two dimensional Echocardiography. *Am J Med* 1982; 72: 894-8.
5. Johannessen KA, Nordrehaug JE, Vander Lippe G, Vollset SE. Risk factors for embolization in patients with left ventricular thrombi and acute myocardial infarction. *Br Heart J* 1988; 60: 104-10.
6. Weintraub WS, Ba-albaki HA. Decision analysis concerning the application of echocardiography to the diagnosis and treatment of mural thrombi after anterior wall acute myocardial infarction. *Am J Cardiol* 1989; 64: 708-16.
7. Tomoda H, Hoshiai M, Furuya H, Kuribayashi S, Ootaki M, Matsuyama S, Koide S, Kawada S, Shotsu A. Evaluation of intracardiac thrombus with computed tomography. *Am J Cardiol* 1983; 51: 843-52.
8. Stratton JR, Lighty GW, Pearlman AS, Ritchie JL. Detection of left ventricular thrombus by two dimensional Echocardiography: Sensitivity, specificity and causes of uncertainty. *Circulation* 1982; 66: 156-66.
9. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J. Embolic potential of left ventricular thrombus after myocardial infarction. A two dimensional echocardiographic study of 119 patients. *J Am Coll Cardiol* 1985; 5: 1276-80.
10. Asinger RW, Mikell FL, Elsperger J, Hodges M. Incidence of left ventricular thrombosis after acute transmural myocardial infarction. Serial evaluation by two dimensional Echocardiography. *N Engl J Med* 1981; 305: 297-302.
11. Perdigo C, Ribeiro C. Intraventricular thrombi in acute myocardial infarction. The Iberian multicenter study. Presentation of the study protocol and various preliminary

- results. *Rev Port Cardiol* 1991; 10: 101-10.
12. Keren A, Goldberg S, Gottlieb S, Klein J, Schuger C, Medina A, Tzivoni D, Stern S. Natural history of left ventricular thrombi: their appearance and resolution in post hospitalization period of acute myocardial infarction. *J Am Coll Cardiol* 1990; 15: 790-800.
  13. Sipula J, Niederle P, Prochazka V, Vykopal M. The effect of thrombolytic therapy on the development of intraventricular thrombus in acute myocardial infarction. *Cor Vasa* 1990; 107-17.
  14. Lamas GA, Vaughan DE, Pfeffer MA. Left ventricular thrombus formation after first anterior wall acute myocardial infarction. *Am J Cardiol* 1988; 62: 31-5.
  15. Quinones MA, Nelson JC, Winters Jr. WL, Waggoner AD, Rosenfeld SP, Young JB, Miller RR. Clinical spectrum of left ventricular mural thrombi in a large cardiac population. Assessment by two dimensional echocardiography. *Am J Cardiol* 1980; 45: 135.
  16. Kupper AJ, Verheugt FW, Peels CH, Galema TW, Roos JP. Left ventricular thrombus incidence and behaviour studied by serial 2-dimensional echocardiography in acute anterior myocardial infarction. *J Am Coll Cardiol* 1989; 13: 1511-20.
  17. Jugdutt BI, Sivaram CA. Prospective two dimensional echocardiographic evaluation of left ventricular thrombus and embolism after acute myocardial infarction. *J Am Coll Cardiol* 1989; 13: 545-64.
  18. Chamsi-Pasha H, Barnes PC. Left ventricular thrombus in acute transmural myocardial infarction. *Postgrad Med J* 1988; 64: 188-90.
  19. Visser CA, Kan G, David GK, Lie KI, Durrer D. Two dimensional Echocardiography in the diagnosis of left ventricular thrombus. A prospective study of 67 patients with anatomic validation. *Chest* 1983; 83: 228-32.
  20. Bhatnagar SK, Al-Yusuf AR. Left ventricular blood flow analysis in patients with and without thrombus after first Q wave acute anterior myocardial infarction. Two dimensional Doppler echocardiographic study. *Angiology* 1992; 43: 188-91.
  21. Spirito P, Bellotti P, Chiarella F, Domenicucci S, Sementa A, Vecchio C. Prognostic significance and natural history of left ventricular thrombi in patients with acute anterior myocardial infarction: a two dimensional echocardiographic study. *Circulation* 1985; 72: 774-80.
  22. Vecchio C, Chiarella F, Lupi G, Bellotti P, Domenicucci S. Left ventricular thrombus in anterior acute myocardial infarction after thrombolysis. A GISSI-2 Connected study. *Circulation* 1991; 84: 512-19.
  23. Penco M, Romano S, Mallus MT, Dagianti A, Fedele F, Dagianti A. The role of compromised regional function of the left ventricle in the evolution of post infarct ventricular thrombosis. *Cardiology* 1993; 38: 157-61.
  24. Motro M, Barbash GI, Hod H, Kaplinski E, Laniadq S, Keven G. Incidence of left ventricular thrombi formation after thrombolytic therapy with recombinant tissue plasminogen activator, heparin and aspirin in patients with acute myocardial infarction. *Am Heart J* 1991; 122: 23-6.
  25. Reeder GS, Lengyel M, Tajik AJ, Smith HC, Danielson GK, Seward JB. Incidence of thrombus in left ventricular aneurysm; sensitivity of the angiogram and relation between anticoagulation and embolization. *Am J Cardiol* 1980; 45: 423.
  26. Bhatnagar SK, Al-Yusuf AR. Effects of intravenous recombinant tissue-type plasminogen activator therapy on the incidence and associations of left ventricular thrombus in patients with a first acute Q wave anterior myocardial infarction. *Am Heart J* 1991; 122: 1251-56.
  27. Sagie A, Strasberg B, Imbar S, Rechavia E, Sclarovsky S. Value of the electro cardiogram for prediction of left ventricular mural thrombus in anterior wall acute myocardial infarction. *Am J Cardiol* 1991; 68: 957-59.
  28. Johannessen KA, Nordrehaug JE, Vander Lippe G. Left ventricular thrombosis and cerebrovascular accident in acute myocardial infarction. *Br Heart J* 1981; 51: 553-6.
  29. Arvan S, Boscha K. Prophylactic anticoagulation for left ventricular thrombi after acute myocardial infarction: A prospective randomized trial. *Am Heart J* 1987; 113: 688-93.
  30. Domenicucci S, Chiarella F, Dellotti P, Lupi G, Scarci G, Vecchio C. Early appearance of left ventricular thrombi after anterior myocardial infarction; a marker of higher in hospital mortality in patients not treated with anti thrombotic drugs. *Eur Heart J* 1990; 11: 51-8.

#### The Authors:

Saulat Siddique  
Associate Professor,  
Department of Cardiology,  
Shaikh Zayed Hospital,  
**Lahore.**

Naeem Sajjad,  
Medical Officer,  
Department of Medicine,  
Shaikh Zayed Hospital,  
**Lahore.**

Zafar Iqbal,  
Professor,  
Department of Medicine,  
Shaikh Zayed Hospital,  
**Lahore.**

#### Address for Correspondence:

Saulat Siddique  
Associate Professor  
Department of Cardiology,  
Shaikh Zayed Hospital,  
**Lahore.**