Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.265983.3152 Volume 30, Issue 8.1, NOV. 2024, Supplement Issue
Manuscript ID: ZUMJ-2402-3170
DOI: 10.21608/ZUMJ.2024.269236.3170
ORIGINAL ARTICLE

Association between Mitral Regurgitation and Left Ventricular Mural Thrombi following Anterior ST Segment Elevation Myocardial Infarction

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Submit Date: 12-02-2024
Revise Date: 25-02-2024
Accept Date: 26-02-2024

ABSTRACT

Background: Acute myocardial infarction patients may be protected against left ventricular thrombus development by mitral regurgitation (MR). This study aimed to assess the incidence of left ventricular thrombosis in patients suffering from acute anterior myocardial infarction and determine if mitral regurgitation prevents the development of left ventricular mural thrombus. **Methods:** Seventy patients with anterior STEMI were enrolled in this prospective cohort study, they were divided into a non-LV thrombus group (n=55) and LV thrombus group (n=15), 2-dimensional and color Doppler echocardiography were done to all cases. **Results:** LV thrombus formation was significantly higher among patients presented with passed time anterior STEMI than those presented within 48 hours of chest pain onset (P value < 0.001). LV EDV and LV ESV were significantly higher among LV thrombus group than non-LV thrombus group (P value =0.032). The severity of MR was significantly lower in LV thrombus group compared to the non-LV thrombus group (P value =0.039). Pain to door time was able to significantly predict LV apical thrombus formation (P <0.001 and AUC = 0.875) at cut-off >32 hours with 80% sensitivity, 80% specificity, 52.2% PPV and 93.6% NPV. In Multivariate regression, pain to door, and severity of MR were independent predictors of LV thrombus formation (P value < 0.05). In univariate regression, ECG, pain to door, passed time (PCI) and (Conservative), LV EDV, LV ESV and severe grade of MR were independent predictors of LV thrombus formation (with P value <0.05). **Conclusion:** Pain to door, and severity of MR were independent predictors of development of LV thrombus while ECG, passed time PCI and Conservative, LV EDV and LV ESV were not. The associated mitral regurgitation could have a protective role against development of left ventricular mural thrombus following anterior STEMI.

Keywords: Mitral Regurgitation, Left Ventricular Mural Thrombi, Anterior ST Segment Elevation Myocardial Infarction

INTRODUCTION

Acute ST-segment elevation myocardial infarction (STEMI) poses a significant risk to human health and life due to its high death and morbidity rates. The incidence of STEMI continues to climb. Complications following a myocardial infarction continue to be a leading cause of death and disability, even though primary percutaneous coronary intervention (PCI) and dual antiplatelet treatment (DAPT) have increased survival rates in

patients with STEMI throughout the last two decades [1].

A major consequence of STEMI is the formation of left ventricular thrombosis (LVT), which has the potential to embolize and cause sudden death. At the same time, it adds unnecessary medical strain by extending patients' overall hospital stays and increasing the utilization of healthcare resources [2]. The presence of Virchow's triad—blood stagnation, endothelial damage, and an enhanced tendency of blood for coagulation—has been linked to thrombus formation within the heart chambers [3].

It is well-established that the development of LV thrombus is closely linked to decreased LV wall motion with apical segment involvement, especially following anterior MI. The left anterior descending coronary artery is the most common site of these infarcts. Nearby intracavitary blood movement is slow (stasis) in comparison to normal areas, and there are big regions of poorly contracting left ventricular muscle in these anteroapical infarcts. The relative stagnation of blood is believed to heighten the likelihood of thrombus development [4].

Patients with dilated cardiomyopathy who have moderate to severe mitral regurgitation are less likely to develop left ventricular thrombosis, according to current research. The mitral annulus or the entire left ventricle may experience higher early diastolic inflow velocities, which may explain why mitral regurgitation has a protective impact. The influence of mitral regurgitation on the formation of left ventricular thrombus in patients with acute myocardial infarction is not well-studied [5].

We hypothesized that the associated mitral regurgitation will have a protective role against left ventricular mural thrombus formation following anterior STEMI. Therefore, we did this work to assess LV systolic function and the degree of mitral regurgitation associated with it with and without LV thrombus formation following anterior STEMI, and to correlate between LV mural thrombus formation and mitral regurgitation following anterior STEMI

PATIENTS AND METHODS

Between March and December of 2023, we carried out this prospective cohort study on 70 patients who presented by Anterior STEMI. Patients were recruited from cardiology departments, Zagazig University Hospitals and National heart institute.

The seventy patients were categorized into 2 groups according to the presence of left ventricular mural thrombus following Anterior STEMI:

First group: 55 patients without LV thrombus following anterior STEMI. Second group: 15 patients with LV thrombus following anterior STEMI.

Following an explanation of the procedures and medical study, all participants were asked to sign an informed written consent form. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. This study was carried out after the approval of the Institutional Review Board (IRB) (#10460/26-2-2023).

Cases who were candidates with anterior STEMI and underwent primary percutaneous intervention, received thrombolytic therapy, anterior STEMI with late presentation "Passed-time Anterior STEMI.

Cases with the following characteristics were excluded: who had history of chronic anticoagulation, cardiogenic shock, and patients with associated valvular disease other than mitral regurgitation.

All cases were assessed for personal history, Cardiovascular risk factors like diabetes mellitus, dyslipidemia, smoking, hypertension, and their duration, and Previous cardiac history including previous coronary intervention and previous hospitalizations. With emphasis on: Total ischemic time, hypertension, diabetes mellitus, smoking, Dyslipidemia and history of coronary artery disease.

Clinical examination

All patients underwent: General examination and Local examination of the heart.

Laboratory investigations

CBC, Serum sodium and potassium, Creatinine, Urea: and Peak high-sensitivity Troponin [6].

Standard 12-leads ECG

All patients were placed in the supine position and an electrocardiogram (ECG) at a speed of 25 mm/s, with a filter range of 0.16-100 Hz and a height of 10 mm/mV, was taken using an electrocardiogram equipment (Philips®). Death, heart failure,

malignant arrhythmia, reinfarction, and post-MI angina were among the major adverse cardiac events (MACE) linked to ECG alterations.

Management Plan

Primary Percutaneous Coronary Intervention:

Mechanical revascularization utilising balloon angioplasty, coronary stents, aspiration thrombectomy, and other procedures is performed on patients who arrive with STEMI straight to the cardiac catheterization laboratory in a method known as primary percutaneous coronary intervention (PCI) [8].

Anterior STEMI with successful thrombolysis

When all of the following were present two hours following thrombolytic treatment, we said that clinical reperfusion had been successful (SCR): (1) substantial improvement in pain levels (a decrease of 5 on a 1–10 subjective scale), (2) a decrease of more than 50% in the total ST segment elevation, and (3) a rapid spike in creatine kinase levels at the beginning [9]. The definition of failed thrombolysis was an ST-segment resolution of less than 50% within 180 minutes of beginning thrombolytic treatment [10].

Rescue PCI was meant to be performed in an emergency situation when full-dose fibrinolysis failed and there were at least one of the following symptoms: persistent ischemic chest pain, unstable hemodynamics, ventricular tachyarrhythmias, and less than 50 % of the ST-segment resolution at 90 minutes [11].

Resting Transthoracic Echocardiography (TTE)

The procedure was carried out using transthoracic transducers operating at 2-2.5 MHZ using commercially available devices from General Electric called Vivid 3 and Vivid e (Norway). While the patient was in the supine or left lateral decubitus posture, an echocardiogram was taken. Two impartial expert operators, who were not privy to any other patient's data, performed conventional 2D, M-mode, and Doppler examinations with standard views for each patient. The following were administered to all patients:

When evaluating left ventricular systolic function and volumes, Simpson's biplane approach was used: [12]:

$$\text{LVEF: } \frac{SV}{EDV}\ x\ 100 = \frac{EDV-ESV}{EDV}\ x\ 100$$

American Society of Echocardiography and European Association of Cardiovascular Imaging-recognized normal ranges for left ventricular ejection fraction (LVEF) measured by two-dimensional echocardiography [12]:

LV thrombus on echocardiography was defined as a discrete echo dense mass in the left ventricle with defined margins that are distinct from the endocardium and seen throughout systole and diastole [13]. Mitral regurgitation was assessed based on Grades of mitral regurge described by Apostolakis & Baikoussis [14]

Assessment of the regional wall motion abnormalities: The absence or aberrant contractility of a specific area of the heart muscle is known as Regional Wall Motion Abnormality (RWMA). Traditional evaluation of RWMA relied on visual analysis of echocardiography recordings for signs of myocardial thickness and endocardial excursion [15].

STATISTICAL ANALYSIS

We used SPSS v26 for our statistical study (IBM Inc., Chicago, IL, USA). Mean and standard deviation (SD) were used to show quantitative variables, and an unpaired Student's t-test was used to compare the two groups. When applicable, the Chi-square test or Fisher's exact test were used to assess qualitative variables, which were provided as percentages and frequencies. Statistical significance was determined by a two-tailed P value less than 0.05 and a high significant P value less than 0. 001.

RESULTS

No statistically significant differences were found as regards age, gender, hypertension, DM, smoking, dyslipidemia and IHD history between the both groups, LV thrombus formation was significantly higher in patients presenting with passed time anterior STEMI than those presenting within 48 hours of chest pain onset (P value < 0.001), Pain to door time was significantly higher among LV thrombus patients than non-LV thrombus patients (P value < 0.001), Passed time (PCI) and Passed time (Conservative) were significantly higher among LV thrombus group than the non-LV thrombus group (P value=0.006 and 0.016 respectively) (Table 1).

The LV thrombus group had significantly higher peak CK-MB and Peak high sensitivity troponin than the non-LV thrombus group (P value <0.001). (Table 2).

The LV thrombus group had significantly higher LV EDV and LV ES than the non-LV thrombus group (P value =0.032). The LV thrombus group had significantly lower severity of MR was than the non-LV thrombus group (P value =0.039) (Table 3).

Heart failure and post-MI Angina were insignificantly different between both groups. Malignant Arrhythmia, reinfarction and death did not occur in any patient in both groups (Table 4).

ROC curve analysis was performed on pain to door time and revealed that it can significantly predict LV apical thrombus formation (P <0.001 and AUC = 0.875) at cut-off >32 hours with 80% sensitivity, 80% specificity, 52.2% PPV and 93.6% NPV (Table 5, Figure 1).

In univariate regression, ECG, pain to door, passed time (PCI) and (Conservative), LV EDV, LV ESV and Severity of MR were independent predictors of development of LV thrombus (P value <0.05). In Multivariate regression, pain to door, and severity of MR were independent predictors of development of LV thrombus (P value < 0.05) while ECG, passed time PCI and Conservative, LV EDV and LV ESV were not (Table 6).

Table 1: Demographic data, medical history, ECG, total ischemic time and managements of the studied groups

		Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
Age (years)	**Mean ± SD**	56.85 ± 11.15	60.4 ± 12.3	0.289
	Range	35 - 85	33 - 77	
Gender	**Male**	47 (85.45%)	11 (73.33%)	0.270
	Female	8 (14.55%)	4 (26.67%)	
Hypertension		25 (45.45%)	9 (60%)	0.318
DM		26 (47.27%)	6 (40%)	0.616
Smoking		39 (70.91%)	9 (60%)	0.420
Dyslipidemia		25 (45.45%)	10 (66.67%)	0.243
IHD history		3 (5.45%)	0 (0%)	1

		Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
ECG	**Anterior STEMI**	21 (38.2%)	3 (20%)	**<0.001***
	Passed time Anterior STEMI	11 (20%)	12 (80%)	
	Extensive Anterior STEMI	23 (41.8%)	0 (0%)	
Pain to door (hours)	**Mean ± SD**	20.36 ± 32.19	75.27 ± 54.71	**<0.001***
	Range	1 - 166	16 - 238	
Door to balloon (hours)	**Mean ± SD**	2.18 ± 0.84	1.80 ± 1.52	0.201
	Range	0 - 3.5	0 - 4	

	Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
PPCI	31 (56.36%)	4 (26.67%)	0.078
Passed time (PCI)	1 (1.82%)	4 (26.67%)	**0.006***
Passed time (Conservative)	6 (10.91%)	6 (40%)	**0.016***
Successful thrombolysis	14 (25.45%)	1 (6.67%)	0.164
Thrombolytic then Rescue PCI	3 (5.45%)	0 (0%)	1

DM: Diabetes mellitus. IHD: Ischemic heart disease, *: significant as P value≤0.05. ECG: Electrocardiogram, PPCI: Primary percutaneous coronary intervention.

Table 2: Laboratory investigation of the studied groups

		Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
Hemoglobin (g/dl)	**Mean ± SD**	13.43 ± 1.54	13.05 ± 1.51	0.403
	Range	9.5 - 16.4	9.1 - 15.4	
Platelets ($\times10^9$/L)	**Mean ± SD**	261.44 ± 73.35	247.73 ± 53.14	0.502
	Range	103 - 495	152 - 341	
TLC (10^3/µL)	**Mean ± SD**	10.87 ± 3.94	9.05 ± 2.85	0.098
	Range	4.5 - 20	5.6 - 14.8	
Peak CK-MB (ng/mL)	**Mean ± SD**	360.62 ± 34.8	567.67 ± 27.37	**<0.001***
	Range	317 - 429	523 - 614	
Peak high sensitivity troponin (ng/L)	**Mean ± SD**	2507.65 ± 271.62	3860.6 ± 512.17	**<0.001***
	Range	2039 - 3015	3196 - 5023	
Creatinine (mg/dL)	**Mean ± SD**	1.01 ± 0.84	1.21 ± 0.82	0.435
	Range	0.5 - 6.8	0.6 - 4.1	
Urea (mg/dL)	**Mean ± SD**	26.24 ± 10.77	25.94 ± 16.42	0.934
	Range	12 - 58	11.8 - 77.3	

*: significant as P value≤0.05. TLC: Total leucocyte count. CK-MB: creatine kinase-myocardial band.

Table 3: Echocardiographic assessment of the studied groups

LV EDV: Left ventricular end-diastolic volume. LV ESV: Left ventricular end-systolic volume. LVEF: Left ventricular ejection fraction. LA: Left atrium, PWT: Posterior wall thickness. IVST: Inter ventricular septal thickness. MR: Mitral regurgitation. *: significant as P value≤0.05

		Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
LV EDV (ml)	**Mean ± SD**	92.48 ± 18.81	104.43 ± 18.24	**0.032***
	Range	50.4 - 130	55.2 - 129	
LV ESV (ml)	**Mean ± SD**	53.87 ± 12.78	62.8 ± 14.19	**0.022***
	Range	31.5 - 92.1	34 - 87	
LVEF (%)	**Mean ± SD**	41.79 ± 5.57	40.16 ± 5.55	0.318
	Range	28.99 - 52.84	32.56 - 49.34	
LA diameter (mm)	**Mean ± SD**	37.75 ± 2.78	37.93 ± 3.31	0.825
	Range	30 - 43	32 - 43	
PWT (mm)	**Mean ± SD**	9.64 ± 1.42	9.8 ± 0.94	0.675
	Range	7 - 13	9 - 12	
IVST (mm)	**Mean ± SD**	9.4 ± 2.19	9.8 ± 1.08	0.497
	Range	1 - 13	8 - 12	
Grade of MR	**No MR**	9 (16.36%)	2 (13.33%)	**0.039***
	Mild	20 (36.36%)	12 (80%)	
	Moderate	22 (40%)	1 (6.67%)	
	Moderate to severe	3 (5.45%)	0 (0%)	
	Severe	1 (1.82%)	0 (0%)	

Table 4: Major adverse cardiovascular events of the studied groups

	Non-LV thrombus group (n=55)	LV thrombus group (n=15)	P value
Heart failure	14 (25.45%)	6 (40%)	0.269
Malignant Arrhythmia	0 (0%)	0 (0%)	---
Reinfarction	0 (0%)	0 (0%)	---
Post-MI Angina	9 (16.36%)	2 (13.33%)	1
Death	0 (0%)	0 (0%)	---

MI: Myocardial infarction.

Table 5: Roc curve of pain to door time to predict LV apical thrombus formation.

Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	P value
>32	80%	80%	52.2%	93.6%	0.875	**<0.001***

PPV: positive predictive value, NPV: negative predictive value, AUC: area under the curve. *: significant as P value≤0.05

Table 6: Univariate and multivariate regression analysis of predictors of LV thrombus formation.

	Univariate			Multivariate		
	Odds ratio	**95% CI**	**P**	**Odds ratio**	**95% CI**	**P**
ECG	16	3.83 - 66.67	**<0.001***	1.165	0.018-75.16	0.942
Pain to door	1.0314	1.012-1.05	**<0.001***	1.052	1.0038-1.103	**0.034***
Passed time (PCI)	19.63	1.998 -192.98	**0.01***	3.835	0.01-1457.37	0.657
Passed time (Conservative)	5.44	1.43-20.71	**0.01***	11.8553	0.25-560.22	0.2
Peak high sensitivity troponin	1.14	0.96-1.35	0.11	---	---	---
LV EDV	1.037	1.002-1.073	**0.03***	1.156	0.9296-1.439	0.191
LV ESV	1.05	1.005-1.098	**0.02***	0.856	0.6362-1.154	0.309
Severity of MR	4.92	1.52-2.35	**0.039***	1.452	1.012-1.958	**0.023***

*Significant as P value≤0.05, ECG: Electrocardiogram. PCI: Percutaneous coronary intervention. LV EDV: Left ventricular end-diastolic volume. LV ESV: Left ventricular end-systolic volume.MR: Mitral regurgitation.

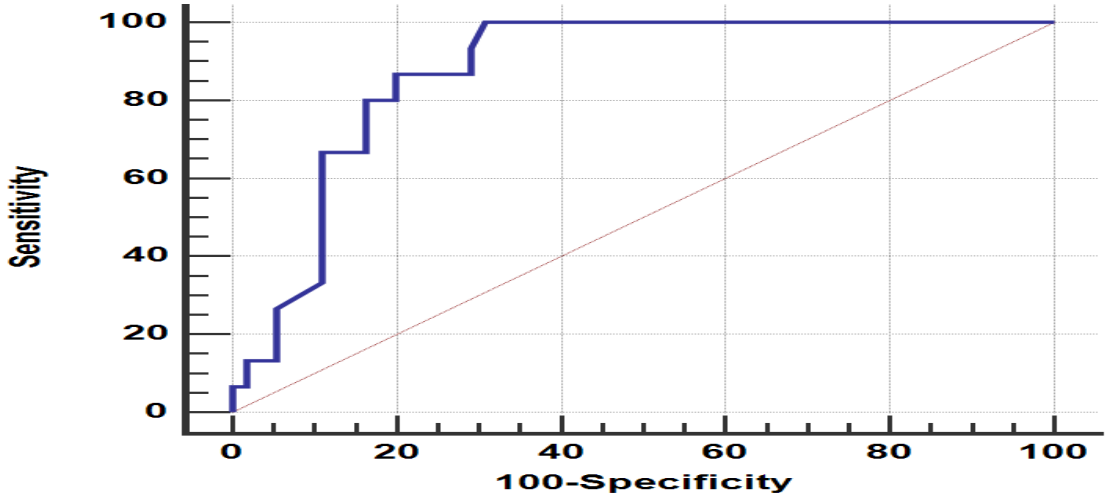

Figure 1: ROC curve of pain to door time to predict LV apical thrombus formation

DISCUSSION

Researchers suggest that mitral regurgitation can prevent left ventricular thrombus formation in people who have suffered an acute myocardial infarction. The mitral regurgitation's protective impact could be due to reduced left ventricular stasis or higher early diastolic inflow velocities throughout the entire left ventricle [5].

In the present study, No statistically significant differences were found as regards age, gender, hypertension, DM, smoking, dyslipidemia and IHD history between the both groups. That means that these factors are not considered as risk factors for thrombus formation.

In contrast to our findings, age seemed to be a risk factor for thrombus formation as detected by Ascione et al. [16] who studied the incidence and risk factors of left ventricular thrombosis in emergency department patients suffering from myocardial infarction. A total of 64 individuals (8% of the total) had left ventricular thrombosis found on at least one test. Left ventricular thrombosis patients had an older mean age (SD) of 64.6 vs. 59.8 years for the other 693 patients. In addition, age and gender showed significant differences in a study performed by Guo et al. [17] explored the associated variables of LVT development during two weeks in individuals with acute (STEMI), The mean age was 56.42 years, with 46 male cases accounting for 88.5% of the total.

In the present study, LV thrombus formation was significantly higher among patients presenting with passed time anterior STEMI than those presenting within 48 hours of chest pain onset (P value < 0.001). Compared to the non-LV thrombus group, the pain to door time in the LV thrombus group was substantially higher (P value < 0.001).

Similar findings were obtained by Cruz Rodriguez et al. [18] who conducted a narrative literature review that revealed a decline in the occurrence of left ventricular thrombosis (LVT) after a myocardial infarction (MI), likely because of enhanced patient care brought about by more effective and earlier reperfusion procedures. They concluded that Lower ejection fraction and anterior ST-segment elevation myocardial infarction (STEMI) patients are at increased risk for left ventricular thrombosis.

Also, Meurin et al. [19] aimed to analyze the prevalence and development of LV thrombi in a high-risk group of patients experiencing LV systolic dysfunction following anterior myocardial infarction (ant-MI). At a median of 12.0 days following ant-MI, 26 patients (26 percent) had thrombi detected. When compared to CMR-DE, TTE had a sensitivity of 94.7% and a specificity of 98.5% for detecting LV thrombi. Thrombi were mostly eliminated with triple antithrombotic treatment. After anti-MI treatment, systolic dysfunction commonly leads to left ventricular thrombus.

This was opposite to Guo et al. [17] who compared onset to door, door to balloon, onset to balloon time between cases with LVT and non-LVT group. The percentage of anti-platelet therapy and anticoagulant therapy was similar between the two groups. Both groups had comparable times from beginning to end in terms of start to door, door to balloon, and onset to

balloon, according to subgroup analysis in patients undergoing primary PCI. Nevertheless, these patients have a reduced risk of LVT if they have a history of angina, collateral circulation of the coronary arteries, primary percutaneous coronary intervention (PCI), or venous thrombolysis.

In the present study, PCI, successful thrombolysis and thrombolytic then rescue PCI were insignificantly different between both studied groups. Passed time (PCI) and Passed time (Conservative) were significantly higher among LV thrombus group compared to the non-LV thrombus group (P value=0.006 and 0.016 respectively).

Similarly, in a retrospective, single center study was conducted by Gianstefani et al. [20] showed that lower left ventricular ejection fraction (LVEF) (47 percent vs. 35 percent, P<0.01), anterior myocardial infarction (88 percent vs. 42 percent, P<0.01), and apical akinesis were more common in patients with LVT (irrespective of the vascular location of the MI). Possible associations with bigger infarcted regions may have been suggested by the questionable statistical relevance of symptoms to balloon time and the use of IIb/IIIa inhibitors in predicting the occurrence of LVT.

Furthermore, another single center study on STEMI patients was conducted by Mao et al. [21], found that the prevalence of LVT was as low as 1.6%. Twenty-eight individuals, or 1.6% of the total, had left ventricular thrombus. Significantly fewer patients (1.6%) were found to have LV thrombus in this large STEMI cohort compared to earlier research. Most echocardiograms made use of a UCA, which enhances the precision of detecting and excluding out LV thrombus.

In the present study, Culprit vessel and number of diseased vessels, Hemoglobin, platelets, TLC, creatinine and urea were insignificantly different between both groups. Whereas the LV thrombus group had significantly higher peak CK-MB and Peak high sensitivity troponin than the non-LV thrombus group (P value <0.001).

These findings were consistent with Khaled et al. [22] who analysed data from 308 consecutive patients who underwent primary percutaneous coronary intervention after presenting with acute ST-elevation myocardial infarction. Coronary artery disease with many vessels and the early invasive technique used (culprit lesion only vs. full revascularization) did not predict LVT.

On the other hand, Shacham et al. [23] stated that it has been observed that patients with LVT exhibit noticeably elevated levels of C-reactive protein, fibrinogen, leukocytes, and platelets compared to MI patients who do not have LVT.

In the present study, the LV thrombus group had significantly higher LV EDV and LV ES than the non-LV thrombus group (P value =0.032). The LV thrombus group had significantly lower severity of MR was than the non-LV thrombus group (P value =0.039). LVEF, LA diameter, PWT and IVST were insignificantly different between both groups.

This agreed with Guo et al. [17] who compared the factors that were associated with the formation of left ventricular thrombus (LVT) within two weeks in patients with ST-elevation myocardial infarction (STEMI) and left ventricular aneurysm (LVA): left ventricular ejection fraction (LVEF) ≤40 percent, preoperative thrombosis in myocardial infarction (TIMI) blood flow grade 0 and postoperative TIMI blood flow grade 2.

Shi et al. [24] also included 111 STEMI patients in a retrospective investigation. Longitudinal strain (LTF) development was associated with acute stage left ventricular ejection fraction (LVEF). In the chronic phase, 29 out of 38 individuals had LVT resolve (76 percent). Compared to individuals whose LVT had resolved, those whose LVT remained had a larger left ventricle, a lower LVEF, and a higher ECV during the acute phase. The left ventricular function, amount of myocardial infarction, strain, and T1 and ECV values were all worse in STEMI patients who developed LVT compared to those who did not. There was a correlation between LVT development and both LVEF and global longitudinal strain during the acute stage. Acute STEMI patients who underwent LVT were more likely to experience unfavourable outcomes during the long-term follow-up.

In the present study, ROC curve analysis was performed on pain to door time and revealed that it can significantly predict LV apical thrombus formation (P <0.001 and AUC = 0.875) at cut-off >32 hours with 80% sensitivity, 80% specificity, 52.2% PPV and 93.6% NPV.

Similarly, Olsen et al. [25] revealed in univariable analyses, LVT was linked with anterior infarcts, previous MI, poorer LVEF, lower e′, lower GLS, and regional strain; these factors were also more common in patients with LVT. When controlling for other variables such as anterior infarcts, previous MI, left ventricular ejection fraction (LVEF), and e′, the independent associations between LVT development, midventricular strain, and apical strain persisted in the multivariable analysis. The combined diagnostic model has a sensitivity of one hundred percent, a negative predictive value of thirty-eight percent, and a specificity of thirty-eight percent for anterior infarct, diminished left ventricular ejection fraction (LVEF) below 42 percent, and apical strain, respectively. It is possible to rule out the formation of LVT in individuals with MI if there is no anterior infarct, preserved LVEF, and apical strain. Apical strain reduction significantly raises the risk of LVT.

In the present study, univariate regression, ECG, pain to door, passed time (PCI) and (Conservative), LV EDV, LV ESV and severe grade of MR were independent predictors of development of LV thrombus (P value <0.05). In Multivariate regression, pain to door, and severity of MR were independent predictors of development of LV thrombus (P value < 0.05) while ECG, passed time PCI and Conservative, LV EDV and LV ESV were not.

This agreed with Guo et al. [17] who revealed that in patients with acute STEMI and LVA, LVT was positively connected with a history of heavy drinking, anemia, LVEF≤40 percent, and a preoperative TIMI blood flow grade of 0. Patients with acute STEMI and LVA were found to have a lower risk of LVT if they had a history of angina, collateral circulation of the coronary arteries, primary PCI, or have undergone venous thrombolysis. Patients with acute STEMI and LVA at early stages of the disease are at a higher risk of LVT if they had a history of heavy drinking, anemia, LVEF ≤40 percent, or a preoperative TIMI blood flow grade of 0. Nevertheless, these patients have a reduced risk of LVT if they have a history of angina, collateral circulation of the coronary arteries, primary percutaneous coronary intervention (PCI), or venous thrombolysis.

Similarly, a retrospective study performed by Shi et al. [24] They found that compared to STEMI patients without LVT, those with STEMI with LVT had lower left ventricular function, more severe myocardial infarction, strain, and higher T1 and ECV values. From the very first week onward, the functional and mapping metrics of the chronic stage patients who still received LVT were much worse. There was a correlation between LVT development and LVEF and global longitudinal strain independently throughout the acute period. Longitudinal follow-up in patients with acute STEMI revealed that LVT, aneurysm, and increased myocardial T1 were risk factors for unfavorable outcomes.

Also, Wang et al. [2] revealed that in patients with anterior STEMI, the total pooled incidence was 10.0 percent, and in acute STEMI treated by primary PCI, the incidence of LVT was 4%. Risk factors for post-PCI LVT included anterior STEMI, left anterior descending-related infarct, anomalies in left ventricular wall motion, and lower LVEF. Within the PCI era, there was still a high prevalence of LVT following acute STEMI.

In contrast to our findings, Ascione et al. [16] revealed that on stepwise multiple logistic regression analysis, The anterior site of infarction and the amount of wall motion asynergy were the only independent variables that were found to be associated to the presence of left ventricular thrombosis. Having moderate to severe mitral regurgitation early on after an acute myocardial infarction may not decrease the risk of left ventricular thrombosis and may even enhance it. The magnitude of the akinetic-dyskinetic area seen on echocardiography within 24-48 hours from the beginning of symptoms is the independent determinant of left ventricular thrombosis.

Furthermore, other risk factors were identified in a study conducted by Garber et al. [26] who found that LVT was independently predicted by a faster heart rate, non-white race, the severity of HF, and the existence of left anterior descending artery (LAD) disease. While no statistically significant link was found, there was a trend suggesting that LVT may increase the risk of death from any cause. In this modern post-STEMI group, more than 4% experienced LVT. Independently linked with LVT were several baseline factors, including heart rate, severity of HF, LAD disease, and non-white race.

Limitations

Several limitations should be acknowledged in our study. First, the sample size, though adequate for initial insights, may not fully represent the diversity

of patients with Anterior ST Segment Elevation Myocardial Infarction. Additionally, the single-center nature of the study could introduce selection bias. We didn't clarify the etiology of mitral regurgitation whether primary (e.g. degenerative disease, rheumatic activity, mitral valve prolapse or primary ischemic MR resulting from papillary muscle ischemia and dysfunction) or secondary to LV poor ejection fraction and remodeling with mitral valve annular dilatation. Prospective studies are needed to determine whether anticoagulation in patients at increased risk for LVT improves outcomes.

CONCLUSION

In conclusion, pain to door, and severity of MR were independent predictors of LV thrombus formation while ECG, passed time PCI and Conservative, LV EDV and LV ESV were not. The associated mitral regurgitation may have a protective role against left ventricular mural thrombus formation following anterior STEMI.

CONFLICTS OF INTEREST

No potential conflict of interest was reported by the authors.

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Citation

Ibrahim, M., Kandil, N., El Zayat, A., El-Shetry, M. Association between Mitral Regurgitation and Left Ventricular Mural Thrombi following Anterior ST Segment Elevation Myocardial Infarction. ***Zagazig University Medical Journal*****, 2024; (3784-3794): -. doi: 10.21608/zumj.2024.269236.3170**