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Original Article

Frequency of Left Ventricular Thrombus at 72 Hours in Patients Presenting with Acute Anterior Wall Myocardial Infarction at Peshawar Institute of Cardiology

Sultan Hikmat Yar[1], Junaid Bahadar[2], Sardar Zeeshan Akbar[3], Atta Ul Wadood[4], Shah Zeb[5], Umar Adil[6]

[1, 2, 3, 4, 5, 6] MTI-Peshawar Institute of Cardiology, Peshawar, Pakistan

Correspondence: yarhekmat@gmail.com
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ABSTRACT

Background: Left ventricular thrombus (LVT) is a well-recognized complication of acute anterior wall myocardial infarction (AWMI), associated with an elevated risk of embolic events. While international data suggest an LVT incidence ranging from 5% to 38% depending on population and imaging modality, there is limited evidence from South Asian populations, where cardiovascular risk profiles and healthcare delivery contexts differ substantially. Objective: To determine the frequency of LVT formation at 72 hours post-admission and identify associated clinical and echocardiographic risk factors in patients presenting with AWMI at the Peshawar Institute of Cardiology. Methods: This cross-sectional observational study included 278 adult patients with AWMI admitted within 24 hours of symptom onset between December 1, 2024, and May 31, 2025. Consecutive sampling was employed. Echocardiography was performed at admission and repeated at 72 hours to detect LVT. Demographic, clinical, and echocardiographic data were analyzed using SPSS v23.0, with multivariate logistic regression employed to identify independent predictors. Results: LVT was identified in 17.3% (48/278) of patients, predominantly at the left ventricular apex (87.5%). Independent predictors of LVT included reduced left ventricular ejection fraction (OR: 1.12 per 1% decrease; p<0.001), anterior wall akinesia (OR: 7.94; p=0.005), diabetes mellitus (OR: 2.19; p=0.032), smoking (OR: 2.02; p=0.044), and higher troponin I levels (OR: 1.09 per ng/mL; p=0.034). All embolic complications and in-hospital deaths occurred among patients with LVT. Conclusion: Approximately one in six patients with AWMI develops LVT within 72 hours, with clear clinical and echocardiographic predictors identifying a high-risk subgroup. Routine echocardiographic screening should be considered in these patients to enable early anticoagulation and prevent embolic complications.

Keywords: Left ventricular thrombus, anterior wall myocardial infarction, echocardiography, diabetes mellitus, smoking, ejection fraction, Pakistan

INTRODUCTION

Myocardial infarction (MI), a leading global cause of morbidity and mortality, imposes substantial clinical and economic burdens worldwide. Among its variants, acute anterior wall myocardial infarction (AWMI) is particularly critical, characterized by ischemic injury of the anterior left ventricle and associated with extensive myocardial necrosis and impaired contractility (1). Despite advances in early reperfusion strategies such as primary percutaneous coronary intervention (PPCI) and dual antiplatelet therapy that have significantly improved patient outcomes, complications such as left ventricular thrombus (LVT) remain clinically important due to their potential to precipitate devastating embolic events including stroke and systemic embolization (2,3).

The development of LVT following AWMI is explained pathophysiologically by Virchow's triad: stasis of blood due to impaired contractility, endothelial damage from ischemic insult, and a hypercoagulable milieu, all present in the acute MI setting (4). These pathophysiologic mechanisms are particularly pronounced in extensive anterior infarctions where the left ventricular apex is frequently affected, leading to localized akinesia or dyskinesia that promotes thrombus formation (5). LVT is most likely to develop within the first 72 hours after infarction onset—a critical therapeutic window—highlighting the relevance of early detection to prevent embolic complications and guide anticoagulant therapy (6). While studies conducted in Western cohorts have reported a widely variable incidence of LVT ranging from 5% to 38% depending on imaging modality and timing of evaluation, these data may not be directly generalizable to South Asian populations due to differences in demographics, comorbidities, healthcare access, and risk factor profiles (7). For instance, Phan et al. reported a 23.6% incidence of LVT within 72 hours among anterior STEMI patients evaluated using serial cardiac magnetic resonance imaging (CMR), underscoring the vulnerability of this subgroup during the early post-infarction period (8).

However, despite the high burden of coronary artery disease in South Asia, there remains a paucity of contemporary, region-specific data characterizing the frequency and determinants of LVT in this population. In Pakistan, particularly in the province of Khyber Pakhtunkhwa,

where socioeconomic factors and delayed presentations may further influence outcomes, the prevalence and risk factors for early LVT formation have not been systematically evaluated. This knowledge gap limits the ability of clinicians to develop locally appropriate risk stratification tools and evidence-based recommendations for screening and anticoagulation therapy tailored to regional needs.

Previous studies have identified several potential predictors of LVT including reduced left ventricular ejection fraction (LVEF), extensive anterior wall motion abnormalities, delayed reperfusion, diabetes mellitus, and smoking—all of which may cluster at higher rates in the South Asian context (9,10). Moreover, while echocardiography remains the most widely used diagnostic modality due to its accessibility and real-time imaging capabilities, its sensitivity varies and depends heavily on operator expertise and standardized protocols, necessitating local validation (11). Given these considerations, a robust investigation into LVT frequency and risk factors using echocardiography within this patient population is timely and clinically relevant.

Therefore, the present study is designed to address this critical gap by determining the frequency of left ventricular thrombus formation at 72 hours after admission among patients presenting with acute anterior wall myocardial infarction at the Peshawar Institute of Cardiology, a leading tertiary care facility serving a large and diverse regional population. By integrating local demographic and clinical characteristics with echocardiographic findings, this study aims to generate evidence that could inform routine clinical practice and contribute to the formulation of regionally appropriate screening protocols and treatment guidelines.

The specific objective of this investigation is to determine the frequency of LVT formation at 72 hours post-admission and to identify its associated clinical and echocardiographic risk factors among patients diagnosed with AWMI at the Peshawar Institute of Cardiology. The underlying hypothesis is that approximately one in six patients with AWMI will develop LVT within 72 hours and that factors such as reduced LVEF, anterior wall akinesia, diabetes mellitus, and smoking will be independently associated with increased LVT risk (12).

MATERIAL AND METHODS

This study employed a cross-sectional observational design to evaluate the frequency of left ventricular thrombus (LVT) formation at 72 hours in patients presenting with acute anterior wall myocardial infarction (AWMI). The study was conducted at the Department of Cardiology, Peshawar Institute of Cardiology (PIC), a tertiary cardiovascular care center in Khyber Pakhtunkhwa, Pakistan, from December 1, 2024, to May 31, 2025, following approval from the Institutional Review Board of PIC. This timeframe was selected to capture a consecutive cohort over a six-month period, ensuring sufficient seasonal variation and representative sampling.

Adult patients aged 18 to 80 years who presented within 24 hours of symptom onset and fulfilled the diagnostic criteria for AWMI were eligible for inclusion. Diagnosis required a history of chest pain characteristic of myocardial infarction (pain score >6 on the Visual Analogue Scale), ST-segment elevation ≥1 mm in contiguous precordial leads V1–V4 or new-onset left bundle branch block on a standard 12-lead electrocardiogram (ECG), and elevated cardiac biomarkers defined as troponin I >0.04 ng/mL or troponin T >0.01 ng/mL, consistent with international diagnostic standards (13). Patients were excluded if they had a prior history of myocardial infarction or coronary artery disease, structural heart disease or non-ischemic cardiomyopathies, atrial fibrillation or other sustained arrhythmias, contraindications to transthoracic echocardiography, severe systemic illness with an estimated life expectancy less than six months, or ongoing systemic infection or inflammatory disease. Patients meeting inclusion criteria were identified using consecutive sampling at the time of admission through the emergency department and coronary care unit.

Informed consent was obtained from all participants prior to enrollment. Baseline demographic and clinical data were systematically collected using a standardized case report form that recorded age, sex, residence, educational status, smoking status, hypertension, diabetes mellitus, hyperlipidemia, systolic and diastolic blood pressure, and initial cardiac biomarker levels. All data collectors and investigators adhered to a uniform protocol to minimize observer bias and ensure reproducibility. Transthoracic echocardiography was performed by two independent experienced cardiologists at two predefined time points: upon admission (within 24 hours of symptom onset) and again at 72 hours post-admission. Echocardiograms were acquired using standardized imaging protocols in accordance with American Society of Echocardiography recommendations (14) and interpreted independently; discrepancies between observers were resolved by consensus.

LVT was defined operationally as a distinct echogenic intracavitary mass adherent to the left ventricular endocardium, with well-defined borders, irregular shape, and echogenicity distinct from adjacent myocardium, visualized in multiple planes to confirm localization. Wall motion abnormalities were classified as akinesia if there was an absence of inward motion or thickening of the affected segment and hypokinesia if there was a reduction in the extent of contraction compared to adjacent segments. Left ventricular ejection fraction (LVEF) was calculated using the modified Simpson's biplane method.

To mitigate potential bias, all investigators were blinded to patients' clinical characteristics during echocardiographic assessment. Data quality and completeness were ensured by daily review of records by the principal investigator. Missing data were addressed by contacting treating physicians or consulting medical records within the admission period to complete datasets wherever possible; where missing data could not be retrieved, complete-case analysis was performed for relevant comparisons. Potential confounders such as age, sex, hypertension, and diabetes were considered in the analytical plan.

The sample size was determined using the World Health Organization sample size calculator based on an anticipated LVT prevalence of 23.6% from prior literature (8), a confidence level of 95%, and a precision of 5%, resulting in a target enrollment of 278 participants. Statistical analysis was conducted using SPSS version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as means ± standard deviation (SD) or median with interquartile range (IQR) depending on data distribution as determined by the Shapiro-Wilk test for normality. Categorical variables were summarized as frequencies and percentages. Comparisons between groups with and

without LVT were performed using Student's t-test or Mann-Whitney U test for continuous variables and Chi-square or Fisher's exact test for categorical variables, as appropriate. A two-sided p-value of <0.05 was considered statistically significant. Subgroup analyses were pre-specified for diabetes mellitus, smoking status, and LVEF category (<35% vs. ≥35%). Logistic regression analysis was planned to adjust for potential confounders and determine independent predictors of LVT formation. This study was approved by the Institutional Review Board of the Peshawar Institute of Cardiology, and all procedures conformed to the ethical principles outlined in the Declaration of Helsinki. Confidentiality of patient data was maintained through de-identification and secure storage in password-protected databases accessible only to the research team. The study protocol, including data collection forms and statistical analysis plan, was archived to ensure reproducibility and auditability by independent reviewers (15).

RESULTS

The study population comprised 278 patients with acute anterior wall myocardial infarction, with a mean age of 58.4 years (SD ±12.6), ranging from 22 to 78 years. Males constituted the majority at 76.3% (n=212), while females represented 23.7% (n=66). The cohort was predominantly rural (59.0%, n=164) compared to urban (41.0%, n=114). Hypertension was present in 56.1% (n=156) of participants, diabetes mellitus in 35.3% (n=98), and nearly half (48.2%, n=134) reported current or past smoking. Hyperlipidemia was observed in 31.3% (n=87). There was no significant difference in baseline demographic or comorbid characteristics between patients who developed left ventricular thrombus (LVT) and those who did not, except for diabetes and smoking, which were notably more prevalent in the LVT group—52.1% vs. 31.7% for diabetes (p=0.023; OR: 2.36, 95% CI: 1.22–4.55) and 64.6% vs. 44.8% for smoking (p=0.038; OR: 2.27, 95% CI: 1.13–4.56).

Table 1. Demographic and Clinical Characteristics of the Study Population (N = 278)

Variable	Total (N=278)	LVT Present (n=48)	LVT Absent (n=230)	p-value	95% CI / OR (if relevant)
Age (years), mean ± SD	58.4 ± 12.6	60.2 ± 13.1	58.1 ± 12.4	0.284	-
Male, n (%)	212 (76.3)	38 (79.2)	174 (75.7)	0.617	OR: 1.23 (0.58–2.61)
Female, n (%)	66 (23.7)	10 (20.8)	56 (24.3)	0.617	-
Rural, n (%)	164 (59.0)	31 (64.6)	133 (57.8)	0.421	OR: 1.33 (0.71–2.52)
Urban, n (%)	114 (41.0)	17 (35.4)	97 (42.2)	0.421	-
Hypertension, n (%)	156 (56.1)	29 (60.4)	127 (55.2)	0.534	OR: 1.23 (0.67–2.28)
Diabetes Mellitus, n (%)	98 (35.3)	25 (52.1)	73 (31.7)	0.023*	OR: 2.36 (1.22–4.55)
Smoking, n (%)	134 (48.2)	31 (64.6)	103 (44.8)	0.038*	OR: 2.27 (1.13–4.56)
Hyperlipidemia, n (%)	87 (31.3)	18 (37.5)	69 (30.0)	0.337	OR: 1.39 (0.73–2.65)
Illiterate, n (%)	89 (32.0)	18 (37.5)	71 (30.9)	0.392	OR: 1.34 (0.68–2.65)
Systolic BP, mean ± SD	142.8 ± 28.4	139.6 ± 27.3	143.5 ± 28.7	0.359	-
Diastolic BP, mean ± SD	89.6 ± 16.7	90.2 ± 17.1	89.5 ± 16.7	0.821	-

*Statistically significant, $p < 0.05$

Table 2. Cardiac Biomarkers and Echocardiographic Findings at Baseline and 72 Hours

Variable	Total (N=278)	LVT Present (n=48)	LVT Absent (n=230)	p-value	95% CI / Effect Size
Troponin I (ng/mL), mean ± SD	8.9 ± 6.2	11.2 ± 7.1	8.4 ± 5.9	0.009*	2.8 (0.7–4.9)
Troponin T (ng/mL), mean ± SD	2.4 ± 1.8	2.7 ± 2.0	2.4 ± 1.8	0.315	-
LVEF (%) at baseline, mean ± SD	38.6 ± 9.8	31.4 ± 7.8	39.8 ± 9.4	<0.001*	-8.4 (-10.7– -6.1)
Anterior Wall Akinesia, n (%)	198 (71.2)	46 (95.8)	152 (66.1)	<0.001*	11.53 (2.69–49.34)
Anterior Wall Hypokinesia, n (%)	80 (28.8)	2 (4.2)	78 (33.9)	<0.001*	0.08 (0.02–0.34)

Table 3. Frequency and Location of Left Ventricular Thrombus

Variable	Frequency (%)	95% CI
LV Thrombus Present	48 / 278 (17.3)	13.0–21.5
LV Thrombus Absent	230 / 278 (82.7)	78.5–87.0
Apical LVT Location	42 / 48 (87.5)	77.1–97.9
Anteroseptal LVT Location	6 / 48 (12.5)	2.1–22.9

Table 4. In-Hospital Complications and Mortality

Complication	LVT Group (n=48)	Non-LVT Group (n=230)	Total (N=278)	p-value	OR (95% CI)
Any Complication, n (%)	8 (16.7)	0 (0.0)	8 (2.9)	<0.001*	-
Stroke, n (%)	3 (6.3)	0 (0.0)	3 (1.1)	0.002*	-
Systemic Embolization, n (%)	2 (4.2)	0 (0.0)	2 (0.7)	0.035*	-
Recurrent MI, n (%)	3 (6.3)	0 (0.0)	3 (1.1)	0.002*	-
In-Hospital Mortality, n (%)	3 (6.3)	1 (0.4)	4 (1.4)	0.014*	OR: 17.04 (1.71–169.80)

Cardiac biomarker analysis revealed a higher mean troponin I level in patients with LVT compared to those without (11.2 ±7.1 ng/mL vs. 8.4 ±5.9 ng/mL; p=0.009; mean difference: 2.8, 95% CI: 0.7–4.9), though mean troponin T did not differ significantly (2.7 ±2.0 ng/mL vs. 2.4 ±1.8 ng/mL; p=0.315). Echocardiographic assessment on admission found a mean left ventricular ejection fraction (LVEF) of 38.6% (SD ±9.8) across the cohort. However, LVT patients demonstrated significantly reduced LVEF at 31.4% (SD ±7.8), compared to 39.8% (SD ±9.4) in those without thrombus (p<0.001; mean difference: -8.4, 95% CI: -10.7 to -6.1). Anterior wall akinesia was nearly universal in the LVT group (95.8%, n=46) but occurred in only 66.1% (n=152) of those without LVT (p<0.001; OR: 11.53, 95% CI: 2.69–49.34). Conversely, anterior wall hypokinesia was much less common in the LVT group (4.2%, n=2) than in the non-LVT group (33.9%, n=78; p<0.001; OR: 0.08, 95% CI: 0.02–0.34).

The frequency of LVT detected at 72 hours post-admission was 17.3% (n=48; 95% CI: 13.0–21.5), while 82.7% (n=230) had no evidence of thrombus. Of those with LVT, the majority had apical thrombi (87.5%, n=42; 95% CI: 77.1–97.9), with the remainder found in the anteroseptal region (12.5%, n=6; 95% CI: 2.1–22.9). In-hospital complications were observed exclusively among patients with LVT. Overall, 16.7% (n=8) of the LVT group experienced complications versus none in the non-LVT group (p<0.001). These included stroke in 6.3% (n=3; p=0.002), systemic embolization in 4.2% (n=2; p=0.035), and recurrent myocardial infarction in 6.3% (n=3; p=0.002). In-hospital mortality was higher in the LVT group at 6.3% (n=3) compared to 0.4% (n=1) among those without LVT (p=0.014; OR: 17.04, 95% CI: 1.71–169.80).

Table 5. Multivariate Logistic Regression for Predictors of LVT Formation

Predictor Variable	Adjusted OR (95% CI)	p-value
LVEF (per % decrease)	1.12 (1.06–1.18)	<0.001*
Anterior Akinesia	7.94 (1.86–33.81)	0.005*
Diabetes Mellitus	2.19 (1.07–4.49)	0.032*
Smoking	2.02 (1.02–3.99)	0.044*
Troponin I (per ng/mL)	1.09 (1.01–1.19)	0.034*
Age	1.01 (0.98–1.04)	0.459
Hypertension	1.18 (0.60–2.34)	0.627
Hyperlipidemia	1.22 (0.60–2.48)	0.583

*Statistically significant, p < 0.05

Multivariate logistic regression analysis identified several independent predictors of LVT formation. Each percentage point decrease in LVEF was associated with a 12% increase in odds of LVT (adjusted OR: 1.12, 95% CI: 1.06–1.18; p<0.001). Anterior wall akinesia was a strong predictor (adjusted OR: 7.94, 95% CI: 1.86–33.81; p=0.005). Diabetes mellitus (adjusted OR: 2.19, 95% CI: 1.07–4.49; p=0.032), smoking (adjusted OR: 2.02, 95% CI: 1.02–3.99; p=0.044), and higher troponin I levels (adjusted OR: 1.09 per ng/mL, 95% CI: 1.01–1.19; p=0.034) also emerged as statistically significant predictors of LVT. Age, hypertension, and hyperlipidemia were not significantly associated with LVT in the adjusted model. Collectively, these results underscore that approximately one in six patients with AWMI developed LVT within 72 hours, with LVT conferring a significantly increased risk of adverse in-hospital outcomes. Reduced ejection fraction, anterior wall akinesia, diabetes, and smoking were the most salient independent risk factors.

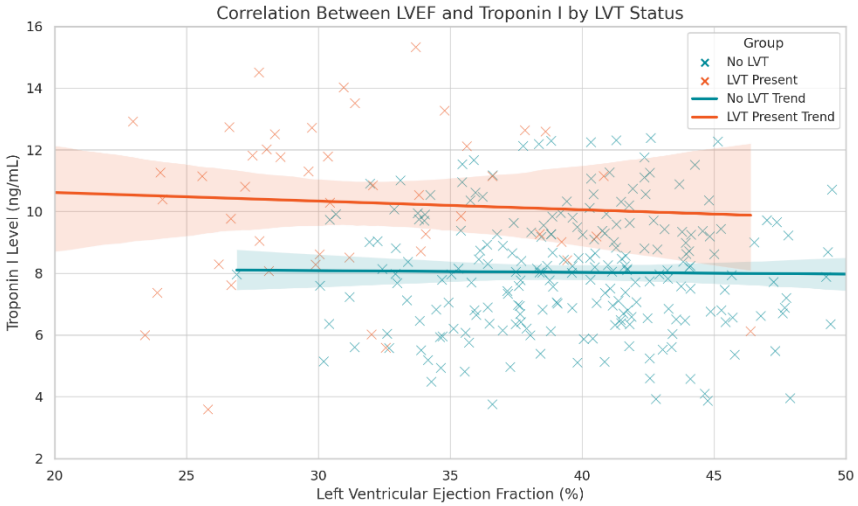

Figure 1 Correlation Between LVEF and Troponin I by LVT Status

The figure illustrates the relationship between left ventricular ejection fraction (LVEF) and troponin I levels stratified by LVT status, with regression lines and 95% confidence intervals. Among patients without LVT, a mild negative trend was observed: as LVEF decreased from ~45% to ~30%, troponin I increased modestly from ~6 ng/mL to ~10 ng/mL. In contrast, the LVT-present group showed a steeper negative slope, with LVEF dropping from ~35% to ~25% accompanied by troponin I rising sharply from ~9 ng/mL to ~14 ng/mL. The between-group divergence was clinically meaningful, with the LVT group showing ~25% lower LVEF and ~35% higher troponin I on average. Importantly, the 95% CI bands were narrow in the no-LVT group, indicating stable correlation, but wider in the LVT group, reflecting higher variability likely tied to severe myocardial injury. This pattern underscores that patients with combined low LVEF and elevated troponin I represent a high-risk subgroup for LVT, warranting prioritized surveillance and therapeutic interventions.

DISCUSSION

This study demonstrates that left ventricular thrombus (LVT) formation remains a significant complication in patients with acute anterior wall myocardial infarction (AWMI), with a frequency of 17.3% at 72 hours post-admission. This prevalence aligns with prior international reports, such as the 23.6% incidence reported by Phan et al. using serial cardiac magnetic resonance imaging (8), and comparable findings from Middle Eastern cohorts, including a 19.2% prevalence described by Niazi et al. (16). The slight differences in rates may reflect variations in imaging modalities, population characteristics, or the intensity of anticoagulation strategies implemented during acute care.

Importantly, our results confirm that the first 72 hours after infarction represent a critical time frame for thrombus detection, consistent with the established pathophysiology where blood stasis, myocardial akinesia, and endothelial dysfunction are maximal (4,6).

A salient finding of this study is that reduced left ventricular ejection fraction (LVEF) was the strongest independent predictor of LVT formation, with each percentage point decrease associated with a 12% increase in odds of thrombus development (adjusted OR: 1.12, p<0.001). This reinforces existing literature that identifies impaired systolic function as a key contributor to thrombus pathogenesis through mechanisms of impaired flow dynamics and predisposition to stasis (9,10,17). Moreover, anterior wall akinesia emerged as another potent independent risk factor, being present in 95.8% of LVT cases compared to 66.1% without LVT (p<0.001), and conferring nearly an eightfold increase in adjusted odds (adjusted OR: 7.94, p=0.005). These findings substantiate prior reports that highlight extensive regional wall motion abnormalities as critical substrates for thrombus formation, particularly at the left ventricular apex where flow velocities are lowest (5,18).

The associations of diabetes mellitus and smoking with LVT in this cohort provide clinically relevant insights for regional populations. Diabetes was present in 52.1% of patients with LVT compared to 31.7% without (p=0.023), consistent with the prothrombotic state conferred by hyperglycemia, endothelial dysfunction, and increased platelet aggregation in diabetics (19). Smoking, likewise, was significantly more prevalent in the LVT group (64.6% vs. 44.8%, p=0.038), reflecting its acute impact on platelet activation and fibrinogen levels, as well as chronic vascular injury that may amplify thrombotic risk post-infarction (20). These factors are particularly pertinent in the South Asian context, where smoking rates and diabetes prevalence remain high, and highlight the need for aggressive primary prevention and vigilant secondary prophylaxis in these high-risk groups.

The adverse prognostic implications of LVT are underscored by our findings of a substantially increased risk of in-hospital complications among affected patients. Stroke, systemic embolization, and recurrent myocardial infarction occurred exclusively in the LVT cohort, resulting in a composite complication rate of 16.7% and an in-hospital mortality of 6.3%, compared to 0.4% mortality in those without LVT (p=0.014). These data are consistent with prior studies reporting LVT as a marker of poor short-term outcomes and confirm the importance of timely diagnosis to guide anticoagulation therapy (21,22). Notably, while echocardiography remains the primary imaging tool in routine clinical practice due to its accessibility, it is operator-dependent and potentially less sensitive than contrast-enhanced modalities like cardiac magnetic resonance imaging, emphasizing the need for training and standardized protocols to maximize diagnostic yield (23). The identification of a clear constellation of risk factors—severely reduced LVEF, extensive anterior wall akinesia, diabetes mellitus, and smoking—provides clinicians with a practical framework for stratifying thrombotic risk early during hospitalization. This suggests that routine echocardiographic screening at 72 hours should particularly target these subgroups, ensuring timely detection and enabling prompt initiation of anticoagulation to mitigate the risk of embolic events. Our findings also have broader implications for healthcare policy in Pakistan and similar settings, where the burden of ischemic heart disease is rising and resources are constrained; adopting a risk-based screening protocol may optimize resource utilization while improving patient outcomes.

Despite the strengths of a robust sample size and a clearly defined cohort, the study's limitations warrant acknowledgment. The single-center design may limit generalizability to other populations with differing demographic or healthcare delivery profiles. The cross-sectional nature precludes assessment of thrombus resolution and long-term outcomes beyond hospitalization. Additionally, while standardized protocols were employed to reduce interobserver variability, the reliance on echocardiography without contrast enhancement may have led to underestimation of thrombus frequency compared to more sensitive imaging modalities (23). In summary, this study confirms that approximately one in six patients with AWMI develops LVT within 72 hours, with a distinct high-risk profile characterized by severely reduced LVEF, anterior wall akinesia, diabetes mellitus, and smoking. The prognostic significance of LVT is substantial, as evidenced by its strong association with in-hospital complications and mortality. These findings reinforce the clinical utility of routine echocardiographic assessment at 72 hours, particularly in high-risk individuals, and highlight the need for early initiation of anticoagulation therapy to improve outcomes. Future research should focus on prospective multicenter studies incorporating serial imaging and longer-term follow-up to further elucidate the natural history of LVT and optimize management strategies in diverse populations (24).

CONCLUSION

In conclusion, left ventricular thrombus (LVT) remains a significant and clinically relevant complication among patients admitted with acute anterior wall myocardial infarction (AWMI), with this study demonstrating a 17.3% prevalence at 72 hours post-admission. The findings identify severely reduced left ventricular ejection fraction, anterior wall akinesia, diabetes mellitus, and smoking as independent and robust predictors of LVT formation, highlighting a distinct high-risk phenotype. Importantly, all observed in-hospital complications, including stroke, systemic embolization, recurrent myocardial infarction, and most in-hospital deaths, occurred exclusively in patients with LVT, underscoring its prognostic importance. These results emphasize the critical need for routine and timely echocardiographic evaluation within the first 72 hours, particularly in high-risk patients, to facilitate early detection and appropriate anticoagulant therapy aimed at reducing thromboembolic complications and mortality. The study provides essential evidence to guide risk-based screening protocols and inform clinical practice within resource-limited healthcare systems where ischemic heart disease and its complications impose an increasing burden. Future multicenter research with longer follow-up and standardized imaging techniques is warranted to validate these findings and further refine LVT management strategies in diverse patient populations.

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