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ORIGINAL RESEARCH

Prognosis and New Predictors of Early Left-Ventricular Thrombus Following ST-Elevation Myocardial Infarction

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Objective: In the current era of primary percutaneous coronary intervention (PPCI), the prognosis of the left ventricular thrombus (LVT) is not well assessed.

Methods: We performed a retrospective, single-center study of 1305 consecutive ST-segment-elevation myocardial infarction (STEMI) patients treated with PPCI. During a mean period of 27 months of follow-up, the major adverse cardiovascular and cerebrovascular events (MACCE) were recorded.

Results: The incidence of LVT (n = 47) was 3.60%. The independent risk factors of LVT included anterior STEMI, left ventricular (LV) aneurysm, reduced LV ejection fraction (LVEF), dilated LV end-diastolic dimension (LVEDD), and delayed door-to-balloon time (DTBT). During follow-up, LVT was an independent risk factor for MACCE [hazard ratio (HR)=3.46; 95% confidence interval (CI) = 2.23–3.38; P < 0.01]. Patients with LVT were more likely to have the following complications: heart failure (P < 0.001), embolic events (P = 0.034), and all-cause mortality (P = 0.020). Notably, the regression of LVT was not independently associated with those three adverse events (P > 0.05).

Conclusion: In the era of PPCI, the presence of early LVT following STEMI was associated with adverse events. Furthermore, the prognosis of patients with LVT did not improve even if the LVT regressed. LVT was likely a generalized indicator of impaired cardiac performance, rather than the cause. This indicated that prophylactic therapy and identifying individuals with a high risk of developing LVT were of substantial importance.

Keywords: left ventricular thrombus, ST-segment elevation myocardial infarction, prognosis, risk factors

Introduction

The left ventricular thrombus (LVT) is a feared complication for its risks of embolization to peripheral arteries and vital organs after acute myocardial infarction.[1,2] Comparing ST-segment-elevation myocardial infarction (STEMI) patients with LVT to those without LVT, the rate of embolic events in patients with LVT was four-fold higher, and the rate of long-term mortality was two-fold higher.[3] In the current era of primary percutaneous coronary intervention (PPCI), the formation of LVT subsequent to STEMI remains relatively common. The estimated incidence is up to 15% despite advances in reperfusion and timely anti-thrombotic therapy.[4–6] Regardless of STEMI treatments, risk factors for LVT that have been identified are an anterior STEMI, large infarct area, and reduction in systolic function.[7–9] Given the high-risk and increasing burden of LVT, data of which we are aware regarding their prediction to predict its

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occurrence remains a clinical challenge. Furthermore, data on the prognosis of LVT-related events are not well assessed in the era of PPCI.[10,11] Consequently, we conducted a retrospective study to evaluate the risk factors and prognosis of LVT in patients admitted for STEMI and treated with PPCI in the Provincial Chest-Pain Center.

Materials and Methods
Study Population and Design

Consecutive STEMI patients (N = 1363), who underwent PPCI from January 2017 to September 2019 at the China-Japan Union Hospital were screened in our study. The STEMI was defined in accordance with the new World Health Organization (WHO) criteria as revised in 2000: a rise in cardiac troponin levels accompanied by clinical symptoms of myocardial ischemia, and pathological Q waves and/or changes in the electrocardiogram (ECG) (new or presumed-new ST segment elevation at the J-point in least two or more consecutive leads, with ST segment elevation within the cut-off of ≥0.2 mV in leads V1, V2, or V3; and ≥0.1 mV in other leads).[12] In total, 58 patients were excluded by the following criteria: combination with previous stress cardiomyopathy (n = 13), myocarditis (n = 11), dilated cardiomyopathy (DCM; n = 8), and patients who died before the first echocardiogram (n = 26). The remaining 1305 patients enrolled in our study were classified as patients with LVT (LVT group, n = 47) and without LVT (control group, n = 1258) (Figure 1). Written informed consent was obtained from the patients prior to enrolment. The trial was approved by the Institutional Review Board of The China-Japan Union Hospital of Jilin University (Approval No. 2019012804).

A retrospective analysis was performed using the agency's electronic records and database. Hematological parameters were collected from the antecubital vein before the patient received any fibrinolytic therapy. Management of vascular lesions were performed during PCI at the operator's discretion. PCI results were interpreted by two
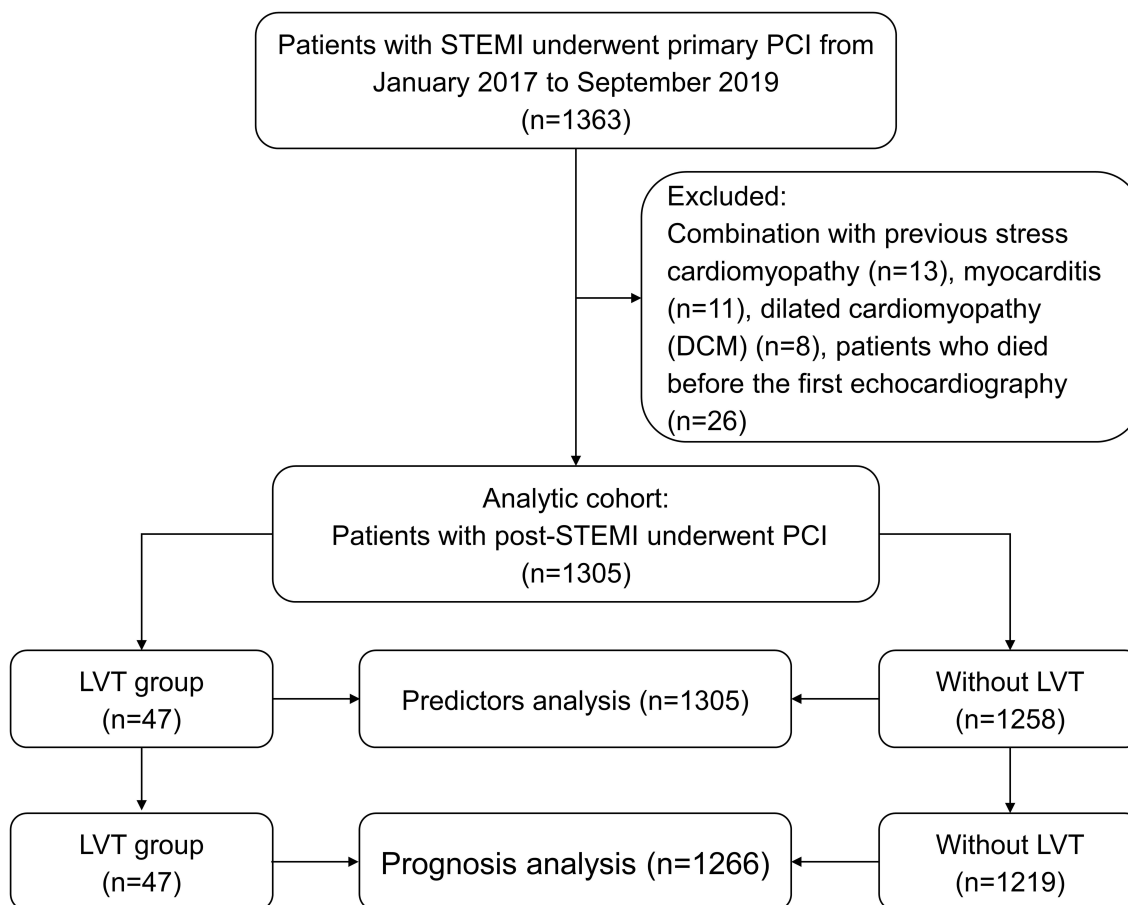

Figure 1 Study flow chart.
Abbreviations: STEMI, ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; LVT, left ventricular thrombus.

experienced senior operators. Two-dimensional echocardiography is generally performed within 72 h of admission by experienced sonographers. If the diagnosis of LVT is suspectable, patients will perform a mandatory second assessment, including two-dimensional echocardiography or cardiac magnetic resonance before discharge. In accordance with the recommendation by the American Echocardiography Association, the Simpson method was used to evaluate the left ventricular ejection fraction (LVEF).[13] LVT was diagnosed as a protruding or independently active mass in the left ventricular cavity throughout systole and diastole that was different from the notochord structure, papillary muscle, trabecula, technical artifact, or tangent view of the left ventricular wall.[14] Treatment of all patients with LVT was left to the discretion of the attending physician based on the patient's clinical status.

Follow-Up

Clinical outcomes were assessed through clinic visits or telephone visits. The major adverse cardiovascular and cerebrovascular event (MACCE) was defined as the first occurrence of any component of the composite outcome of all-cause death, heart failure, non-fatal myocardial reinfarction, revascularization, stroke, or systemic thromboembolism during a mean period of 27 months of follow-up. Stroke was diagnosed based on neurological examinations and MRI scans.[15] The secondary outcome was LVT regression and the incidence of bleeding events, including clinically relevant bleeding events and major bleeding events. The standard of bleeding was defined by the Bleeding Academic Research Consortium.[16]

Statistical Analysis

Continuous variables were presented as "mean ± standard deviation" or "median [quartile]" (ie, "M [Q1, Q3]"), and compared using the Student's t test or rank-sum test. Categorical variables were presented as counts and percentages and compared using the Rao-Scott chi-squared test. Multivariate, logistic regression was performed to identify the independent risk factor of early occurrence of LVT, adjusted for sex, age, and the covariates that were statistically significant in univariate analysis [Diagnosis, left ventricular (LV) aneurysm, fibrinogen, delayed door-to-balloon time (DTBT),LV ejection fraction (LVEF), dilated LV end-diastolic dimension (LVEDD)]. For the primary endpoint, the association of LVT with time-to-MACCE was estimated by Kaplan-Meier curves.

The association between LVT and MACCE was examined using the Cox proportional-hazard, regression model, adjusted for age, hypertension, and diabetes mellitus status. R version 3.6.3 (University of Auckland, Oakland, New Zealand) was used to perform the statistical analyses. Statistical significance was set at P < 0.05.

Results
Clinical Characteristics Between the Two Groups

A total of 1305 STEMI patients were included in this retrospective, single-center study. LVT was diagnosed before hospital discharge in 47 of the 1305 STEMI patients (3.60%). Baseline characteristics are listed in Table 1. There were no statistically significant differences between the LVT and the control groups regarding age, sex, smoking history, or concomitant diseases (such as hypertension and diabetes). Patients who were admitted to the hospital within 12 h after experiencing chest pain were less likely to develop LVT. The average DTBT of the LVT group was statistically significantly longer than that of the control group.

The results from hematological parameters obtained upon admission were similar between the two groups, except for the level of fibrinogen and creatinine, which was significantly higher in the LVT group (P < 0.05). The echocardiography findings demonstrated that more patients in the LVT group had signs of lower LVEF, LV aneurysm, and dilated LVEDD; all were significantly different between the two groups (P < 0.05). Moreover, coronary angiography showed that the anterior descending artery was the chief culprit of all patients in LVT group.

Predictors of LVT

To further identify the risk factors related to LVT, a multivariate, logistic, regression analysis was performed (Table 2). After adjusting for sex and age, the independent risk factors of LVT after STEMI were an anterior STEMI [odds ratio (OR): 35.72, 95% confidence interval (CI): 7.26–647.22, P = 0.001], LV aneurysm (OR: 41.33, 95% CI: 10.25–247.31, P < 0.001), lower LVEF (OR: 0.95, 95% CI: 0.91–0.98, P = 0.005), delayed DTBT per 20 (OR: 1.18, 95% CI: 1.04–1.35, P = 0.011), and dilated LVEDD (OR: 1.08, 95% CI: 1.02–1.15, P = 0.010).

Table 1 Baseline Demographics

	LVT Group (n=47)	Control Group (n= 1258)	P value
Baseline clinical characteristics			
Age (years)	60.0±11.9	61.3±11.8	0.951
Male sex, n (%)	34(72.3)	924(73.4)	0.866
Smoker, n (%)	25(53.2)	701(55.7)	0.732
HTN, n (%)	16(34)	556(44.2)	0.168
DM, n (%)	10(21.3)	235(48.8)	0.889
Chest pain onset duration<12h	22(46.8)	298(23.7)	0.557
Killip≥2	17(36.2)	858(68.2)	<0.001
Blood examinations on admission			
WBC (10^9/L)	11.1(8.2, 13.6)	10.1(8.2,12.3)	0.143
MPV (fl)	9.6(9.1,10.7)	9.5(8.9,10.3)	0.362
Platelets (10^9/L)	241.0(164.0,304.0)	216.0(181.0,256.0)	0.113
Neutrophil (10^9/L)	7.0(4.77,10.09)	7.4(5.61,9.97)	0.342
Lymphocyte (10^9/L)	1.4(1.1,2.3)	1.6(1.1,2.3)	0.773
PLR	140.2(100.0,208.7)	133.2(94.0,190.3)	0.590
NLR	3.7(2.7,6.8)	4.6(2.6,8.1)	0.355
Fibrinogen(mg/dl)	4.1(3.2,5.6)	3.3(2.8,3.9)	<0.001
Creatinine (mg/dL)	90.3(71.2,101.3)	76.3(65.0,86.1)	0.001
Blood urea nitrogen (mg/dL)	6.1(4.8,7.6)	5.9(4.8,6.8)	0.280
Echocardiography			
LVEDD (mm)	51.2(48.1,56.4)	47.8(44.1,53.1)	<0.001
LVEF, %, (means ± SD)	41.4±12.3	57.4±10.8	<0.001
LV aneurysm n (%)	15(31.9)	4(0.3)	<0.001
Culprit Artery			
LM	2(4.3)	17(1.4)	0.103
LAD	47(100)	454(36.1)	<0.001
LCX	1(2.1)	184(14.6)	0.016
RCA	1(2.1)	613(48.7)	0.18
Procedural characteristics			
DTBT (min) (means ± SD)	84(50.0,106.5)	59(42.0,76.7)	0.001
Pre-PCI TIMI flowgrade≤1	47(100)	915(72.7)	<0.001
Post-PCI TIMI flowgrade≤1	0(0)	2(0.2)	0.784
Medical therapy at discharge			
Dual antiplatelet therapy	47(100)	1250(99.4)	0.583
Oral anticoagulant	45(95.7)	23(1.83)	<0.001
ACEI	47(100)	1158(92.1)	0.044
Beta blockers	47(100)	1221(97.1)	0.603
Statins	47(100)	1242(98.7)	0.085

Note: The chest pain onset duration referred to the period of the occurrence of acute myocardial infarction to admission.
Abbreviations: HTN, hypertension; DM, diabetes mellitus; WBC, white blood cell count; MPV, mean platelet volume; NLR neutrophil/lymphocyte ratio; PLR, platelet/lymphocyte ratio; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic dimension; LV aneurysm; left ventricular aneurysm; LM, left main; LAD, left anterior descending coronary artery; LCX, left circumflex; RCA, right coronary artery; DTBT, Door-to-balloon time.

Clinical Outcomes

Among the 1305 patients without LVT, the follow-up data for 39 patients were incomplete, and these patients were excluded from this study. During a mean period of 27 months of follow-up, the primary endpoint of MACCE was significantly higher in the LVT group vs the control group (46.8%

Table 2 Multivariate Logistic Regression Analysis for Assessment of Independent Predictors of LVT

Variable	Unadjusted		Adjusted	
	OR[95% CI]	P value	OR[95% CI]	P value
Age	0.99[0.97–1.02]	0.464	0.98[0.95–1.01]	0.172
Sex(female)	1.06[0.53–1.98]	0.866	2.26[0.91–5.5]	0.073
LVD	83.75[18.23–1486.07]	<0.001	35.72[7.26–647.22]	0.001
LV aneurysm	146.95[50.21–538.34]	<0.001	41.33[10.25–247.31]	<0.001
Fibrinogen	1.56[1.29–1.88]	<0.001	1.22[0.93–1.58]	0.130
LVEF	0.89[0.87–0.92]	<0.001	0.95[0.91–0.98]	0.005
LVEDD	1.16[1.11–1.21]	<0.001	1.08[1.02–1.15]	0.010
DtoB time(per 20)	1.32[1.20–1.44]	<0.001	1.18[1.04–1.35]	0.011
Creatinine (mg/dL)	1.01[0.99–1.01]	0.370	1.00[0.10–1.01]	0.549

Abbreviations: LAD, left anterior descending coronary artery; LV, aneurysm; left ventricular aneurysm; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic dimension; DTBT, Door-to-balloon time.

vs 18%, P < 0.001) (Table 3 and Figure 2). Heart failure (29.8% vs 9.1% P < 0.001), embolic events (6.4% vs 1.9% P = 0.034), and all-cause mortality (12.8% vs 5% P = 0.020) were significantly increased in the LVT group. There were no significant differences in other components of MACCE in the LVT group vs the control group (P > 0.05 for all). There was no significant difference in the incidence of bleeding events between the two groups (P = 0.195). After adjusting for the relevant factors (age, hypertension, and diabetes) in the multivariate Cox proportional hazard model (Table 4), LVT remained a significant, independent predictor of MACCE (HR = 3.46; 95% CI 2.23–3.38; P < 0.001).

Adverse Outcomes According to Occurrence of LVT Regression

As shown in Table 5, total LVT regression occurred in 33 patients (70.21%) within a median time of 14 months

(interquartile range: 6 to 25 months). Heart failure occurred in 24.24% and 42.86% of LVT-regression and LVT-persistent patients, respectively. All-cause mortality occurred in 9.10% and 21.43% of LVT-regression and LVT-persistent patients, respectively. Reduced stroke was observed in patients with LVT regression (6.10% vs 14.28%). However, no statistically significant differences were observed between patients with LVT regression and patients with persistent LVT for these adverse events (heart failure, embolic events, and all-cause mortality; P > 0.05 for all parameters).

Discussion

This study provided new data and insights into the contemporary incidence, prediction, and prognosis of LVT in patients with STEMI in PPCI practice. Based on the results of this study, we have made three essential findings: (1) Studies have consistently demonstrated that the independent risk factors of LVT were anterior STEMI, lower LVEF, LV aneurysm, and

Table 3 Univariate Logistic Regression for MACCE

	All (n=1266)	LVT Group (n=47)	Control Group (n=1219)	Chi-Square	P value
Primary Outcome					
MACCE	251(19.8)	23(48.9)	228(18.7)	26.022	<0.001
Individual 1-Year Outcomes					
Heart failure	129(10.2)	14(29.8)	115(9.4)	20.486	<0.001
Reinfarction	16(1.3)	1(2.1)	15(1.2)	0.292	0.589
Revascularization	28(2.2)	0	28(2.3)	1.104	0.293
Stroke	30(2.4)	4(6.4)	26(2.1)	7.957	0.005
All-cause mortality	68(5.4)	6(12.8)	62(5.1)	5.251	0.022
Bleeding event	40(3.2)	3(6.4)	37(3.0)	1.658	0.198

Abbreviation: MACCE, major adverse cardiovascular and cerebrovascular event.
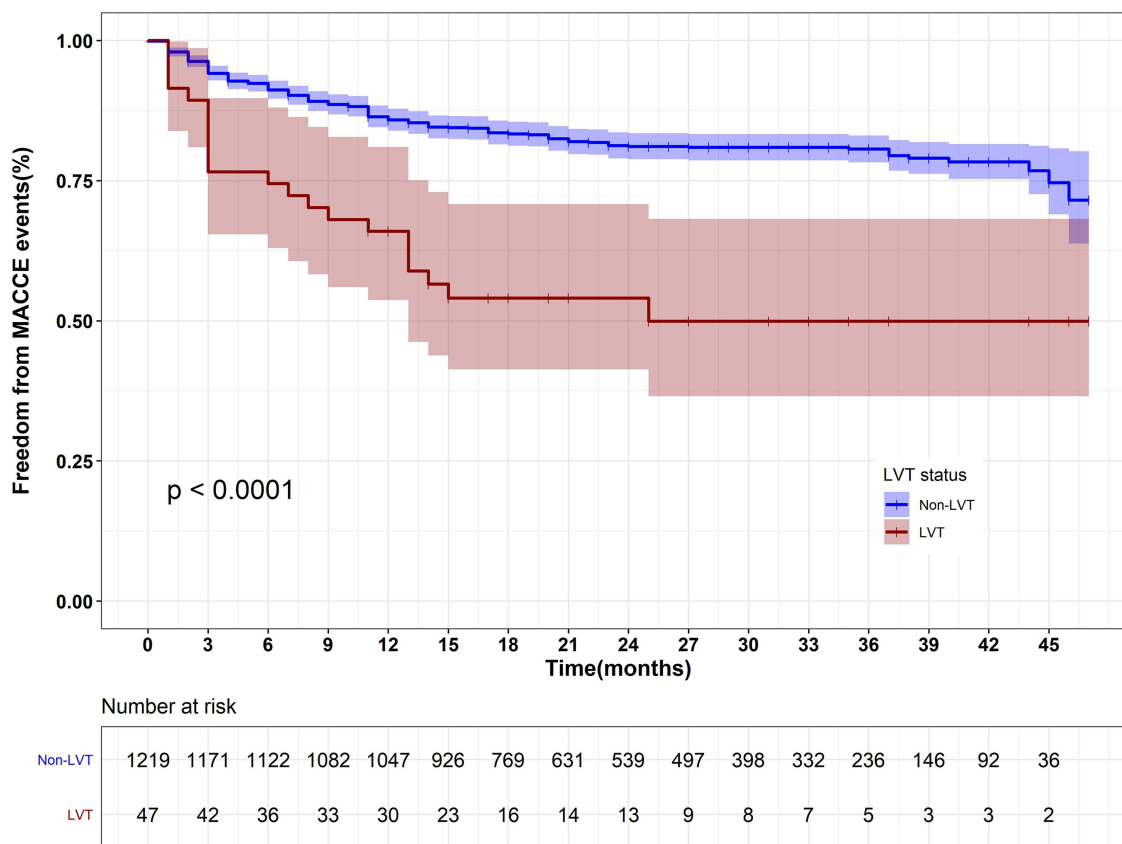
Figure 2 Cumulative incidence of primary outcomes (all-cause death, heart failure, non-fatal myocardial reinfarction, revascularization, stroke, or systemic thromboembolism), stratified by the presence of LVT (red) or no LVT (blue) in patients admitted for STEMI and treated by PPCI during a mean period of 27 months of follow-up.

dilated LVEDD. Furthermore, our study showed that a shorter DTBT significantly reduced the incidence of LVT. (2) The presence of LVT was independently associated with an increased incidence of MACCE over a mean period of 27 months of follow-up. Notably, the regression of LVT was not independently associated with adverse events.

Incidence and Predictors of LVT in the Current Era

The incidence of LVT following STEMI was 3.60% in our study, which was consistent with previous studies.[6,17,18]

There is growing evidence that the principal risk factors closely associated with LVT after STEMI are both the presence of anterior-territory STEMI and LV dysfunction.[18–20] In our study, we also found that the location of the vessel infarct in the left anterior descending (LAD) was the single most important independent predictor of LVT after STEMI. Patients with anterior STEMI had a 10-fold increased risk of LVT formation. Furthermore, LVEF was the strongest independent predictor of LVT formation, which was significantly lower in patients with LVT. Dilated LVEDD and an aneurysm appeared to be

Table 4 Multivariate Analysis of Predictors of MACCE in Patients with LVT During a Mean Period of 27 Months

	HR	L95	U95	P value
LVT	3.46	2.23	5.38	3.42E-08
Age	1.03	1.02	1.04	1.83E-06
DM	0.86	0.62	1.19	0.367389
HTN	1.39	1.08	1.79	0.009856

Abbreviations: LVT, left ventricular thrombus; HTN, hypertension; DM, diabetes mellitus.

Table 5 The Relationship Between LVT Resolution and the Adverse Outcomes

	LVT Resolution		P value
	Yes (n=33)	No (n=14)	
All-cause mortality	4(12.12)	2(14.28)	0.767
Stroke	2(6.06)	1(7.14)	0.840
Heart failure	8(24.24)	6(42.86)	0.146

more common in patients with LVT. LV remodeling and stasis of blood might play a positive role in LVT formation. This finding has frequently been described in previous studies.[4,21,22] In addition, though many studies have demonstrated that shorter DTBTs are associated with a promising prognosis in STEMI patients,[23,24] the value of DTBT in LVT formation following STEMI has been controversial.[25–27] In our study, patients with delayed DTBT had a significantly higher probability of demonstrating LVT, emphasizing the importance of shorter DTBT. It is a well-known information that kidney injury predisposes to thrombus formation. Notably, previous studies confirmed that acute kidney injury (AKI) had a predictive value both in in-hospital outcomes and long-term outcomes of patients with STEMI, suggesting that creatinine and blood urea nitrogen might be helpful in patients at higher clinical risk. We also found that the level of creatinine in the LVT group was significantly higher. Unfortunately, the multivariate analysis is not statistically significant, which needs to be confirmed by prospective studies. In addition, a total of 19 STEMI patients were treated with IABP in our study.[28,29] Consistent with prior study, the prognosis remained poor despite IABP support in patients with STEMI. We found IABP support therapy had no relationship with the formation of LVT.[30]

Clinical Outcome of LVT

In our study, we found that LVT was an independent risk factor for MACCE over a mean period of 27 months of follow-up, after adjusting for principal confounders. Increased risk of heart failure, embolic events, and all-cause mortality were variables that also reached statistically significant differences between the two groups. Heart failure is the principal adverse event in patients with LVT. It is well established that heart failure is a strong predictor of LVT in STEMI patients.[31,32] Inversely, LVT might be an important trigger of heart failure. In the pre-thrombolytic period, LVT was associated with a 5.5-fold increase in embolic events.[33] Chen et al concluded that the relative risk of embolic complications in patients with LVT after acute myocardial infarction (AMI) increased nearly 4-fold, regardless of follow-up time and detection tools.[3] Consistent with prior studies, a 3.37-fold higher risk of an embolic event was observed in patients with LVT in our study. A retrospective study of 128 LVT patients reported that all-cause mortality occurred in 13% of the patients within a 1-year follow-up.[22] Another study followed-up the patients who underwent PCI for STEMI

for 1-year, and they detected a mortality rate of 10%.[10] Likewise, in our study, we reported a mortality rate of 12.8% in patients with LVT, and found that mortality was statistically significantly different between the LVT group and non-LVT group. These findings highlight the worst prognosis associated with LVT.

During the follow-up of the patients with LVT, we did not note any significant differences in the rates of adverse events (heart failure, embolic events, or all-cause mortality) between patients with and without LVT regression. Several studies before the PCI era tried to determine the association between the presence of LVT and embolic events.[33,34] Surprisingly, these studies did not demonstrate a convincing relationship. Lemaitre et al, in a study of 105 heart failure (HF) patients with LVT, reported a lower risk of long-term consequences (death <3%) and the potential for the patients to experience reverse remodeling. Notably, however, the patients with LVT had a higher incidence of embolic events within 30 d.[35] Consistent with our findings, Lemaitre et al demonstrated that the presence of LVT did not seem to be associated with mortality or preclude reverse remodeling. In addition, Lattuca et al recently performed a retrospective study of 159 LVT patients who were screened from ordinary patients.[36] Similar to our results, they found that the presence of LVT presented a high risk of clinical complications and LVT regression was not associated with heart failure or embolic events. The only difference between our results and theirs was that they reported that LVT regression was associated with reduced mortality. Variations in study subjects and treatment factors (DTBT, antithrombotic combinations, and treatment duration) might have contributed to the differing results. Consequently, our results emphasized that patients with LVT after STEMI were likely a generalized indicator of impaired cardiac performance, rather than the cause.

Some limitations of the current study should be considered. First, we based the diagnosis of LVT mainly on echocardiography, which might have less sensitivity and specificity than other imaging methods, such as CMR and left ventriculography. However, CMR and left ventriculography are not suitable as the basic screening tests in STEMI patients. Furthermore, we used CMR and left ventriculography to confirm the diagnosis of LVT obtained by routine echocardiography in cases when diagnostic confidence was low. Second, a different therapy strategy might affect the clinical outcome. However, several recent studies have shown that there is no statistical difference in rates of MACCE or LVT regression

between novel oral anticoagulants (DOACs) and vitamin K antagonist therapeutic strategies.[36–38]

Conclusion

In conclusion, our study indicated that LVT was not uncommon in patients with STEMI treated with PPCI. Anterior STEMI, LV dysfunction, LV aneurysm, delayed DTBT, and increased LVEDD were independent risk factors of LVT. Furthermore, LVT was an independent predictor of MACCE over a mean period of 27 months of follow-up. Importantly, we showed that LVT was likely a generalized indicator of impaired cardiac performance; patients with LVT had a particularly high risk of subsequent cardiovascular events after STEMI. This further indicated that prophylactic therapy and identifying individuals at high risk are of substantial importance.

Abbreviations

PPCI, primary percutaneous coronary intervention; LVT, left ventricular thrombus; STEMI, ST-segment elevation myocardial infarction; LVEF, left ventricular ejection fraction; MACCE, major adverse cardiovascular and cerebrovascular event; LVEDD, left ventricular end-diastolic dimension; DTBT, delayed door-to-balloon time; CMR, cardiac magnetic resonance.

Data Sharing Statement

The datasets used during the current study are available from the corresponding author upon request (Wenqi Zhang).

Ethical Approval and Consent to Participate

The study was approved by the Institutional Review Board of The China-Japan Union Hospital of Jilin University (Approval No. 2019012804). All participants provided informed consent to participate in this research and it was conducted in accordance with the Declaration of Helsinki.

Consent for Publication

The authors confirm that all contents of this study can be published.

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Disclosure

The authors have no conflicts of interest to declare.

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