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Predictors of left ventricular thrombus formation and delayed resolution in post-acute myocardial infarction and severe left ventricular dysfunction

Abstract

Background: The predictors of left ventricular thrombus (LVT) formation and resolution, post-acute myocardial infarction (MI), and left ventricular (LV) dysfunction significantly impact management strategies and need updating to reflect contemporary practice.

Methods: Transthoracic echocardiography was used to screen and assess post-acute MI patients with LV ejection fraction (LVEF) <35% or <40% with apical akinesis or dyskinesis.

Results: We enrolled 979 patients. Of them, 67 (6.84%) patients had an LVT at the baseline. Additionally, 22 and 7 patients developed new LVT at 1 and 3 months. The predictors of LVT formation were the presence of LV aneurysm (HR: 1.45, 95% CI: 1.11-2.07, P=0.024), apical wall motion score index (WMSI) (HR: 1.36, 95% CI: 1.07-2.82, P=0.036), late presentation after MI (HR: 1.32, 95% CI: 1.16-3.16, P=0.042), older age (HR: 1.24, 95% CI: 1.08-3.36, P=0.043), lower baseline LVEF (HR: 1.23, 95% CI: 1.06-2.75, P=0.046) and higher level of low-density lipoprotein-cholesterol (LDL-C) (HR: 1.18, 95% CI: 1.02-2.54, P=0.049). The LVT was resolved in 40 (59.7%) and 32 patients (65.3%) at 1 and 3 months, respectively. The predictors of LVT persistence beyond 3 months were LV aneurysm (HR: 1.55, 95% CI: 1.03-1.87, P=0.024), LVT size at baseline (HR: 1.43, 95% CI: 1.32-2.74, P=0.031), apical WMSI (HR: 1.43, 95% CI: 1.32-2.74, P=0.031), lower LVEF (HR: 1.29, 95% CI: 1.02-2.54, P=0.043) and late presentation after MI (HR: 1.18, 95% CI: 1.07-2.16, P=0.047).

Conclusions: The global and apical LV systolic dysfunction, older age, late presentation, and high LDL-C predict LVT formation post-MI. These factors and LVT size predict LVT persistence beyond 3 months. These findings should guide anticoagulation therapy in this high-risk population.

Keywords: ventricular, thrombus, myocardial infarction, heart failure

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Abbreviations

LVT, left ventricular thrombus; LV, left ventricle; TTE, Transthoracic echocardiography; WMSI, wall motion score index; HR, hazard ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; LDL-C, low-density lipoprotein cholesterol; MI, Myocardial infarction; OAC, Oral anticoagulant; PCI, Percutaneous coronary intervention; INR, international normalized ratio; SD, Standard deviation; GDMT, Guidelines-directed medical therapy; DAPT, Dual antiplatelet therapy

Introduction

The incidence of left ventricular (LV) thrombus (LVT) in patients with acute ST-elevation myocardial infarction (MI) has decreased significantly in the contemporary era to range from 6.2% to 10.5%,[1-4] compared to multiples of these in the older eras.[5] Some of the predictors of LVT formation and resolution are reported in the literature but are either non-contemporary or not informative enough to guide decision-making.[4] The chance of LVT formation increases in patients with severe LV dysfunction, and anterior MI is a commonly reported predictor of LVT formation,[1-3] while others found definite LVT following anterior MI with LV dysfunction challenging to predict.[4] This is reflected in the infirm recommendations for prophylactic use of anticoagulation in patients with severe LV dysfunction post-acute MI.[5,6] Additionally, the rate of LVT resolution is controversial,[3,4,7] and updates of the predictors of LVT formation and resolution under modern management are needed for the medical community.

Materials and methods

Study design and population

We reviewed the database from February 2017 till April 2022. We also included prospectively enrolled patients from two dedicated left ventricular thrombus studies. All enrolled patients were above the age of 18 years and had a history of either acute (within a week) or recent (within a month) anterior wall MI, with LV ejection fraction (LVEF) <35% or <40% with apical wall motion score index (WMSI) ≥ 2 or apical aneurysm on conventional transthoracic echocardiography (TTE). All patients were naïve to oral anticoagulants (OAC) at the baseline. The study excluded the following patients: those with other indications for OAC, those with right ventricular or atrial thrombus, and those with technically challenging echocardiography windows that hampered the assessment of actual LVT existence. Consent was obtained from patients included in two prospective substudies for participation and publication. These studies were approved and overseen by the institutional research and ethics committee (IRB-2018-08) and (IRB-2018-11), date of approval: August 18, 2018) of Saud AlBabtain Cardiac Centre, Dammam, Saudi Arabia. The

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anonymized data obtained (retrospectively) earlier or later from the database were waived from consent.

The study has been performed following the ethical standards of the 1964 Declaration of Helsinki and its later amendments.

The primary study endpoint was the clinical and echocardiographic predictors of LVT formation at baseline and 1-month echocardiography follow-up, and LVT resolution after 1 or 3 months. Two independent echocardiography experts analyzed each echocardiographic study. In cases of discordance, a third expert independently evaluated the echocardiographic study, and a consensus was reached to confirm the findings.

Baseline and data collection

The database and medical files data include the patients' demographics, cardiovascular risk factors, medical history, underlying diseases, medication use, and long-term use of antithrombotic therapy before and after LVT diagnosis. They also include routine laboratory investigations, cardiac catheterisation data, the timing of clinical events, and the investigations performed.

Echocardiographic assessment and thrombus evaluation

The LVT was an echo-dense mass clear-cut from the endocardium with well-defined edges adjoining a hypokinetic, akinetic, or aneurysmal myocardial segment.[8,9] The LVT had to be visible in two or more views during the cardiac cycle. The LVT diameter (mm), area (cm^2) and characteristics were assessed during each TTE without echo-contrast and were followed to study LVT evolution over time. Thrombus resolution was the complete disappearance in all available echocardiography views. The echocardiographic assessment included LVEF, LV volume, identification of apical aneurysm formation and apical WMSI. Echocardiography data were obtained from studies performed at baseline, 1- and 3-months. When LVT was confirmed, the OAC was administered and continued until LVT resolution was confirmed for at least 3 months. The treatment was extended for 6 months or more when LVT resolution could not be proven. Experienced sonographers performed the TTE studies, and all examinations involved a Vivid E9 (1.5–4.6 MHz gauge; second harmonic imaging, Vingmed GE) and Philips IE33 (5-1 MHz gauge, S5-1 sector array probe, Philips) echocardiography machines. Views were acquired in the standard imaging planes following the American Society of Echocardiography recommendations.[9] If an earlier study showed suspicion or a higher risk of developing LVT, another TTE is repeated before discharge.

The full study and related report were archived and reviewed from the electronic database. Images were interpreted by consensus of two independent, experienced readers (Level III trained in echocardiography). A pre-designated third reader was consulted in cases of interpretive discordance. Thrombus morphology was classified as protuberant (protrusion into LV cavity) or mural (borders were contiguous and parallel with adjoining endocardial contours).[7,8] The Simpson method measured the left ventricular volumes and ejection fraction, and the apical WMSI was calculated.[9,10] We defined 1-month TTE follow-up as 1 month ±1 week and 3-month follow-up as 3 months ± 2 weeks.

Intervention, medical therapy, and follow-up

All patients were treated according to the standards of acute anterior MI management.[5,6,11] including reperfusion with primary percutaneous coronary intervention (PCI) or lytic therapy followed by PCI. Parenteral anticoagulation with heparin continued until culprit vessel revascularization, or TTE confirmed no LVT, whatever occurred later, and all patients had dual antiplatelet therapy. Routine clinical assessment was performed at 1 month and 3 months. The local institutional practice for the OAC was either apixaban (5 mg twice daily) or dose-adjusted warfarin to achieve a target international normalized ratio (INR) of 2 to 3. Heparin bridging for patients receiving warfarin and the type and duration of triple therapy were decided at the treating physician's discretion. A clinical pharmacist followed and managed the patients on warfarin in a dedicated clinic. Oral anticoagulation was routinely given for three months, classically with a single antiplatelet agent after the first 7 to 30 days, as per physician discretion and guidelines[6,11-13] and discontinued when thrombus resolution had been confirmed at the 3-month visit. When LVT persisted beyond 3 months, anticoagulation and follow-up were extended.

Statistical analysis

The study enrolled all consecutive patients with the eligibility criteria and the statistical analysis included all LVT patients to detect LVT formation predictors. The analysis of LVT resolution predictors only used those with LVT who completed the 3-month echocardiography follow-up (Figure 1). Data are reported as mean ± SD for continuous variables and n (%) for categorical variables. Unpaired Student t-test and Fisher exact test were used to compare differences between continuous and categorical variables, respectively. All the clinical and echocardiography variables were entered in a regression analysis, as a univariate and multivariate, to assess the primary study endpoints. We used the Cox proportional hazards model with calculations of the hazard ratios and 95% confidence intervals. $P < .05$ was considered statistically significant.

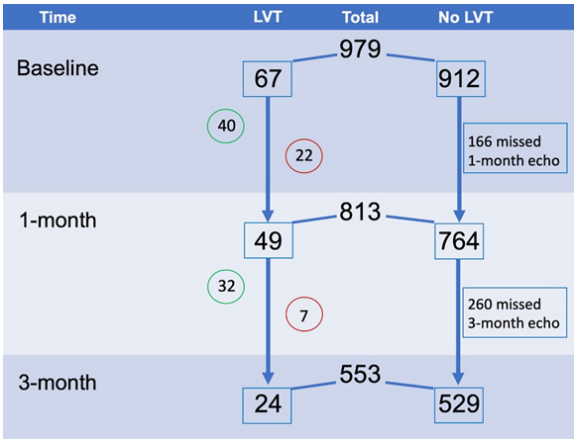

Figure 1 Study consort.

Results

The retrospective echocardiography analysis for all consecutive patients derived 720 eligible for the study. Concurrently, we enrolled 203 patients and 56 patients from two parallel prospective studies of exact eligibility criteria. The total number of patients at the baseline was 979 (Figure 1); 67 (6.84%) patients had a confirmed LVT, and medical treatment using OAC and antiplatelets was prescribed. At 1 month, echocardiography was available for 813 (83%) patients; 40 patients had the LVT disappeared, and 22 patients had a new LVT. At 3 months, echocardiography was available for 553 (56.5%) of the patients; 32 patients had the LVT disappear, and 7 new patients had an LVT. Of the 67 specific patients with LVT at the baseline, the LVT disappeared in 40 (59.7%) patients at 1 month, and in 57

(85.1%) patients, the LVT vanished by the third month. Regarding the echocardiography missing rates, 166 (16.9%) patients missed the 1-month, and another 260 (26.6%) missed the 3-month echocardiography follow-up.

Baseline demographic, laboratory, angiographic and echocardiography data

Most of the study population (93.3%) and all those with LVT were males (Table 1). The patients in the LVT group were significantly older and more non-citizens (61.2%). The history of having common cardiovascular risk factors didn't differ between LVT and no LVT groups. However, patients with LVT had more history of old MI (25.4% versus 11.2%, p=0.033) and more prior PCI (25.4% versus 12.7, p=0.047). Also, they had a significantly later presentation (65.7% versus 18.5%, p=0.006) and a longer time from MI to PCI. The laboratory findings showed significantly higher total cholesterol levels and low-density lipoprotein cholesterol (LDL-C) in the LVT group. The two groups didn't vary significantly in the number of diseased or PCI-treated coronary arteries. However, the group with LVT had less primary PCI (58.2 % vs 76.1%, p=0.039) and worse coronary flow (TIMI-3: 83.6% vs 96.2%, p=0.035). The group with LVT had a longer hospital stay (4.5±3.5 vs 2.3±2.6 days, p<0.001), and most of the patients in both groups (84.6%) had the baseline echocardiography within 48 hours from admission.

Table 1 Comparison of the baseline demographics of patients with LVT versus no LVT

	Thrombus (n=67)	No thrombus (n=912)	p-value
Male, *n* (%)	67 (100)	846 (92.8)	0.372
Age (mean±SD)	57±3.6	53±7.9	0.027
Nationality:			
- Citizen, *n* (%)	26 (38.8)	465 (51.0)	0.163
- Non-citizen, *n* (%)	41 (61.2)	447 (49.0)	
BMI (kg/m2)	26.86±8.2	27.28±6.7	0.352
Smoking, *n* (%)			
- Never	28 (41.8)	349 (38.3)	0.273
- Current or former	39 (58.2)	563 (61.7)	
HTN, *n* (%)	32 (47.8)	446 (49.0)	0.342
DM, *n* (%)	44 (65.7)	558 (61.2)	0.363
Dyslipidaemia, *n* (%)	23 (34.3)	238 (26.1)	0.297
History of old MI	17 (25.4)	102 (11.2)	0.033
Previous PCI, *n* (%)	17 (25.4)	116 (12.7)	0.047
MI to PCI			
- Within 24 hrs, *n* (%)	39 (58.2)	695 (76.2)	0.045
- Over 24 hrs, *n* (%)	28 (41.8)	215 (23.6)	
Late presentation#	44 (65.7)	169 (18.5)	0.006
Anterior MI, *n* (%)	67 (100)	894 (98.0)	0.617
Hemoglobin level (gm/dL)	13.7±2.2	13.5±3.2	0.538
Serum creatinine	1.28±0.57	1.17±0.63	0.344
Total cholesterol	223±37.2	183±63.4	0.027
LDL	168.6±25.3	125.3±39.5	<0.001
No. of diseased vessels			
- One	31 (46.3)	372 (40.8)	0.549
- Two	11 (16.4)	260 (28.5)	
- Multivessel	25 (37.3)	280 (30.7)	
No. of treated vessels			
- One	45 (67.2)	653 (71.6)	0.784
- Two	20 (29.8)	239 (26.2)	
- Three	2 (3.0)	20 (2.2)	

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Table 1 Continued..

	Thrombus (n=67)	No thrombus (n=912)	p-value
Primary PCI*	39 (58.2)	694 (76.1)	0.039
Post PCI TIMI III flow	56 (83.6)	877 (96.2)	0.035
Hospital stay (mean±SD)	4.5±3.5	2.3±2.6	<0.001
Duration to echo$			
- Within 48 hours	55 (82.1)	773(84.8)	0.738
- After 48 hours	12 (17.9)	139 (15.2)	

Late presentation is beyond 12 hours of the typical chest pain onset. @ Index admission PCI: either PCI during the same admission as that of LVT diagnosis or primary PCI. *PCI in the first 24 hours from myocardial infarction onset unpreceded by using a lytic. $ from the diagnosis of acute myocardial infarction tell doing the echocardiography study. LVT, left ventricular thrombus; HTN, hypertension; DM, diabetes milletus; MI, myocardial infarction; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

The group with LVT had a nonsignificant trend of larger LV volumes (Table. 2), significantly more existence of wider LV apex or LV aneurysm (49.3% vs 28.6%, p=0.026), lesser LVEF at baseline (26.5±3.8 vs 29.8±4.6, p=0.038) and 3-month follow up (28.4±3.2 vs 35.2±5.8, p=0.004). The apical WMSI was significantly higher in the LVT group with worsening from baseline to follow-up. However, the group without LVT had improved apical WMSI at follow-up compared to the baseline.

Table 2 Baseline echocardiographic data of LVT versus no LVT

	Thrombus (n=67)	No thrombus (n=912)	p-value
LVEDV (mL) (Mean±SD)	173.6 ± 32.2	169.6 ± 29.7	0.437
LVESV (mL)	126.6±34.3	123.5 ± 28.6	0.282
Aneurysm/wide apex	33 (49.3)	261 (28.6)	0.026
EF at baseline (Mean±SD)	26.5±3.8	29.8±4.6	0.038
EF after 3 months (Mean±SD)	28.4±3.2	35.2±5.8	0.004
Apical WMSI at baseline	2.9±0.35	2.6±0.18	0.048
Apical WMSI after 1 month	2.92±0.41	2.41±0.56	0.001
Apical WMSI after 3 months	2.93±0.51	2.4±0.62	0.005

LVT, left ventricular thrombus; LV, left ventricle; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; EF, ejection fraction; WMSI, wall motion score index.

Medical treatment and anticoagulation

More patients in the LVT group were treated with fibrinolytic (31.3% versus 22.4%, p=0.012) (Table 3). During PCI, the LVT group used tirofiban more frequently (37.3% vs. 32.1%, p=0.039). The use of different medications and oral antiplatelets (including ticagrelor before LVT diagnosis) was similar between the two groups. However, after LVT diagnosis, clopidogrel replaced ticagrelor once OAC was planned, and the aspirin was discontinued except for 2-4 weeks in 28 (41.8%) of the patients with LVT. Warfarin was added in 39 (58.2%) patients with LVT and apixaban in 28 (41.8%). There was no difference in other GDMT use between the two groups except more significant use of loop diuretics in the patients with LVT (73.1 vs. 57.2%, p=0.016) at follow-up. In 8 (0.9%) patients without LVT, apixaban was used as they had new atrial fibrillation.

Table 3 Medication use in the study groups

Medication used in the study groups during the index hospitalization			
Variable	**Thrombus (n=67)**	**No thrombus (n=912)**	**p-Value**
Thrombolytic	21 (31.3)	204 (22.4)	0.012
Tirofiban	25 (37.3)	293 (32.1)	0.039
DAPT use	67 (100)	902 (98.9)	0.801
Clopidogrel	17 (25.4)	231 (25.3)	0.250
Ticagrelor*	50 (74.6)	681 (74.7)	0.701
Beta-blocker	62 (92.5)	874 (95.8)	0.342
ACEi/ARB	67 (100)	875 (95.9)	0.349
Spironolactone	38 (56.7)	537 (58.9)	0.108
Loop diuretics	44 (64.7)	522 (57.2)	0.182
Statins	67 (100)	907 (99.5)	0.362

Table 3 Continued..

Medications used in the study groups after LVT detection			
Variable	**Thrombus (n=67)**	**No thrombus (n=912)**	**p-Value**
DAPT use	28 (41.8)	902 (98.9)	0.003
Clopidogrel	12 (100)	235 (25.8)	0.002
Ticagrelor	0	677 (74.2)	0.001
OAC			
- none	0	188 (98.9)	<0.0001
- Warfarin	39 (58.2)	0	
- Apixaban	28 (41.8)	8 (0.9)	
Beta-blocker	62 (92.5)	874 (95.8)	0.365
ACEi/ARB	67 (100)	877 (96.2)	0.349
Spironolactone	41 (61.2)	537 (58.9)	0.128
Loop diuretics	49 (73.1)	522 (57.2)	0.016
Statins	67 (100)	904 (99.1)	0.429

*Ticagrelor was used in these patients before the left ventricular thrombus diagnosis. *Medication used in the study groups during the index hospitalization* at baseline before LVT was diagnosed. DAPT, dual antiplatelet therapy; OAC, oral anticoagulants; ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

Predictors of LVT development

At 1 month follow-up, 22 new LVT cases were detected. Additionally, 7 more cases were detected at 3-month follow-up (Figure 1). The predictors of LVT development in the study population using multivariate regression analysis (Table 4) were the presence of LV aneurysm (HR: 1.45, 95% CI: 1.11 to 2.07, P=0.024), baseline apical WMSI (HR: 1.36, 95% CI: 1.07 to 2.82, P=0.036), late presentation to the hospital after acute MI (HR: 1.32, 95% CI: 1.16 to 3.16, P=0.042), advanced patient age (HR: 1.24, 95% CI: 1.08 to 3.36, P=0.043), lower baseline LVEF (HR: 1.23, 95% CI: 1.06 to 2.75, P=0.046) and higher level of LDL-C in the patients (HR: 1.18, 95% CI: 1.02 to 2.54, P=0.049).

Table 4 Multivariate analyses of predictors of LVT formation

	HR	**95% CI**	**p-value**
LV aneurysm	1.45	1.11 – 2.07	0.024
Baseline apical WMSI	1.36	1.07 – 2.82	0.036
Late presentation	1.32	1.16 – 3.16	0.042
Age	1.24	1.08 – 3.36	0.043
Lower LVEF	1.23	1.06 – 2.75	0.046
LDL-C	1.18	1.02 – 2.54	0.049

HR, Hazard ratio; CI, confidence interval; LVT, left ventricular thrombus; LV, left ventricle; LDL-C; low-density lipoprotein-cholesterol, LVEF; left ventricular ejection fraction.

Predictors of LVT persistence beyond 3 months

Echocardiography data were available for 813 patients at 1 month, where 40 (59.7%) patients got the LVT resolved and 22 got new LVT (Figure 1). At a 3-month follow-up, 32 (65.3%) patients with LVT at 1 month got the LVT resolved. At 3-month follow-up, LVT was persistent in 10 (15%) out of the 67 patients with LVT at baseline, while 57 (85%) patients had complete resolution of the LVT (Table 5). The group with persistent LVT at 3 months had numerically larger LV volumes, significantly lower LVEF, more existence of LV aneurysm and higher value for apical WMSI. The LVT morphology of being fresh-looking or protruding didn't significantly affect LVT resolution.

The multivariate regression analysis of the predictors of LVT persistence, as detected by TTE at 3 months follow-up (Table 6), were the presence of LV aneurysm (HR: 1.55, 95% CI: 1.03 to 1.87, P=0.024), LVT size at baseline (HR: 1.43, 95% CI: 1.32 to 2.74, P=0.031), baseline apical WMSI (HR: 1.43, 95% CI: 1.32 to 2.74, P=0.031), lower baseline LVEF (HR: 1.29, 95% CI: 1.02 to 2.54, P=0.043) and late presentation to the hospital after acute MI (HR: 1.18, 95% CI: 1.07 to 2.16, P=0.047). Importantly, the type of OAC used for LVT didn't predict LVT resolution.

Table 5 Echocardiographic characteristics of persistence versus resolution LVT at 3 months

Variable	Persistent LVT (n =10)	Resoluted LVT (n =57)	p-value
LVESV (mL)	171.5±29.5	166.2 ± 28.6	0.075
LVEDV (mL)	128.8±32.1	124.8±31.2	0.054
LVEF (%)	24.4 ± 6.1	27.3 ± 9.2	0.046
Apical WMSI	2.92 ±0.25	2.4 ± 0.39	0.042
Apical aneurysm, *n* (%)	10 (100)	42 (73.7)	0.024
LVT length (mm)	28.6±9.6	25.6±11.5	0.057
LVT width (mm)	15.4±7.1	14.9±6.4	0.055
LVT area (cm^2)	2.9±1.3	2.6±1.2	0.033
Mobile LVT, *n* (%)	5 (50.0)	27 (47.4)	0.702
Fresh and /or protruding LVT, *n* (%)	6 (60.0)	34 (59.6)	0.385

LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; WMSI, wall motion score index; LVT, left ventricular thrombus.

Table 6 Multivariate analyses of predictors of LVT persistence beyond three months

	HR	95% CI	p-value
LV aneurysm	1.55	1.03 – 1.87	0.024
Bigger baseline LVT area	1.43	1.32 – 2.74	0.031
Apical WMSI	1.42	1.07 – 2.82	0.036
Lower LVEF	1.29	1.02 – 2.54	0.043
Late presentation	1.18	1.07 – 2.16	0.047

HR, Hazard ratio; CI, confidence interval; LVT, left ventricular thrombus; LV, left ventricle; LVEF, left ventricular ejection fraction.

Discussion

Low incidence of LVT in the contemporary era

Our study confirms a reduced incidence of LVT in the high-risk post-acute MI population with severe LV dysfunction, reflecting contemporary practice's impact on better outcomes.[1-4] However, the lower sensitivity of TTE for LVT detection can't be denied,[8] even though TTE is the standard for screening and follow-up,[5,6,12,13] and most of the potential echocardiography-missed LV thrombi are typical, smaller, and mural with a lower risk of embolization.[8,10,21] Our study also reported lower incidences of new LVT at and beyond 1 month. With the modern management of MI, noncomplicated cases are discharged within 72 hours.[6] Most of our study baseline TTE was done within 48 hours, which could be too early to detect LVT, but this did not lead to a higher LVT rate in the following TTE study. Studies reported that the peak incidence of LVT formation in this high-risk group is 1 to 2 weeks,[4,8] and the clinical impact of scheduling high-risk patients with multiple LVT predictors to have TTE at this potential time deserves evaluation. These lower LVT incidences, especially the new ones at follow-up, can be explained by the modern management standards of acute MI, including coronary revascularization, the use of potent dual antiplatelet therapy (DAPT),[14,15] our high rates of GDMT, and recovery from myocardial stunning[16,17] manifested in our study as a regression of LV dilation and improvement of LVEF and apical WMSI.

Predictors of LVT formation

Our study assured a bigger insult to the LV as the most detrimental of LVT formation.[1-4,16,17] It was relatively more significant in those with a history of prior MI, more in alien laborers, who probably have a delayed response to MI symptoms, lack a fast response to appropriate medical access and lack social support. The later presentation post-acute MI, the less primary PCI, the longer time from MI to PCI and the suboptimal post-PCI coronary flow were mirrored in higher LVT rates in our study. The patients with LVT are relatively older and have significantly higher total cholesterol and LDL-cholesterol. Elevated total serum cholesterol and elevated LDL-cholesterol are all well-established risk factors for atherothrombotic disorders.[6] LDL-cholesterol promotes platelet activation and tissue factor expression.[18] However, the LDL-cholesterol effect was modest in our adjusted analysis. Importantly, the LV size itself wasn't a significant predictor of LVT development, but LV aneurysms and higher apical WMSI were the strongest predictors of LVT formation.[3,8,17] The later presentation harms the myocardium and delays the use of appropriate medications

and their anticoagulation and anti-inflammatory effects.[14,15,19] The system improvement of early recognition and optimal control of the ischemia time is very reasonable in ameliorating the MI consequences.[5,6] Lower incidences of new LVT at 1 month and 3 months could be a triumph from earlier nuisances. Also, it reflects the impact of modern management, the prevailing high rate of GDMT, the potential recovery of high atherothrombotic milieu and myocardial stunning. Finally, these predictors of LVT formation post-acute MI can guide individualized intervals of TTE screening (at 1 to 2 weeks post MI) and early detection of new LVT or developing a risk score for using OAC in the highest-risk patients post MI.

Predictors of LVT persistence beyond 3 months

We confirmed the prior finding that the type OAC did not predict LVT resolution at three months,[7] this is also supported by more recent research.[20,21] The presence of an LV aneurysm, larger LVT area, higher apical WMSI and lower LVEF were independent predictors of slower LVT resolution and could be a reason for longer use of OAC. Patients with MI who presented late typically have higher chances of all these factors; however, late presentation still stands as an independent risk for LVT formation and persistence. This finding is comparable to several prior studies[16,17] and is potentially the most important modifiable target to improve care systems.[5,6] These predictors could also escort the duration of OAC use in patients with LVT, especially when more sophisticated follow-up modalities like cardiac magnetic resonance are unattainable.[19,22]

Limitations of the study

Our study has some limitations; a large section of the studied cases came from retrospective data with inherent limitations. TTE studies were routine rather than prespecified to rule out LVT, which could impact the reported incidences. We solely used echocardiography, which has known limitations for LVT detection; however, it remains the standard imaging modality for LVT screening and related decisions in the standard practice and recommendations by guidelines.[9,12] Additionally, the potentially missed thrombi by TTE are at a low risk of embolization.[20] The use of OAC was mainly based on physician discretion without a unified protocol, yet contemporary research doesn't favour a specific OAC regimen for the treatment of LVT.[7,21] Finally, the follow-up missing rates were large as many noncitizens who got MI left back home. This could reduce the chance of better assessment of LVT resolution, and can be overcome by separating the analysis of those who completed the follow up.

Conclusion

Compared to older data, we reported a much lower incidence of new LVT formation in the high-risk population of post-acute MI with severe LV dysfunction using TTE, and the incidences of additional new LVT at 1 and 3 months of follow-up are even lower. Global and apical LV systolic dysfunction parameters, older age, and late presentation predict the development of LVT. A bigger size of LVT added to these parameters as predictors of the persistence of LVT beyond 3 months.

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Conflicts of interest

The authors have no relevant financial or non-financial interests to disclose.

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