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Original Article

Left Ventricular Thrombus in Myocardial Infarction After Successful Primary Percutaneous Coronary Intervention: Prevalence and Predictors—A Middle Eastern Single-Centre Experience

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ABSTRACT

Background: Left ventricular thrombus (LVT) is a well-recognized complication of myocardial infarction that affects patient outcomes and warrants screening.

Methods: This retrospective study included 308 consecutive patients who presented with acute ST-elevation myocardial infarction and were treated with primary percutaneous coronary intervention.

Results: Early screening for LVT by echocardiography and cardiac magnetic resonance revealed the following: LVT (+) group (36 patients [11.7%]) and LVT (−) group (272 patients [88.3%]). The 2 powerful independent variables associated with LVT formation were left anterior descending–related infarct (odds ratio, 10.17; $P < 0.0001$) and severe left ventricular systolic dysfunction (odds ratio, 8.3; $P = 0.0001$).

RÉSUMÉ

Contexte : La thrombose du ventricule gauche (TVG) est une complication notoire de l'infarctus du myocarde qui influe sur l'évolution de l'état de santé du patient et nécessite un dépistage.

Méthodologie : Cette étude rétrospective inclu 308 patients consécutifs ayant subi un infarctus aigu du myocarde avec élévation du segment ST et traité par une angioplastie coronaire percutanée primaire.

Résultats : Le dépistage précoce de la TVG par échocardiographie et résonance magnétique cardiaque a révélé que 36 patients (11,7 %) présentaient une TVG, et 272 patients (88,3 %) n'en présentaient pas. Les deux variables indépendantes fortement associées à la TVG étaient l'infarctus dans le territoire de l'artère interventriculaire antérieure

Left ventricular thrombus (LVT) is a well-known complication after ST-segment elevation myocardial infarction (STEMI) and a known cause of systemic embolic events.[1] The incidence of LVT varies, but is reported as being up to 46% in some studies before the thrombolytic era.[2] This has improved with the advancement of reperfusion therapies for acute myocardial infarction, including primary percutaneous coronary intervention (PCI),[3-6] but is still of significant concern.[7]

This is a single cardiac centre study with 2 aims: (1) to determine the prevalence of early LVT formation in patients with acute STEMI who are treated successfully with primary PCI (PPCI); and (2) to determine its predictors and test if the risk of LVT formation increases with dehydration caused by certain environmental factors (i.e., hot climate, exercise).

Methods

Study population

A total of 308 patients who were admitted to King Abdullah Medical City in Makkah with acute STEMI and successfully treated with PPCI in 2017 were included. Successful angioplasty was defined as post-treatment residual stenosis < 30% with thrombolysis in myocardial infarction flow grade 3. All patients presented directly or were referred to our institution, a tertiary hospital with 24-hour/7-day acute interventional facilities. We excluded (1) patients who had a history of intracardiac thrombus before the acute STEMI (the reason for admission) or arterial or venous thrombosis; and (2) patients who were receiving anticoagulation treatment for different reasons (i.e., atrial fibrillation, valve prosthesis).

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Ethics Statement: The research reported has adhered to the relevant ethical guidelines.

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The lower the left ventricular ejection fraction, the higher the risk of LVT was. Multivessel coronary artery disease and the type of early invasive strategy (culprit lesion only vs complete revascularization) were not predictive of LVT. The impact of environment (i.e., hot climate, exercise) and dehydration on the risk of LVT formation is uncertain.

Conclusion: **Early LVT formation is a frequent complication in acute ST-elevation myocardial infarction despite timely intervention. Its independent predictors are left anterior descending–related infarct and severe left ventricular systolic dysfunction. In patients with multivessel coronary artery disease, there was no significant difference between lesion-only culprits and complete revascularization in reducing the risk of LVT development. Further studies in larger numbers of patients are needed because of the uncertainties regarding the links between the biological effects of the environment and the risk of LVT formation.**

(rapport de cotes : 10,17; $p < 0,0001$) et une sévère dysfonction systolique ventriculaire gauche (rapport de cotes : 8,3; $p = 0,0001$). Plus la fraction d'éjection ventriculaire gauche était faible, plus le risque de TVG était élevé. La présence d'une coronaropathie multitronculaire et le type de stratégie de perfusion précoce (revascularisation de la coronaire responsable seulement ou revascularisation complète) ne permettaient pas de prédire la TVG. L'impact des facteurs environnementaux (p. ex. chaleur, effort physique) et de la déshydratation sur le risque de TVG est mal connu.

Conclusion : **La TVG précoce demeure une complication fréquente de infarctus aigu du myocarde avec elevation du segment ST malgré une intervention rapide. L'infarctus lié a l'artère interventriculaire antérieure et une sévère dysfonction systolique ventriculaire gauche sont les facteurs de prédiction indépendants de la TVG. Chez les patients présentant une coronaropathie multitronculaire, il n'y avait pas de différence significative entre l'effet de la revascularisation de la coronaire responsable seulement et celui de la revascularisation complète sur la réduction du risque de TVG. D'autres études auprès d'un plus grand nombre de patients s'imposent, en raison des incertitudes quand aux liens entre les effets biologiques des facteurs environnementaux et le risque de TVG.**

Clinical data

Clinical data included baseline patient characteristics and post–myocardial infarction complications. Electrocardiographic data included territory of STEMI: anterior vs non-anterior (lateral, inferior with or without right ventricle extension). Laboratory data included troponin levels measured on admission and indirect markers of dehydration: creatinine, protein, and hematology parameters (haemoglobin, haematocrit, and platelet count). Angiographic data included the number of significantly diseased coronary arteries, defined as stenosis $> 50\%$ for the left main and $> 70\%$ for the left anterior descending (LAD) arteries, left circumflex artery (LCX), and right coronary artery (RCA), infarct-related artery (IRA), and revascularization strategy: culprit-only vs multivessel revascularization. Culprit-only or single-vessel revascularization was defined as PPCI on the IRA only, and multivessel revascularization was defined as percutaneous intervention on 2 or more lesions in different coronary artery territories (during the initial procedure or planned later as a staged intervention during the same hospitalization). The decision to perform culprit-only or multivessel reperfusion was at the discretion of the interventional cardiologist.

Cardiac imaging data

All patients underwent a baseline transthoracic Doppler echocardiography within 24 hours of hospitalization for acute STEMI and PPCI. In all patients, the first echocardiogram was performed after the initial PPCI procedure. Echocardiography data included heart chamber size, left ventricular ejection fraction (LVEF), wall motion abnormality, valve structure and function, and presence or absence of LVT. LVT was recognized as a delineated echo-dense mass adjacent to, but distinct from, the endocardium in an area of abnormal wall motion seen throughout the cardiac cycle in more than 1 view. A special zoom on the left ventricular (LV) apex was applied and harmonic imaging was used because the majority of thrombi were located at the apex. Two to 3 experienced examiners were involved in reading. If LVT could not be excluded by the baseline echocardiogram, patients underwent a second transthoracic echocardiography study within the next 72 hours or targeted cardiac magnetic resonance imaging to improve thrombus detection. Cardiovascular magnetic resonance (CMR) was used as the second tool, featuring late gadolinium enhancement sequences with a prolonged inversion time (TI 600). With CMR, we assessed the infarct size, scar extension, and presence or absence of LVT, recognized as a hypo-enhanced mass adjacent to the myocardial area of abnormal wall motion. The maximum interval between baseline echocardiography and CMR was 7 days.

Statistical analysis

Continuous data were expressed as mean $\pm$ standard deviation and compared using the Student t test. Categorical data were given as a percentage and compared with a chi-square test. Variables with a P value < 0.20 as tested in univariate analysis were incorporated into the multivariate models as continuous or dichotomous variables. A P value < 0.05 was considered statistically significant. Statistical analyses were performed with JMP 13 software (SAS Institute Inc., Cary, NC).

Results

All patients underwent imaging for LVT during their hospital treatment, which lasted 3 to 15 days. From the transthoracic echocardiographic studies, baseline and serial, LVT was visualized in 29 patients, excluded in 263 patients, and suspected in 16 patients. The latter received CMR during the same hospitalization (with a maximum interval of 7 days), allowing us to confirm LVT in 7 patients and rule it out in 9 patients. Thereafter, patients were divided into 2 groups: (1) LVT (+): 36 patients with LVT (11.7% of 308); (2) LVT (−): 272 patients without LVT (88.3% of 308).

Table 1. Baseline data in the whole cohort and comparison of both groups

Variables	Whole cohort n = 308	Group LVT (+) n = 36 (11.7%)	Group LVT (−) n = 272 (88.3%)	*P* value
I Clinical data				
Age, y	56 ± 10	55 ± 10	56 ± 11	NS
Male gender, n (%)	257 (83)	33 (92)	224 (82)	NS
Race, n (%)				
Asian	276 (90)	33 (92)	243 (89)	NS
African	32 (10)	3 (8)	29 (11)	NS
Pilgrim patient	81 (26)	7 (19)	74 (27)	NS
Obesity, n (%)	208 (67)	27 (75)	181 (66)	NS
Hypertension, n (%)	144 (47)	17 (47)	127 (47)	NS
Diabetes, n (%)	167 (54)	19 (53)	148 (54)	NS
Dyslipidemia, n (%)	45 (15)	6 (17)	39 (14)	NS
History of CAD, n (%)	27 (9)	2 (6)	25 (9)	NS
Killip class IV, n (%)	8 (2.6)	0	8 (3)	NS
Peri PPCI CPR, n (%)	11 (3.6)	1 (3)	10 (4)	NS
Peri PPCI IABP, n (%)	21 (7)	7 (19)	14 (5)	NS
IIb/IIIa inhibitor, n (%)	99 (32)	16 (44)	83 (31)	NS
Inotropes, n (%)	33 (11)	4 (11)	29 (11)	NS
II Electrocardiographic data				
Anterior STEMI, n (%)	171 (55.5)	34 (94.4)	137 (50)	< 0.0001
Nonanterior STEMI, n (%)	137 (44.5)	2 (6)	135 (50)	< 0.0001
III Laboratory data				
Peak troponin (ng/mL)	161 ± 276	204 ± 49	155 ± 18	NS
Creatinine (mg/dL)	1.3 ± 1.2	1.6 ± 0.2	1.3 ± 0.1	NS
Protein (g/dL)	6.5 ± 1.1	6.7 ± 0.4	6.5 ± 0.2	NS
Platelet count (platelet/μL)	255 ± 92	271 ± 16	252 ± 6	NS
Haemoglobin (g/dL)	14 ± 2	14 ± 0.3	14 ± 0.1	NS
Hematocrit (%)	38 ± 6	38.2 ± 2.7	38.0 ± 7	NS

CAD, coronary artery disease; CPR, cardiopulmonary resuscitation; IABP, intra-aortic balloon pump; LVT, left ventricular thrombus; NS, not significant; PPCI, primary percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction.

As shown in Table 1, the demographic data, cardiovascular risk factors, and history of CAD were similar in both groups. Eight patients presented with cardiogenic shock, but none were in the LVT (+) group; 11 patients had in-hospital resuscitated cardiac arrest, but only 1 was in the LVT (+) group. No significant difference was found between the 2 groups regarding the need for inotrope support, glycoprotein IIb/IIIa inhibitors, and intra-aortic balloon pump. More than half of the patients (171, 55.5%) had anterior STEMI diagnosed by ECG, and this was predominant in the LVT (+) group. There was no significant difference between groups in cardiac enzyme release. Other laboratory parameters we tested as indirect markers of dehydration and hemoconcentration showed increased levels in the LVT (+) group (creatinine, protein, haematocrit

Table 2. Angiographic findings and intervention in the whole cohort and comparison of both groups

Variables	Whole cohort (n = 308)	Group LVT (+) n = 36 (11.7%)	Group LVT (−) n = 272 (88.3%)	*P* value
No. of significantly diseased coronary arteries				
Single-vessel disease, n (%)	107 (35)	15 (42)	92 (34)	NS
Multivessel disease, n (%)	201 (65)	21 (58)	180 (66)	NS
2 coronary arteries, n (%)	86 (28)	9 (25)	77 (28)	NS
3 coronary arteries, n (%)	115 (37)	12 (33)	103 (38)	NS
IRA				
LM, n (%)	1 (0.3)	0	1 (0.4)	
LAD, n (%)	169 (55)	34 (94)	135 (50)	< 0.0001
LCX, n (%)	34 (11)	1 (3)	33 (12)	< 0.0001
RCA, n (%)	104 (34)	1 (3)	103 (38)	< 0.0001
Procedure in 107 patients with single-vessel disease				
PPCI to LM, n (%)	1 (1)	0	1	
PPCI to LAD, n (%)	76 (71)	15 (100)	62 (67)	0.009
PPCI to RCA, n (%)	21 (19.6)	0	21 (100)	0.03
PPCI to LCX, n (%)	9 (8.4)	0	9 (100)	NS
Procedure in 201 patients with multivessel disease:				
Culprit lesion-only PPCI, n (%)	167 (83)	20 (95)	147 (82)	NS
Complete revascularization,* n (%)	34 (17)	1 (5)	33 (18)	NS

IRA, infarct-related artery; LAD, left anterior descending; LCX, left circumflex; LM, left main; NS, not significant; PPCI, primary percutaneous coronary intervention; RCA, right coronary artery.

*Complete revascularization at the time of PPCI.

Table 3. Echocardiographic data in the whole cohort and comparison of both groups

Variables	Whole cohort (n = 308)	Group LVT (+) n = 36 (11.7%)	Group LVT (−) n = 272 (88.3%)	*P* value
Dilated LVEDV, n (%)	12 (4)	1 (3)	11 (4)	NS
LVEF (mean ± SD), %	39 ± 1	31 ± 2	40 ± 1	< 0.0001
LV systolic dysfunction				
Mild dysfunction, n (%)	133 (43)	3 (8.3)	130 (48)	< 0.0001
Moderate dysfunction, n (%)	92 (30)	13 (36)	79 (29)	< 0.0001
Severe dysfunction, n (%)	83 (27)	20 (55)	63 (23)	< 0.0001
LV diastolic dysfunction				
Grade I, n (%)	244 (79)	27 (75)	217 (80)	NS
Grade II/III, n (%)	44 (14)	9 (25)	35 (13)	NS
MR grade III/IV, n (%)	31 (10)	1 (3)	30 (11)	NS

LV, left ventricle; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVT, left ventricular thrombus; MR, mitral regurgitation; NS, not significant; SD, standard deviation.

concentration, and platelet counts); however, the difference between groups was not statistically significant.

Table 2 shows that there was no significant difference between groups with regard to the extent of CAD as reflected by the total number of coronaries with angiographically significant stenosis. The majority of the study population had multivessel disease (65%). The LAD vessel was the cause in more than half of patients (55%), followed by the RCA in more than one-third (34%), and then the LCX in more than one-tenth (11%). The culprit LAD lesion was significantly more prevalent in the group with LVT: 94% vs 50% in the LVT (−) group ($P < 0.0001$). Regarding the revascularisation strategy in the subset of patients who presented with multivessel disease (201 patients), culprit lesion-only PPCI was preferred over complete revascularisation in both groups. Only 17% of them were considered for complete reperfusion at the time of PPCI. In addition, there was no significant difference with regard to these 2 reperfusion strategies between the 2 groups (Table 2).

Multi-modality imaging (echocardiography and CMR) showed that all LV thrombi were located apically, and nearly all (94%) occurred in the context of LAD-related infarct. Only 6% of LVT cases were concomitant to RCA or LCX artery−related infarct. As shown in Table 3, left ventricle systolic dysfunction was significantly associated with LVT development. In fact, LVEF was remarkably lower in the LVT (+) group: 31% vs 40% in the LVT (−) group. Also, the proportion of patients with moderate LV dysfunction (30% < LVEF ≤ 40%) and severe LV dysfunction (LVEF ≤ 30%) was significantly higher in the LVT (+) group than in the LVT (−) group: 36% and 55% vs 29% and 23%, respectively ($P < 0.0001$). No remarkable difference in LV diastolic dysfunction or mitral regurgitation was noted between the 2 groups (Table 3).

Analysis of outcome predictors

On univariate analysis (Table 4), LV systolic dysfunction expressed in continuous and dichotomous format was significantly associated with increased risk of LVT development. The lower the LVEF, the higher the risk: Odds ratios (ORs) were 7.1 and 13.7 for moderate and severe LV systolic dysfunction, respectively ($P < 0.0001$). Anterior localization of STEMI, or LAD-related STEMI, was the second significant risk factor for LVT development ($P < 0.0001$). However, patients with multivessel CAD diagnosed were not at higher risk for LVT compared with patients with single-vessel disease. In addition, with regard to multivessel disease, the present study did not show a superior protective effect of one reperfusion strategy over another (culprit-only vs multivessel PCI) with regard to occurrence of LVT.

Age, gender, race, cardiovascular risk factors (diabetes mellitus, hypertension, dyslipidemia), peak of troponin, and LV dilatation were tested and not predictive of LVT occurrence. On multivariate analysis (Table 4), the remarkable independent predictors of LVT occurrence identified in the early course of acute STEMI were (1) occluded LAD as culprit lesion (OR, 10.17; $P < 0.0001$); and (2) significant LV systolic dysfunction (the highest risk was found with severe LV dysfunction (OR, 8.3; $P = 0.0001$).

Discussion

LVT prevalence

An important finding in our study is the high incidence (11.7%) of LVT early in post−acute infarct despite successful reperfusion with PPCI. Our finding is consistent with other studies from the contemporary era of coronary stenting, reporting a prevalence ranging from 2.9% to 15% in the early course post-STEMI.[1,3-6] Of note, comparison of data from the thrombolytic era with earlier data from the pre-thrombolytic era, when LVT prevalence was up to 46%, indicates a declining incidence of LVT with the widespread adoption of PPCI and the use of aggressive antithrombin and antiplatelet therapy.[8] This can be attributed to timely and effective reperfusion by means of PPCI, a technique that has been shown to be more beneficial than fibrinolysis and to result in better myocardial salvage, less reinfarction, and lower mortality.[9,10]

The relatively high prevalence of LVT (11.7%) in our study can be explained by the site and size of myocardial infarction in our population. The majority of the LVT (+) group (94%) had STEMI in the LAD territory vs 50% in the LVT (−) group ($P < 0.0001$). Anterior STEMI is known to be associated with the highest prevalence of LVT, as high as 34% to 57%,[11-14] irrespective of the reperfusion strategy used. According to a recent meta-analysis[15] of 19 studies that included 10,076 patients treated with PPCI, the rate of LVT formation was substantially higher after anterior STEMI compared with overall STEMI (9.1% vs 2.7%, respectively). Furthermore, in our study patients with anterior STEMI and LVT, a proximal LAD lesion was the culprit in 73% of cases

Table 4. Univariate and multivariate analysis of predictors of LVT formation

	Univariate analysis		Multivariate analysis	
Whole cohort (n = 308)	*P* value	OR (95% CI)	*P* value	OR (95% CI)
Age	NS	1.01 (0.98-1.05)		
LVEF, %	< 0.0001	1.08 (1.05-1.12)		
Moderate LV systolic dysfunction	< 0.0001	7.1 (2.21-31.7)	0.0264	3.9 (1.16-18.03)
Severe LV systolic dysfunction	< 0.0001	13.7 (4.5-59.9)	0.0001	8.3 (2.59-37.58)
Anterior STEMI	< 0.0001	12.2 (4.7-41.4)	< 0.0001	10.17 (2.87-64.89)
Single vs multivessel CAD	NS	0.71 (0.35-1.47)		
CL-only PCI vs MV-PCI*	NS	0.22 (0.022-1.12)		

Increment for age is 1 year and for LVEF is 1%.

CAD, coronary artery disease; CI, confidence interval; CL-only PCI, culprit lesion-only percutaneous coronary intervention; LV, left ventricle; LVEF, left ventricular ejection fraction; NS, not significant; OR, odds ratio; STEMI, ST-elevation myocardial infarction.

*In patients with multi-CAD.

and middle LAD in 27%. This reflects the larger infarct area in most of the anterior STEMI subgroup and subsequently more extensive wall motion abnormality and greater risk of LVT formation. Even the remaining 2 patients from the LVT (+) group who had nonanterior STEMI had a significant LAD lesion (stenosis > 70%) in addition to the culprit non-LAD lesion with diffuse regional wall motion abnormalities involving the apex and severe LV systolic dysfunction. Myocardial damage and the extent of wall motion abnormality in the infarct zone and the surrounding noninfarcted myocardium (adjacent nonischemic dyskinesia phenomenon) are powerful factors that may result in a more substantial asynergic area in the acute phase of infarction and that influence thrombus formation.[16,17] Furthermore, echocardiographic studies have shown that severe apical asynergy or dyskinesia is often present in anterior STEMI and highly predisposes to LVT formation.[11,18]

The other potential factor contributing to the high prevalence of LVT in our study is the repeated cardiac imaging for LVT screening. In fact, 96 patients (31%) required a second echocardiographic examination a few days after the baseline study done early postadmission. This helped to confirm some of the initially suspicious LVT and probably to detect other thrombi that developed later, that is, over the first week after the PPCI, as has been shown in previous reports.[19,20] Optimizing imaging by combining standard transthoracic echocardiography to contrast CMR improved screening of LVT in 16 of our patients and led to the detection of 7 additional cases with LVT. Although the specificity of transthoracic echocardiography for LVT diagnosis is high (95% to 98%), its sensitivity is low, only 21% to 35%.[21] The superiority of CMR to transthoracic echocardiography has been emphasized in previous studies and is considered the diagnostic tool of choice with the highest sensitivity (82% to 88%) and specificity (99% to 100%).[8,21,22] The integration of echocardiography, cardiac computed tomography, and magnetic resonance imaging has incremental diagnostic value and is warranted in at-risk patients or whenever suspicion of thrombi is high.

LVT predictors

Our study supports previous data[6,23] that LAD-related STEMI has the highest risk for early appearance of LVT in patients post-STEMI. Our findings showed a 10-fold increase in the risk of LVT formation in patients with anterior STEMI. Similar data were reported by Mao et al.[6] in a large series of patients with acute STEMI treated with PPCI that showed LAD intervention was independently associated with LV thrombus (a 7.58-fold increased risk of LVT). On the other hand, the presence of multivessel CAD (compared with single-vessel disease) in our cohort did not emerge as a significant predictor for LVT. With respect to reperfusion therapy in patients with multivessel disease, PPCI of the IRA was predominantly the treatment of choice; however, there was no statistically significant difference between culprit lesion-only PCI and complete revascularization in preventing LVT development. However, this result is limited by the small sample size of patients who had complete revascularization at the time of PPCI.

The second major independent predictor of LVT formation we reported is LV systolic dysfunction, which is consistent with previous reports.[4,5,24-26] The lower the LVEF, the higher the risk of LVT (3.90-fold and 8.3-fold higher for LVEF 30% to 40% and < 30%, respectively). The severity of LV systolic dysfunction affects not only the risk of LVT formation but also its timing. The early appearance of thrombus in our cohort, as early as a few hours after myocardial infarction, is of importance and was emphasized in a previous series.[5,25,27] As in our study, the study by Nesković et al.[27] showed an early in-hospital LVT formation after acute myocardial infarction in the setting of initial low LVEF of ≤ 40%.

On the other hand, we hypothesized that thrombus formation is affected by dehydration, a condition made more likely in our study population by multiple factors. First, Makkah province features a hot desert climate, and more than half of the patients who had LVT (20/36 patients) received their diagnosis during the long hot season. This lasts from May to September, with daily temperatures often exceeding 102°F. Second, more than half of the patients are aged more than 55 years (57% in the whole cohort and half of the LVT (+) group), making them more vulnerable to fluid loss and dehydration. Third, our cohort included 81 pilgrim patients (26%). While performing hajj (during the hot season), physical effort and transpiration are particularly high in these patients, who represent 19% of the LVT (+) group. Consistent with the diagnosis of dehydration, laboratory test results reflecting blood concentration (creatinine, protein, and

haematocrit levels) tended to be higher in the LVT (+) group. However, against our hypothesis, LVT was not significantly more prevalent during the sweltering summer season, when heatwaves are extreme, compared with other seasons: 12.5% and 11.5%, respectively (P = not significant). In addition, appropriate dehydration markers (i.e., plasma and urine osmolality) were not systematically tested, which would be helpful for diagnostic accuracy. Further studies in a larger number of patients are necessary to examine the relationship between the biological effects of environment (i.e., hot climate, exercise) and LVT formation in post–acute myocardial infarction.

Conclusion

Early thrombus formation is still a common complication of STEMI even after timely reperfusion. It is predictable in cases of large LAD-related infarct and significant LV systolic dysfunction. The multivessel CAD and reperfusion strategy (culprit-only vs complete revascularization) did not emerge as a significant predictor for LVT. Screening of LVT in patients at risk should integrate complementary imaging modalities using contrast agents (contrast echocardiography, CMR, cardiac computed tomography). Because of uncertainties regarding the relationship between the biological effects of environment (i.e., hot climate, exercise) and the formation of LVT, further studies are needed with larger numbers of patients.

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Disclosures

All authors have no conflicts of interest to declare.

References

1. Bhatnagar SK, Al Yusuf AR. Left ventricular thrombi after acute myocardial infarction. Postgrad Med J 1983;59:495-9.
2. Lamas GA, Vaughan DE, Pfeffer MA. Left ventricular thrombus formation after first anterior wall acute myocardial infarction. Am J Cardiol 1988;62:31-5.
3. Kalra A, Jang IK. Prevalence of early left ventricular thrombus after primary coronary intervention for acute myocardial infarction. J Thromb Thrombolysis 2000;10:133-6.
4. Zielinska M, Kaczmarek K, Tylkowski M. Predictors of left ventricular thrombus formation in acute myocardial infarction treated with successful primary angioplasty with stenting. Am J Med Sci 2008;335:171-6.
5. Gianstefani S, Douiri A, Delithanasis I, et al. Incidence and predictors of early left ventricular thrombus after ST-elevation myocardial infarction in the contemporary era of primary percutaneous coronary intervention. Am J Cardiol 2014;113:1111-6.
6. Mao TF, Bajwa A, Muskula P, et al. Incidence of left ventricular thrombus in patients with acute ST-segment elevation myocardial infarction treated with percutaneous coronary intervention. Am J Cardiol 2018;121:27-31.
7. Osherov AB, Borovik-Raz M, Aronson D, et al. Incidence of early left ventricular thrombus after acute anterior wall myocardial infarction in the primary coronary intervention era. Am Heart J 2009;157:1074-80.
8. McCarthy CP, Vaduganathan M, McCarthy KJ, et al. Left ventricular thrombus after acute myocardial infarction: screening, prevention, and treatment. JAMA Cardiol 2018;3:642-9.
9. Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet 2003;361:13-20.
10. Nielsen PH, Maeng M, Busk M, et al. Primary angioplasty versus fibrinolysis in acute myocardial infarction: long-term follow-up in the Danish acute myocardial infarction 2 trial. Circulation 2010;121:1484-91.
11. Asinger RW, Mikell FL, Elsperger J, Hodges M. Incidence of left-ventricular thrombosis after acute transmural myocardial infarction. Serial evaluation by two-dimensional echocardiography. N Engl J Med 1981;305:297-302.
12. Domenicucci S, Chiarella F, Bellotti P, et al. Early appearance of left ventricular thrombi after anterior myocardial infarction: a marker of higher in-hospital mortality in patients not treated with antithrombotic drugs. Eur Heart J 1990;11:51-8.
13. Keren A, Goldberg S, Gottlieb S, et al. Natural history of left ventricular thrombi: their appearance and resolution in the posthospitalization period of acute myocardial infarction. J Am Coll Cardiol 1990;15:790-800.
14. Mooe T, Teien D, Karp K, Eriksson P. Left ventricular thrombosis after anterior myocardial infarction with and without thrombolytic treatment. J Intern Med 1995;237:563-9.
15. Robinson AA, Jain A, Gentry M, McNamara RL. Left ventricular thrombi after STEMI in the primary PCI era: a systematic review and meta-analysis. Int J Cardiol 2016;221:554-9.
16. Kerber RE, Marcus ML. Evaluation of regional myocardial function in ischemic heart disease by echocardiography. Prog Cardiovasc Dis 1978;20:441-50.
17. Delemarre BJ, Visser CA, Bot H, Dunning AJ. Prediction of apical thrombus formation in acute myocardial infarction based on left ventricular spatial flow pattern. J Am Coll Cardiol 1990;15:355-60.
18. Friedman MJ, Carlson K, Marcus FI, Woolfenden JM. Clinical correlations in patients with acute myocardial infarction and left ventricular thrombus detected by two-dimensional echocardiography. Am J Med 1982;72:894-8.
19. Habash F, Vallurupalli S. Challenges in management of left ventricular thrombus. Ther Adv Cardiovasc Dis 2017;11:203-13.
20. Shacham Y, Leshem-Rubinow E, Ben Assa E, et al. Frequency and correlates of early left ventricular thrombus formation following anterior wall acute myocardial infarction treated with primary percutaneous coronary intervention. Am J Cardiol 2013;111:667-70.
21. Srichai MB, Junor C, Rodriguez LL, et al. Clinical, imaging, and pathological characteristics of left ventricular thrombus: a comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathological validation. Am Heart J 2006;152:75-84.

22. Delewi R, Nijveldt R, Hirsch A, et al. Left ventricular thrombus formation after acute myocardial infarction as assessed by cardiovascular magnetic resonance imaging. Eur J Radiol 2012;81:3900-4.

23. Sürder D, Gisler V, Corti R, et al. Thrombus formation in the left ventricle after large myocardial infarction — assessment with cardiac magnetic resonance imaging. Swiss Med Wkly 2015;145:w14122.

24. You J, Wang X, Wu J, et al. Predictors and prognosis of left ventricular thrombus in post-myocardial infarction patients with left ventricular dysfunction after percutaneous coronary intervention. J Thorac Dis 2018;10:4912-22.

25. Driesman A, Hyder O, Lang C, et al. Incidence and predictors of left ventricular thrombus after primary percutaneous coronary intervention for anterior ST-segment elevation myocardial infarction. Clin Cardiol 2015;38:590-7.

26. Okuyan E, Okcun B, Dinçkal MH, Mutlu H. Risk factors for development of left ventricular thrombus after first acute anterior myocardial infarction-association with anticardiolipin antibodies. Thromb J 2010;8:15.

27. Nesković AN, Marinković J, Bojić M, Popović AD. Predictors of left ventricular thrombus formation and disappearance after anterior wall myocardial infarction. Eur Heart J 1998;19:908-16.