Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/jptcp.v30i2.3013

PREDICTORS OF LEFT VENTRICULAR THROMBUS IN PATIENTS UNDERGOING PRIMARY PERCUTANEOUS CORONARY INTERVENTION FOR ANTERIOR ST SEGMENT ELEVATION MYOCARDIAL INFARCTION

Arafa G. Ibrahim[1]*, Esam Ahmed khalil[2], Mahmoud F. Elshahat[3]

[1]*,[3]MD Cardiology Department, Helwan University, Cairo, Egypt.
[2]MD Cardiology Department, Al-Azhar university, Cairo, Egypt.

***Corresponding Author**: Arafa G Ibrahim
*MD Cardiology Department, Helwan University, Cairo, Egypt,
Email: arafa.gomaa@med.helwan.edu.eg

Submitted: 10.01.2023; **Accepted:** 05.02.2023; **Published:** 15.02.2023

Abstract

Background: Acute ST-segment elevation myocardial infarction (STEMI) is linked to substantial morbidity and death. The purpose of this research was to investigate the frequency and probability for LV thrombus (LVT) in acute anterior STEMI patients receiving primary percutaneous intervention (PPCI).

Methods: In our research, 200 patients over the age of 20 who had acute anterior STEMI and received PPCI. It was an observational study. Demographic data, clinical examination, electrocardiogram (ECG), laboratory, two-dimensional echocardiography (2D ECHO), coronary angiography and PPCI data of all patients were collected.

Results: The prevalence of LVT was 15 (7.5%) of all the study population. On univariate regression analysis, D-dimer, poor EF, E/A ratio, time for deceleration, E/e', E', WMSI and post primary PCI impaired flow less than TIMI 2 flow were significant predictors of the incidence of thrombus. On multiple regression analysis, only D-dimer, E/A ratio, E/e' and WMSI were significant predictors of the incidence of LVT.

Conclusions: LVT is a frequent complication of patient presented with anterior STEMI. The prevalence of LVT was 15 (7.5%) of all the study population. Poor LV EF, post primary PCI impaired flow less than TIMI 2 flow, WMSI, E/A ratio, time for deceleration, E', E/e' and D-dimer was associated was higher prevalence of LVT.

Keywords: Left Ventricular Thrombus; St Segment Elevation Myocardial Infarction; Primary Percutaneous Coronary Intervention

INTRODUCTION

Acute ST-segment elevation myocardial infarction (STEMI) poses a major hazard to human life and health due to its high morbidity and deaths. The frequency of STEMI is increasing. [1]. Although dual antiplatelet treatment (DAPT) and primary percutaneous coronary intervention (PPCI) have enhanced survival in STEMI suffers during the last 20 years. Complications after myocardial infarction continue

to be a major contributor to high mortality and disability. [2, 3].
Following anterior STEMI, the advancement of a left ventricular thrombus (LVT) that causes a cerebrovascular accident is a potentially fatal complication that lengthens hospital stays and consumes excessive amounts of medical resources. [4].
When compared to the thrombolytic period, the rate of LVT has reduced in the PPCI era, yet it still ranges from 2.9% to 15%. In individuals with anterior and apical STEMI, LVT is more common. Systemic anticoagulation with warfarin reduces the likelihood of systemic embolization when an LV thrombus is verified. [5]. Previous research showed that in patients with AMI who had primary PCI, LVEF <45% is an effective predictor of death. [6, 7].
In the PPCI for STEMI period, the overall rate of LVT by transthoracic echocardiography (TTE) was 2.7%, with 9.1% for anterior STEMI, according to a recent meta-analysis. [8]. While CMR with gadolinium contrast agent may detect LV thrombus depend on both its shape and tissue properties, TTE depends on the morphological identification of LVT. The prognosis of LVT in post-MI and LV malfunction individuals who had PCI is seldom described, however, and neither are the predictors of LVT in these individuals. [9, 10].
to assess the frequency and risks for LVT in patients with acute anterior STEMI receiving initial PCI.

PATIENTS AND METHODS

Our study included 200 patients presented with acute anterior STEMI and underwent PPCI treatment admitted to Cardiology department Helwan University Hospitals from October 2021 to December 2022. Each patient freely supplied their informed permission in writing.
Participants in the research had anterior STEMI.
Exclusion criteria were Significant valvular disease, thrombotic hematological diseases, history of arterial or venous thrombosis, or non-anterior STEMI, dilated cardiomyopathy (DCM).
The 4th Universal Definition of Myocardial Infarction states, STEMI is characterized by ischemic signs, an increase in enzyme (CK-MB, TNT, and MYO) levels that is three times above the upper limit of typical, along with concurrent dynamic changes in the electrocardiogram (ECG), such as new left bundle branch blocks, Q waves, ST segment alterations, and irregular ventricular wall motion as seen by echocardiography. [11].
Demographic data of all patients were collected including: (age, sex, BMI), history of comorbidities (HTN, diabetes, smoking, Hyperlipaemia, smoking, previous medication, family history of CAD. Presenting hemodynamic data was recorded including heart rate and blood pressure. Initial laboratory investigations including cardiac biomarker assay (TNT, CK-MB, and Myoglobin), complete blood count (Hb, Platelets, WBCS), HbA1C, Kidney function, Lipid profile (TG, TC, LDL, and HDL), PT, Baseline INR, D-dimer and CRP.

Investigations:

12-lead ECGs were taken at the time of arrival, just after the angioplasty, and again at 3, 6, 12, 24, and 48 hours.

The procedures

In accordance with the guidelines of the most recent guideline, femoral or radial approaches will used to conduct coronary angiography and PCI [12].
Clopidogrel (600 mg) aspirin (300 mg) and or ticagrelor (180 mg) will administer as a loading dose. the operation date, intraprocedural drugs, procedural findings, and results will be examined in cardiac catheterization procedure records and reports. the degree of coronary perfusion is quantified using TIMI flow grades. there is no antegrade flow or perfusion beyond the blockage in a grade 0 situation. grade 1 indicates that the contrast material was able to flow through the blockage during the cineangiographic recording series without totally obstructing the coronary bed distal to the obstruction. the coronary artery distal to the occlusion is opacified by the contrast material, indicating grade 2 perfusion. contrast material enters and leaves the distal bed at a much slower pace than in

regions unaffected by the prior closure, such as the neighboring coronary bed or the opposite coronary artery. complete perfusion (grade 3) is indicated when contrast material is cleared from the affected bed at the same rate as it is cleared from an unaffected bed in the same or opposite artery. This indicates that blood is flowing from the bed distal to the obstruction at the same rate as it is flowing from the bed proximal to the obstruction (antegrade flow). [13]

Two dimensional (2D) and Doppler echocardiography:

A Doppler and 2D echocardiography will performed approximately 7 days after the onset of symptoms, with a possible range of 5 to 9 days. standard parasternal and apical views. Regional function evaluation followed the American Society of Echocardiography's 17-segment model, using a grading scale from 1 to 4 (1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia and 5= aneurysmal). calculate the wall motion score index (WMSI). An LV thrombus typically appears as an echo-dense mass distinct from the surrounding endocardium and adjacent to an akinetic or hypokinetic LV segment or aneurysm most commonly observed at the LV apex because this is the region of slowest flow and greatest stasis after large anteroseptal MI in the territory supplied by the LAD. ejection fraction (EF) was determined using the Simpson method, incorporating the apical 2- and 4-chamber images. Pulsed-wave Doppler was employed to measure the mitral valve inflow velocity, with the Doppler sample volume positioned at the leaflet tips in the apical 4 chamber view. Additionally, parameters such as the E/A ratio, E velocity deceleration time (DTE), and peak atrial contraction velocity (A) were recorded [15].

Sample size calculation

Utilizing the statistical software program EpI-Info 2002 created by the Centres for Disease Control and Prevention (CDC), the World Health Organization (WHO), the sample size calculation was carried out. N > 185 was chosen as the sample size based on the following factors: In a comprehensive review and meta-analysis with 80% research power, the incidence of LVT was discovered in around 2.7-9.1% of post-MI patients in the modern PCI period. [8]. To prevent dropouts, 15 cases were added to each group.

STATISTICAL ANALYSIS

IBM, Chicago, Illinois, USA's SPSS v27 was utilized for the statistical study. Histograms and the Shapiro-Wilks test were utilized to assess the normality of the data distribution. Unpaired student t-test was utilized to evaluate quantitative parametric data that were shown as mean and standard deviation (SD). The Mann Whitney-test was utilized to assess quantitative non-parametric data, which were provided as the median and interquartile range (IQR). When applicable, qualitative data were examined utilizing the Chi-square test or Fisher's exact test and provided as frequency and percentage (%). Regression analysis, both univariate and multivariate, was utilized to predict the likelihood of thrombus formation. A two-tailed P value of< 0.05 was deemed statistically substantial.

RESULTS

In our study, the rate of thrombus was 7.5% (15/200 patients. There was no statistically substantial variation in the demographics of the two groups, however the percentage of patients with LVT who had a family history of CAD was much greater (80% vs. 7.03, P <0.001) than that of the control group. Table 1

SBP was substantially reduced in LVT group compared to control group (P=0.006) whereas HR and DBP were insignificantly different between both groups. D-dimer, Cholesterol and CKMB were substantially greater in LVT group contrasted to control group (P<0.001, 0.021, 0.002 respectively) whereas other investigations were insignificantly different between both groups. Table 2

LVT group had significantly lower EF, deceleration time, E' and TIMI (P<0.001). LVT group had substantially higher E/A, WMSI, and E/e' (P<0.001) compared to control group whereas LVEDD was comparable between both groups. TIMI flow was substantially reduced in LVT group than in control

group. Table 3

During hospitalization, using of Clopidogrel, LMWH and Beta blocker was substantially lower among patients in LVT group (P<0.001, 0.018, 0.001 respectively), using of Cilostazol, Warfarin and Diuretics was substantially higher among patients in LVT group (P=0.013, <0.001, 0.024 respectively). Table 4

Cardiogenic shock found in 2 (13.33%) patients in LVT group and in 66 (33%) individuals in control group. The incidence of cardiogenic shock was insignificantly varied between the studied groups (P=0.094).

On univariate regression analysis, D-dimer, lower EF, E/A ratio, time for deceleration, E/e', E', WMSI and TIMI flow < 2 were significant predictors of the incidence of LVT. On multiple regression analysis, only D-dimer, E/A ratio, E/e' and WMSI were significant predictors of the incidence of thrombus. Table 5

Table 1: Baseline clinical features of the studied groups

		LVT group (n=15)	Control group (n=185)	P value
Age (year)		60.3 ± 12.31	56.8 ± 10.76	0.277
Sex	**Male**	12 (80%)	170 (91.89%)	0.426
	Female	3 (20%)	15 (8.11%)	
BMI (Kg/m^2)		24.8 ± 1.21	24.5 ± 1.06	0.369
Family history of CAD		12 (80%)	13 (7.03%)	**< 0.001***
HTN		10 (66.67%)	110 (59.46%)	0.784
DM		7 (46.67%)	63 (34.05%)	0.482
Hyperlipidemia		3 (20%)	54 (29.19%)	0.562
Smoking		7 (46.67%)	135 (67.5%)	0.062
Prior MI		3 (20%)	79 (42.7%)	0.105

Data presented as mean ± SD or frequency (%), BMI: body mass index, HTN: hypertension, *: statistically substantial as P value <0.05

Table 2: Vital signs and laboratory examination of the studied groups

	LVT group (n=15)	Control group (n=185)	P value
Vital signs			
HR (beats/min)	81.3 ± 8.5	82.8 ± 7.9	0.471
SBP (mmHg)	115.3 ± 7.4	120.6 ± 7.1	**0.006***
DBP (mmHg)	74.7 ± 10.6	74.9 ± 9.6	0.939
Laboratory examination			
Hb (g/dL)	11.6 ± 1.2	11.8 ± 1.3	0.557
Platelets (*10^9/L)	287.7 ± 78.5	259.3 ± 75.9	0.166
WBCs (*10^9/L)	7.4 ± 1.8	7.9 ± 1.9	0.416
PT (s)	11.5 ± 0.89	11.6 ± 0.96	0.649
INR	1.1 ± 0.11	1.2 ± 0.14	0.121
CRP (mg/dL)	9.0 ± 0.65	8.5 ± 1.05	0.079
D-dimer	8.9 ± 2.1	0.9 ± 0.22	**<0.001***
Serum creatinine (mg/dL)	1.7 (1.5-2)	1.7 (1.5-1.9)	0.452
Urea (mg/dL)	22.6 ± 7.27	19.4 ± 6.01	0.051
TG (mg/dL)	162.6 ± 17.74	164.6 ± 19.76	0.705
Cholesterol (mg/dL)	194.9 ± 18.38	182.6 ± 19.8	**0.021***
LDL (mg/dL)	98.5 ± 9.01	94 ± 8.95	0.066
HDL (mg/dL)	5.47 ± 0.52	5.52 ± 0.5	0.699
CKMB	57.1 ± 6.71	45.6 ± 13.8	**0.002***
HbA1C %	6.4 ± 0.42	6.3 ± 0.49	0.214

Data showed as mean ± SD, median (IQR) or frequency (%), SBP: systolic blood pressure, DBP: diastolic blood pressure, PT: prothrombin time; Hb: hemoglobin, TG: triglyceride; WBCs: white blood cell count, CK-MB: creatinine kinase MB *: statistically substantial as P value <0.05

Table 3: Echocardiography, Tissue Imaging data and TIMI of the studied groups

	LVT group (n=15)	Control group (n=185)	P value
EF %	36.5 ± 3.2	39.5 ± 3.0	**<0.001***
LVEDD (mm)	55.7 ± 1.44	56 ± 1.39	0.469
E/A	1.9 ± 0.74	1.1 ± 0.22	**<0.001***
Deceleration time (ms)	133.6 ± 13.39	156.1 ± 8.44	**<0.001***
WMSI	1.97 ± 0.21	1.6 ± 0.14	**<0.001***
E'	5.4 ± 1.12	6.5 ± 1.1	**<0.001***
E/e'	16.3 ± 1.5	13.9 ± 1.45	**<0.001***
TIMI	1 (0-1)	2 (1-3)	**<0.001***
The time between diagnosis and Primary PCI (in minutes)	96.7 ± 2.09	97.5 ± 1.82	0.099

LVEDD: left ventricular end-diastolic dimension; E/A: early to atrial filling velocity ratio; *: statistically substantial as P value <0.05

Table 4: Medications taken by the examined groups during hospitalization

	LVT group (n=15)	Control group (n=185)	P value
Aspirin	14 (93.3%)	178 (96.2%)	0.470
Clopidogrel	8 (53.3%)	183 (98.9%)	**<0.001***
Ticagrelor	3 (20%)	15 (8.1%)	0.140
Cilostazol	4 (26.7%)	10 (5.4%)	**0.013***
LMWH	9 (60%)	160 (86.5%)	**0.018***
Warfarin	10 (66.7%)	40 (21.6%)	**<0.001***
Statin	15 (100%)	174 (94.1%)	1.0
ACEI/ARB	11 (73.3%)	151 (81.6%)	0.492
Beta blocker	9 (60%)	169 (91.4%)	**0.001***
CCB	3 (20%)	55 (29.7%)	0.560
Diuretic	15 (100%)	136 (73.5%)	**0.024***

LMWH, low molecular weight heparin; ACEI, angiotensin converting enzyme inhibitor; CCB, calcium channel blocker, ARB, angiotensin receptor blocker; *: statistically substantial as P value <0.05

Table 5: Univariate and multiple regression analysis for prediction of the incidence of thrombus

	Univariate regression analysis				
	Coefficient	**Std. Error**	**95% CI**	**t**	**P**
D dimer	8.006	0.160	7.68 - 8.32	49.79	**<0.001***
EF %	-3.036	0.808	-4.630 - -1.44	-3.75	**0.002***
E/A	0.748	0.077	0.597 - 0.899	9.739	**<0.001***
Declaration time (ms)	-22.46	2.384	-27.16 - -17.76	-9.42	**<0.001***
E/e'	2.414	0.389	1.65 - 3.18	6.196	**<0.001***
E'	-1.103	0.297	-1.688 - -0.518	-3.716	**0.003***
WMSI	0.356	0.040	0.278 - 0.434	8.985	**<0.001***
TIMI	-1.378	0.209	-1.790 - -0.967	-6.609	**<0.001***
	Multiple regression analysis					
	Coefficient	**Std. Error**	**t**	**P**	$r_{partial}$	$r_{semipartial}$
D dimer	0.100	0.003	30.096	**<0.001***	0.908	0.516
EF %	0.0002	0.001	0.174	0.862	0.012	0.003
E/ A	0.093	0.015	6.215	**<0.001***	0.410	0.107
Declaration time (ms)	-0.0004	0.0005	-0.952	0.3421	-0.068	0.016
E/e'	0.006	0.003	1.981	**0.049***	0.141	0.034
E'	0.0004	0.004	0.102	0.919	0.007	0.002
WMSI	0.113	0.030	3.756	**0.002***	0.262	0.064
TIMI	-0.003	0.005	-0.67	0.504	-0.048	0.011

CI: confidence interval, EF: ejection fraction; E/A: early to atrial filling velocity ratio; WMSI: wall motion score index;

DISCUSSION

LVT remains a common finding in the setting of acute STEMI, the frequency of LVT declined from 40% in the prethrombolytic era to 28% in thrombolytic era [16]. PPCI considerably enhanced the clinical results for STEMI patients, with significant death and morbidity reductions. [17]. The main PCI period saw a considerable decrease in LVT thanks to quick reperfusion and new, very effective anti-thrombotic drugs. [18, 19].

The incidence of LVT in our study was 7.5%. Literature showed marked variation of the incidence of LVT reported. Contrary to our results LVT incidence of 0.3% in non-anterior SEMI. Khoury et al. [20] a successful primary PCI for STEMI patients showed a low rate of LVT of 1.25%. Our results were inconstant with You et al. [6] reported LVT of 9.36 % in post-MI patients with LV malfunction underwent PCI. Driesman et al., [23] recorded higher LVT incidence of 15.2% in patients with anterior STEMI with substantial LV malfunction managed with PCI. Our results were inconstant with meta-analysis and systematic review reported incidence of LVT approximately 2.7–9.1% in the contemporary PCI era [8]. LVT incidence was independently correlated with heart rate, LAD illness, severity of heart failure or lower EF, involved anterior apex wall, and non-white race. [21, 22]. Incidence of LVT likely related to lower LV dysfunction selection criteria in these studies [20, 22].

The results of univariate regression analysis suggested that D-dimer, poor EF, E/A ratio, time for deceleration, E/e', E', WMSI and post primary PCI impaired flow less than TIMI 2 flow were significant predictors of LVT. However, multiple regression analysis indicated that only D-dimer, E/A ratio, E/e' and WMSI were significant predictors of LVT.

Our study suggested that successful timely restoration of Blood flow was linked to a reduced rate of LVT, in line of our results Bulluck et al. [24] revealed that reduction of TIMI flow <2 was linked to a higher rate of LVT. In comparison to balloon angioplasty, using either drug-eluting or bare-metal stents was independently linked to a decreased rate of LV thrombus. Several investigations reported that suboptimal revascularization with lower TIMI flow was associated with larger infarct size that is likely related to higher LVT incidence [25, 26]. When compared to angioplasty, stents reduce the risk of reinfarction and target artery revascularization; nevertheless, those with no reflow or inadequate flow cannot utilize stents. [27].

In our investigation, D-Dimer was shown to be a reliable predictor of LVT. You et al. [6] demonstrated that it was possible to predict the incidence of LVT independently using D-dimer levels with a cut-off value of 1.53 mg/L. Higher levels of D-dimer have been previously recorded in patients with systolic heart failure and was connected with worse adverse events [28, 29], [30]. The D-dimer blood level is simpler to measure in a clinical context compared to other predictors, which may enable doctors to take action sooner. Limitations: Our study is a single centre study that may be affected by selection criteria and referral bias, LVT detection by 2D ECHO may underestimate LVT compared with computed tomography (CT) contrast-enhanced echocardiography, or cardiac MRI. Further limitation is the absence of later 2D ECHO follow-up. Remote LVT within 1-3 months after STEMI could be missed [31]. In our next study, a multicentre, randomised controlled research with a bigger sample is anticipated.

CONCLUSIONS

Our research shows that LVT, particularly in anterior STEMI patients, is still a somewhat frequent problem in STEMI patients. The rate of LVT was 15 (8%) of all the study population. Additionally, we verified Poor LV EF is one of the causes of LVT in post-PCI STEMI patients, post primary PCI impaired flow less than TIMI 2 flow, WMSI, E/A ratio, deceleration time, E', E/e' and D-dimer.

monetary assistance and sponsorship: Nil

Conflict of Interest: Nil.

REFERENCES

1. Barbur MCM, Martínez-Ávila MC, Madero AI, Nuñez JFG, Dominguez JCG. Acute ST-segment elevation myocardial infarction: to be or not to be? Arch Clin Cases. 2022; 9:19-23.
2. Hebbal VP, Setty HS, Sathvik CM, Patil V, Sahoo S, Manjunath CN. Acute ST-segment elevation myocardial infarction: The prognostic importance of lead augmented vector right and leads V(7) V(9). J Nat Sci Biol Med. 2017; 8:104-9.
3. Zalewski J, Nowak K, Furczynska P, Zalewska M. Complicating acute myocardial infarction. Current status and unresolved targets for subsequent research. J Clin Med. 2021; 10:125-9.
4. Niazi AK, Kassem H, Shalaby G, Khaled S, Alzahrani MS, Ali HM, et al. incidence and predictors of left ventricular (LV) thrombus after st-elevation myocardial infarction (STEMI) in the Holy Capital of Saudi Arabia. J Saudi Heart Assoc. 2021; 33:101-8.
5. Lechner I, Reindl M, Tiller C, Holzknecht M, Fink P, Plangger J, et al. Association between inflammation and left ventricular thrombus formation following ST-elevation myocardial infarction. Int J Cardiol. 2022; 361:1-6.
6. You J, Wang X, Wu J, Gao L, Wang X, Du P, 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.
7. Khaled S, Jaha N, Shalaby G. Clinical characteristics and short-term outcomes of patients presenting with acute myocardial infarction having multi-vessel disease - a single middle- eastern tertiary-care center experience. Indian Heart J. 2022; 74:28-33.
8. 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.
9. Barbagallo M, Naef D, Köpfli P, Hufschmid U, Niemann T, Gebker R, et al. Right ventricular thrombus, a challenge in imaging diagnostics: a case series. Eur Heart J Case Rep. 2021; 5:340-8.
10. Chaosuwannakit N, Makarawate P. Left ventricular thrombi: Insights from cardiac magnetic resonance imaging. Tomography. 2021; 7:180-8.
11. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). J Am Coll Cardiol. 2018; 72:2231-64.
12. Lee OH, Kim Y, Son NH, Roh JW, Im E, Cho DK, et al. Comparison of distal radial, proximal radial, and femoral access in patients with ST-elevation myocardial infarction. J Clin Med. 2021; 10:25-9.
13. Suleiman S, Coughlan JJ, Arockiam S, Arnous S, Kiernan TJ. Pre-percutaneous coronary intervention TIMI flow grade in STEMI patients treated with pre-hospital ticagrelor loading. Ir Med J. 2022; 115:564-75.
14. Weinsaft JW, Kim J, Medicherla CB, et al. Echocardiographic algorithm for post-myocardial infarction LV thrombus: a gatekeeper for thrombus evaluation by delayed enhancement CMR. J Am Coll Cardiol Img. 2016; 9:505–515.
15. Lashin H, Olusanya O, Smith A, Bhattacharyya S. Left ventricular ejection fraction correlation with stroke volume as estimated by doppler echocardiography in cardiogenic shock: A retrospective observational study. J Cardiothorac Vasc Anesth. 2022; 36:3511-6.
16. Zhang Q, Zhang Z, Jin L, Wang C, Zheng H, Li S, et al. Prognosis and new predictors of early left-ventricular thrombus following ST-elevation myocardial infarction. Int J Gen Med. 2021; 14:8991-9000.
17. Redfors B, Dworeck C, Angerås O, Haraldsson I, Petursson P, Odenstedt J, et al. Prognosis is similar for patients who undergo primary PCI during regular-hours and off-hours: A report from SCAAR. Catheter Cardiovasc Interv. 2018; 91:1240-9.
18. Ram P, Shah M, Sirinvaravong N, Lo KB, Patil S, Patel B, et al. Left ventricular thrombosis in acute anterior myocardial infarction: Evaluation of hospital mortality, thromboembolism, and bleeding. Clin Cardiol. 2018; 41:1289-96.

19. Toufic El Hussein M, Mikhail M. Evidence-based recommendations: Management of left ventricular thrombus post-acute myocardial infarction. Dimens Crit Care Nurs. 2022; 41:313-20.
20. Khoury S, Carmon S, Margolis G, Keren G, Shacham Y. Incidence and outcomes of early left ventricular thrombus following ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. Clin Res Cardiol. 2017; 106:695-701.
21. Gianstefani S, Douiri A, Delithanasis I, Rogers T, Sen A, Kalra S, 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.
22. Garber AM, Mentz RJ, Al-Khalidi HR, Shaw LK, Fiuzat M, O'Connor CM, et al. Clinical predictors and outcomes of patients with left ventricular thrombus following ST-segment elevation myocardial infarction. J Thromb Thrombolysis. 2016; 41:365-73.
23. Driesman A, Hyder O, Lang C, Stockwell P, Poppas A, Abbott JD. 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.
24. Bulluck H, Chan MHH, Paradies V, Yellon RL, Ho HH, Chan MY, et al. Incidence and predictors of left ventricular thrombus by cardiovascular magnetic resonance in acute ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention: a meta-analysis. J Cardiovasc Magn Reson. 2018; 20:72-9.
25. Parsa SA, Nourian S, Safi M, Namazi MH, Saadat H, Vakili H, et al. The association between hematologic indices with TIMI flow in STEMI patients who undergo primary percutaneous coronary intervention. Cardiovasc Hematol Disord Drug Targets. 2022:105-12.
26. Bi X, Wang B, Tse G, Dai C, Chen X, Meng F, et al. Clinical manifestation of cardiac rupture in patients with ST-segment elevation myocardial infarction: early versus late primary percutaneous coronary intervention. Glob Heart. 2022; 17:69-75.
27. Hlinomaz O, Motovska Z, Knot J, Miklik R, Sabbah M, Hromadka M, et al. Stent selection for primary angioplasty and outcomes in the era of potent antiplatelets. Data from the multicenter randomized prague-18 trial. J Clin Med. 2021; 10:50-9.
28. Zorlu A, Yilmaz MB, Yucel H, Bektasoglu G, Refiker Ege M, Tandogan I. Increased d-dimer levels predict cardiovascular mortality in patients with systolic heart failure. J Thromb Thrombolysis. 2012; 33:322-8.
29. Aispuru GR, Clavier MM, Cardone AJ, Gilberto DO, Barousse AP. Thrombotic biomarkers and left ventricle characteristics as short-term predictors of thrombotic events in patients hospitalized for acute decompensated heart failure. Eur J Intern Med. 2012; 23:545-51.
30. Solheim S, Seljeflot I, Lunde K, Bratseth V, Aakhus S, Forfang K, et al. Prothrombotic markers in patients with acute myocardial infarction and left ventricular thrombus formation treated with pci and dual antiplatelet therapy. Thromb J. 2013; 11:15-9.
31. 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 Mar;335(3):171–6.