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ORIGINAL ARTICLE

Pattern of TIMI Flow in Late Arrival STEMI its impact on mortality and Factors that Predict the TIMI ≥ II in Patients with Late Arrival Pattern of TIMI

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Abstract

Background: Acute ST Elevation Myocardial Infarction has high mortality but timely reperfusion is related to improved LV function and improved mortality. Clinical trials demonstrated decreased infarct size and improved mortality in late-arrival STEMI patients. However a significant number of these late arrival patients had TIMI flow ≥II at the baseline. We performed a study to observe the prevalence of Pre-procedure TIMI ≥II flow, its predictors and its impact on In-hospital mortality.

Methodology: It was a two-center observational study conducted at the National Institute of Cardiovascular Disease and its satellite from November 2022 to September 2023. Late-arrival STEMI patients who presented >12 hours after symptoms onset and had undergone cardiac catheterization were included. The baseline TIMI flow in the culprit artery was noted. The patients were followed until discharge.

Results: A total of 305 patients were included in the study. Hypertension was the most prevalent risk factor in 50% (153) followed by diabetes in 34% (104). The prevalence of TIMI ≥II flow was present in 24.6% (75). There was no significant difference for in-hospital mortality between patients with baseline TIMI-0/I or Baseline TIMI-≥II at baseline (7.0% vs. 6.7%). The presence of chest pain within 24 hours (OR: 2.2), and Killip class-II (OR: 2.99) at arrival were predictors associated with TIMI ≥II at baseline angiogram.

Conclusion: Chest pain within 24 hours and killip class-II at presentation were associated with open artery at baseline on angiogram. These factors may be considered for beneficial effect while choosing patients with late arrival STEMI for revascularization.

Keywords

STEMI, Primary PCI, TIMI Flow, Late Arrival

Introduction

Acute ST- segment Elevation Myocardial Infarction (STEMI) is most important and serious condition that has high mortality. Timely reperfusion (<12 hours from onset of pain) is related to salvageable myocardium and improved LV function and improved mortality[1,2]. Primary Percutaneous Coronary Intervention (PCI) is the preferred treatment of choice if done within 12 hours of onset of symptoms. But still up to 40% present Late after 12 hours of symptoms onset[3].

PCI performed beyond 12 hours is still controversial without symptoms. Both European Society of Cardiology (ESC) and American Heart Association (AHA) guidelines recommend PCI in these patients with persistent symptoms, arrhythmias and hemodynamic instability[4]. The Beyond 12 Hours Reperfusion Alternative Evaluation 2 trial (BRAVE-2) trial demonstrated decrease infarct size in patients who underwent PCI between 12- 48 hours of onset of symptoms[5]. However in BRAVE 2 trial 43% of patients had TIMI flow ≥ 2 at the baseline which cannot underestimate the importance of open artery in these patients. Another recent study showed PCI improved survival amongst asymptomatic patients presenting with STEMI Late arrival > 12 hours, again in this study 32.5% had pre PCI TIMI flow of ≥ 2 at the baseline[6].

The assumption that the Infarct Related Artery remains permanently closed with no blood circulation to the subtended myocardium may be incorrect in a high proportion of late presenters with STEMI, especially in improved without persistent symptoms. The instability of culprit artery plaque is a dynamic process there is continuous occlusion and reperfusion of infarct related artery leading to ischemic preconditioning of myocardium[7]. As already mentioned BRAVE-2 trial 43.4% of patients in the invasive therapy group had a Thrombolysis in Myocardial Infarction (TIMI) flow grade of ≥2 in the IRA at the time of intervention, and 29% of patients had some degree of collateral circulation which shows benefit of PCI in these late arrival STEMI patients. A meta-analysis of randomized trials on the value of PCI in patients presenting beyond 12 hours after acute myocardial infarction showed that PCI in these patients is associated with a more favorable pattern of left ventricular remodeling[8] another study also showed TIMI ≥ II in the infarct related artery was associated with smaller infarct size, improved Left ventricular function and better outcome and as compared to with TIMI-0 or TIMI-I flow at baseline[9]. However OAT trial demonstrated that no benefit of primary PCI between 3-28 days of onset and based on this trail current AHA guidelines recommend against PCI after 24 hours of symptoms onset[1, 10]. There is lack of sufficient data to support or negate the baseline TIMI flow on mortality. The objective of the study was to observe the incidence of TIMI ≥ II or greater in the patients with late arrival STEMI arriving beyond 12 hours and factors that can predict the pre PCI TIMI ≥ II. Also to compare In-hospital mortality between two groups. As already mentioned these patients pre PCI TIMI ≥ II gets more benefit from revascularization as shown in studies.

Methodology

The study was descriptive cross sectional observational study performed in NICVD Karachi and its satellite center at Tando Mohammad Khan center after approval from ethical review committee. Sample size was calculated by non-probability consecutive technique with expected TIMI flow of ≥ 2 rates of 32.5% in late arrival STEMI (> 12 hours), 95% confidence level, and 5% margin of error, the required sample size was 305. All patients meeting the criteria of ST segment elevation MI (>1 mm ST segment elevation from J points in two contagious leads and presenting beyond 12 hours of symptoms and undergoing coronary angiography were included in the study. The decision about angiography and its timing were left on discretion of treating physician. Patients having cardiogenic shock were excluded from this study. We collected observational data of 305 patients with late arrival STEMI from November 2022 to august 2023. Both genders were included in the study. All patients were pretreated with 300 mg Aspirin and 600 mg Clopidogrel along with weight based unfractionated heparin before proceeding for coronary angiography. Clinical and demographic

parameters were entered in Performa. Baseline TIMI flow was noted and correlation with different parameters was done. TIMI 0 was defined as no antegrade flow beyond occlusion, TIMI-I was defined when dye pass beyond occlusion but fail to pacify the whole artery, TIMI-II: when the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance from the distal bed (or both) are perceptibly slower than its flow into or clearance from comparable areas not perfused by the previously occluded vessel and TIMI 3 as complete perfusion with normal antegrade flow. Patients were again divided as two groups 1) TIMI 0 or TIMI-I flow at baseline angiogram 2) TIMI-II or TIMI-III flow at baseline angiogram. Extracted data were entered into Statistical Package for the Social Sciences (SPSS) version 25 (IBM Corp., Armonk, NY, USA) and appropriate mean ± standard deviation (SD) or frequency percentages as descriptive statistics were computed for the quantitative and qualitative variables respectively. Their association with different parameters and outcomes were determined with the help of Chi-square test and t-test/Mann-Whitney U test. We have performed univariate and multivariable binary logistic regression analysis to assess the associated factors with TIMI-II or TIMI-III flow. Odds ratio (OR) and 95% confidence interval (CI) were reported and significance criteria was p-value ≤ 0.05.

Results

Baseline clinical, angiographic and characteristics are of enrolled patients are shown in Table 1, mean age was 58.5 years (±10.8), 78% (n-239) were male and 22% (n-66) female. Hypertension was prevalent in 50 % (n-153) of patients followed by diabetes in 34% (n-104) and smoking in 23.3% (71) as a risk factor. Around 80% (225) of late arrival STEMI had history of chest pain of 12 to 48 hours and 20% (80) had > 48 hours history of chest pain. In angiographic observations 53% of patients had LAD as culprit and 66% (203) had TIMI-0, 9% (27) had TIMI-I, 11% (33) had TIMI-II and 14%(42) had TIMI-III in culprit artery at baseline angiogram (Table 1).

Table 1: Baseline and angiographic characteristics of patients

Characteristics		Total
Total (N)		305
Gender	Male	78.4% (239)
	Female	21.6% (66)
Age (years)		58.5 (±10.8)
40 years or below		4.6% (14)
41 to 60 Years		57.4% (175)
61 to 79 years		34.1% (104)
> 80 years		3.9% (12)
Duration of symptom (hours)	12- 24 hours	40.7% (124)
	24- 48 hours	39.6% (121)
	48- 72 hours	13.8% (42)
	>72 hours	5.9% (18)
Co-morbid conditions	Diabetes mellitus	34.1% (104)
	Hypertensive	50.2% (153)
	Smoking	23.3% (71)
	Hyperlipidaemia	4.9% (15)
	Family history of CAD	3.0% (9)
	Chronic kidney disease	3.9% (12)
	Prior MI	3.6% (11)
	Anterior wall MI	51.8% (158)

Type of STEMI	Inferior wall MI	39.7% (121)
	Posterior wall MI	3.6% (11)
	Lateral wall MI	4.9% (15)
Ejection fraction (%)	Ejection fraction not assessed	42.6% (130)
	25 – 35%	12.1% (37)
	35 to 50%	45.2% (138)
Magnitude of ST Elevation	ST elevation < 3mm	56.1% (171)
	ST elevation 3-6 mm	40.7% (124)
	ST elevation > 6mm	3.3% (10)
Access site	Femoral	32.5% (99)
	Radial	67.5% (206)
No Of Vessel with Significant Disease	Left main Stem	5.2% (16)
	Single Vessel Disease	34.8% (106)
	Double vessel Disease	35.4% (108)
	Triple Vessel Disease	29.8% (91)
Culprit artery	LAD	53.4% (163)
	LCX	14.8% (45)
	RCA	31.8% (97)
Killip Class at admission	Killip class –I	77.4% (236)
	Killip class –II	12.5% (38)
	Killip class –III	10.2% (31)
Mechanical ventilation		14.4% (44)
Pre PCI TIMI Flow	TIMI -0	66.6% (203)
	TIMI -1	8.9% (27)
	TIMI -2	10.8% (33)
	TIMI -3	13.8% (42)
Revascularization done		93.8% (286)
Collateral Grade (Rentrope Grade)	grade 0 collaterals	73.4% (224)
	grade 1 collaterals	11.5% (35)
	grade 2 collaterals	12.1% (37)
	grade 3 collaterals	3% (9)
In Hospital mortality		6.9% (21)

* LAD= Left anterior desending artery or its braches LCX: left circumflex artery or its branches, RCA: Right coronary artery or its branches, Rentrope classification: Grade 0= no visible collaterals, Grade 1= the filling of the side branch via collateral vessels without visualization of the epicardial segment Grade 2= the partial filling of epicardial coronary artery, Grade 3= the complete filling of epicardial coronary artery

The prevalence of TIMI-0/I in Late arrival STEMI was 74.6% (230) and of TIMI ≥ II was 24.4% (75). In hospital mortality was 6.9% (21) in all patients; in-hospital mortality among patients with baseline TIMI-0/I flow was 7.0% (5) in patients with baseline TIMI-0/I flow in culprit artery vs. 6.7% (16) with TIMI ≥ II flow in culprit artery. Presence of chest pain within 24 hours (OR: 2.20, p value-0.16) of arrival and Killip class-II (OR: 2.99, p value-0.001) were independent predictors of TIMI ≥ II at baseline on multivariate and univariate analysis (Figure 1). Diagnosis of AWMI was non-significantly associated with baseline TIMI ≥ II (p- 0.16). Magnitude of ST elevation > 3mm at baseline was also associated with baseline TIMI ≥ II (OR: 1.12, p-0.21). There was no association of baseline TIMI ≥ II at baseline with gender, age, smoking status and CKD (Table 2).

Table 2: Association of risk factors with baseline TIMI flow

Variables	Total	TIMI 0 – 1	TIMI 2 – 3	p-value
	N=305	N=230	N=75	
Gender				0.69
Female	66 (21.6%)	51 (22.2%)	15 (20.0%)	
Male	239 (78.4%)	179 (77.8%)	60 (80.0%)	
Age; Mean ± SD	58.5 ± 10.9	58.7 ± 11.3	57.8 ± 9.6	0.53
Diabetes	104 (34.1%)	87 (37.8%)	17 (22.7%)	0.016
Hypertension	153 (50.2%)	121 (52.6%)	32 (42.7%)	0.13
Family history	9 (3.0%)	7 (3.0%)	2 (2.7%)	0.87
Smoking	71 (23.3%)	50 (21.7%)	21 (28.0%)	0.27
Previous mi	11 (3.6%)	8 (3.5%)	3 (4.0%)	0.83
Hyperlipidemia	290 (95.1%)	215 (93.5%)	75 (100.0%)	0.023
Chronic kidney disease	12 (3.9%)	10 (4.3%)	2 (2.7%)	0.52
Total Duration of chest pain				0.28
12-24 hours	124 (40.7%)	95 (41.3%)	29 (38.7%)	
24-48 hours	121 (39.7%)	86 (37.4%)	35 (46.7%)	
48-72 hours	42 (13.8%)	36 (15.7%)	6 (8.0%)	
More than 72 hours	18 (5.9%)	13 (5.7%)	5 (6.7%)	
Chest pain in last 24 hours	276 (90.5%)	205 (89.1%)	71 (94.7%)	0.16
Type of STEMI				0.16
AWMI	158 (51.8%)	111 (48.3%)	47 (62.7%)	
IWMI	121 (39.7%)	99 (43.0%)	22 (29.3%)	
Lateral MI	15 (4.9%)	11 (4.8%)	4 (5.3%)	
PWMI	11 (3.6%)	9 (3.9%)	2 (2.7%)	
Magnitude of Maximum ST elevation				0.21
<3mm	171 (56.1%)	135 (58.7%)	36 (48.0%)	
3-6 mm	124 (40.7%)	87 (37.8%)	37 (49.3%)	
> 6 mm	10 (3.3%)	8 (3.5%)	2 (2.7%)	
Culprit vessel				0.045
LAD	163 (53.4%)	114 (49.6%)	49 (65.3%)	
LCX	45 (14.8%)	35 (15.2%)	10 (13.3%)	
RCA	97 (31.8%)	81 (35.2%)	16 (21.3%)	
Killip class at Presentation				0.001
Killip class –I	236 (77.4%)	183 (79.6%)	53 (70.7%)	
Killip class –II	38 (12.5%)	20 (8.7%)	18 (24.0%)	
Killip class –III	31 (10.2%)	27 (11.7%)	4 (5.3%)	
Heart failure	62 (20.3%)	47 (20.4%)	15 (20.0%)	0.94
Mechanical ventilation	44 (14.4%)	41 (17.8%)	3 (4.0%)	0.003
Access site				0.45
Radial	206 (67.5%)	158 (68.7%)	48 (64.0%)	
Femoral	99 (32.5%)	72 (31.3%)	27 (36.0%)	
Revascularization done	286 (93.8%)	223 (97.0%)	63 (84.0%)	<0.001
Number of diseased vessels				0.85
SVD	106 (34.8%)	78 (33.9%)	28 (37.3%)	

2VD	108 (35.4%)	82 (35.7%)	26 (34.7%)	
3VD	91 (29.8%)	70 (30.4%)	21 (28.0%)	
Significant LMS	16 (5.2%)	11 (4.8%)	5 (6.7%)	0.53
In Hospital mortality	21 (6.9%)	16 (7.0%)	5 (6.7%)	0.93

* AWMI= Anterior wall mi, IWMI=Inferior wall mi, PWMI= Posterior wall mi, Significant LMS= left main stem> 50 % stenosis, SVD= single vessel disease, 2VD= two vessel disease, 3VD= three vessel disease.

TIMI ≥ II was more present in LAD as culprit artery than in patients with LCX or RCA as culprit artery. Presence of diabetes (OR: 0.47 p value-0.016) and Killip class-III (OR: 0.48 p value- 0.001) at presentation were associated with TIMI-0/I in the culprit artery on univariate and multivariate analysis (Figure 1). Presence of hyperlipidemia (p-0.023) was also associated with TIMI-0/I at baseline in the culprit artery.

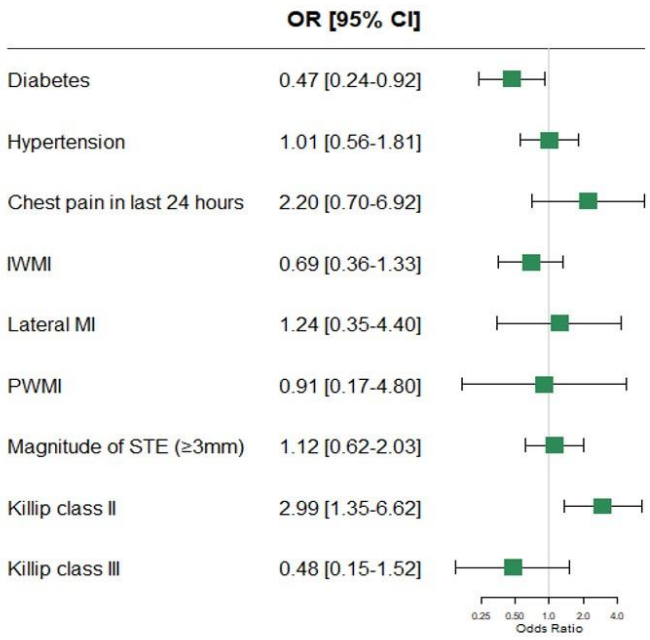

Figure 1: Culprit artery on univariate and multivariate analysis

Discussion

Our study was conducted over a period of 01 year to see the impact of baseline TIMI flow on the in-hospital mortality and factors that may predicts TIMI ≥ II of culprit artery in patients who arrive late after STEMI. We also observed Killip class-II and presence of chest pain as predictors of the baseline TIMI ≥ II in culprit artery. Studies have shown benefit of revascularization in late arrival STEMI presenting after 12 hours of symptoms onset,[6,7] also there it has been proved that there is no benefit in late comers with STEMI and Closed artery[10,11]. Hypothesis has been made for the occurrence of open artery ≥ TIMI-II flow at baseline is the factor responsible for the improvement in survival. Our study found no association of open artery (baseline≥ TIMI-II flow) with in-hospital mortality. To our best knowledge this is the first documentation of association of baseline TIMI flow with the in-hospital survival.

Recent AHA guidelines have recommended routine STEMI PCI between 12- 24 (Class IIa, Level of Evidence: B) and routine ESC guidelines have recommended routine STEMI PCI for Late arrival STEMI between 12-48 hours (Class IIa, Level of Evidence: B). Both guidelines recommended PCI in Late arrival STEMI >12 hours in the presence of

compelling indications like severe heart failure, ongoing ischemia and presence of electrical or hemodynamic instability[12, 13]. A Korean study in late arrival STEMI patients have demonstrated 25% of patients had TIMI-II or TIMI-III at baseline in culprit artery and 6.7% were in killip class IV and in-hospital of mortality of 8%[14] while our study in hospital mortality was lower 6.9% and similarly has TIMI ≥II flow of 24.4% at baseline but killip class IV patients were excluded from our study. A study conducted in the same region in STEMI arriving < 12 hours showed that pre- PCI TIMI ≥ II in the culprit artery was 28% with younger patients and LCX as culprit artery has higher TIMI ≥ II flow at baseline and diabetes and smoking had no effect on prediction of pre PCI TIMI flow,[15] While our study demonstrated diabetes as risk factor for closed culprit artery (TIMI-0/I) at baseline, presence LAD as culprit was associated with TIMI ≥ II at baseline and age has neutral effect on prediction of pre PCI TIMI flow. In another international study on patients with < 12 hours onset of symptoms showed smoking, diabetes, longer delay to PCI and diffuse disease were associated with increase proportion of patients having baseline TIMI-III flow who received GP-IIb/IIa inhibitors[16] while we found no association of smoking with baseline patent artery and as mentioned earlier diabetes as risk factor for closed artery in late comers STEMI patients.

Baseline TIMI ≥ II flow in past studies has also been shown to result in reduction in infarct size and improvement in mortality especially when re-vascularized. Studies have demonstrated reduction in infarct size and up to 39% mortality reduction in patients with baseline TIMI-III flow in the culprit artery[16,17]. In another study shown administration of fibrinolytics as a part of facilitated PCI result in benefit of reduction in mortality and incidence of cardiogenic shock in patient with baseline TIMI-III flow in infarct related artery[18]. However these studies were done the patients with < 12 hours and GP- IIb /IIIa and fibrinolysis were given before randomization, while our study did not demonstrate any impact of baseline TIMI flow on hospital mortality and only oral antiplatelet and heparin were given as part of STEMI protocol.

Presence of Collateral grade has been linked to improved prognosis and improved survival. Grade 2/3 collaterals are associated with myocardial viability, reduced incidence of cardiogenic shock, heart failure and improvement in survival in studies[19-21]. In Brave-2 trial PCI of late arrival STEMI presenting beyond 12 hours had decrease in infarct size although 43% patients had TIMI ≥ II at baseline angiogram 10% of patients had also grade 2/3 collaterals showing importance of collateral grade as well. Our study 18% of patients had grade 2/3 collaterals in patients with baseline TIMI-0/I in the culprit artery which might have impacted the improved survival in these patients.

Conclusion

Chest pain within 24 hours and killip class-II at presentation were associated with open artery or TIMI ≥ II flow at baseline on angiogram and there was no significant impact of baseline TIMI ≥ II flow in culprit artery on in-hospital mortality. Further studies are needed to observe the impact of open artery at baseline on long term mortality.

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References

1) O'gara PT, Kushner FG, Ascheim DD, Casey Jr DE, Chung MK, De Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;127(4):529-55.
2) Nepper-Christensen L, Lønborg J, Høfsten DE, Ahtarovski KA, Bang LE, Helqvist S, et al. Benefit from reperfusion with primary percutaneous coronary intervention beyond 12 hours of symptom duration in patients with ST-segment–elevation myocardial infarction. Circulation: Cardiovasc Interv. 2018;11(9):e006842.
3) Nepper-Christensen L, Lønborg J, Høfsten DE, Sadjadieh G, Schoos MM, Pedersen F, et al. Clinical outcome following late reperfusion with percutaneous coronary intervention in patients with

ST-segment elevation myocardial infarction. Eur Heart J Acute Cardiovasc Care. 2021;10(5):523-31.

4) Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 39(2), pp.119-177.
5) Nepper-Christensen L, Lønborg J, Høfsten DE, Ahtarovski KA, Bang LE, Helqvist S, et al. Benefit from reperfusion with primary percutaneous coronary intervention beyond 12 hours of symptom duration in patients with ST-segment–elevation myocardial infarction. Circulation: Cardiovasc Interv. 2018;11(9):e006842.
6) Bouisset F, Gerbaud E, Bataille V, Coste P, Puymirat E, Belle L, et al. Percutaneous myocardial revascularization in late-presenting patients with STEMI. J Am Coll Cardiol. 2021;78(13):1291-305.
7) Dauerman HL, Ibanez B. The edge of time in acute myocardial infarction. J Am Coll Cardiol. 2021;77(15):1871-4.
8) Abbate A, Biondi-Zoccai GG, Appleton DL, Erne P, Schoenenberger AW, Lipinski MJ, et al. Survival and cardiac remodeling benefits in patients undergoing late percutaneous coronary intervention of the infarct-related artery: evidence from a meta-analysis of randomized controlled trials. J Am Coll Cardiol. 2008;51(9):956-64.
9) Ndrepepa G, Kastrati A, Schwaiger M, Mehilli J, Markwardt C, Dibra A, et al. Relationship between residual blood flow in the infarct-related artery and scintigraphic infarct size, myocardial salvage, and functional recovery in patients with acute myocardial infarction. J Nuclear Med. 2005;46(11):1782-8.
10) Hochman JS, Lamas GA, Buller CE, Dzavik V, Reynolds HR, Abramsky SJ, et al. Coronary intervention for persistent occlusion after myocardial infarction. New Eng J Med. 2006;355(23):2395-407.
11) Hochman JS, Reynolds HR, Džavík V, Buller CE, Ruzyllo W, Sadowski ZP, et al. Long-term effects of percutaneous coronary intervention of the totally occluded infarct-related artery in the subacute phase after myocardial infarction. Circulation. 2011;124(21):2320-8.
12) Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018;39(2):119-77.
13) Writing Committee Members, Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2022;79(2):e21-129.
14) Cho KH, Han X, Ahn JH, Hyun DY, Kim MC, Sim DS, et al. Long-term outcomes of patients with late presentation of ST-segment elevation myocardial infarction. J Am Coll Cardiol. 2021;77(15):1859-70.
15) Hussain M, Kumar R, Ammar A, Alishan S, Muhammad AS, Farooq F, et al. Frequency of Thrombolysis in Myocardial Infarction III Flow in Patients With Primary Percutaneous Coronary Intervention: Not All Culprit Vessels Are Completely Occluded in ST Elevation Myocardial Infarction. Cureus. 2020;12(12)
16) Brener SJ, Mehran R, Brodie BR, Guagliumi G, Witzenbichler B, Cristea E, et al. Predictors and implications of coronary infarct artery patency at initial angiography in patients with acute myocardial infarction (from the CADILLAC and HORIZONS-AMI Trials). Am J Cardiol. 2011;108(7):918-23.
17) Stone GW, Selker HP, Thiele H, Patel MR, Udelson JE, Ohman EM, et al. Relationship between infarct size and outcomes following primary PCI: patient-level analysis from 10 randomized trials. J Am Coll Cardiol. 2016;67(14):1674-83.
18) Schaaf MJ, Mewton N, Rioufol G, Angoulvant D, Cayla G, Delarche N, et al. Pre-PCI angiographic TIMI flow in the culprit coronary artery influences infarct size and microvascular obstruction in STEMI patients. J Cardiol. 2016;67(3):248-53.
19) Meier P, Hemingway H, Lansky AJ, Knapp G, Pitt B, Seiler C. The impact of the coronary collateral circulation on mortality: a meta-analysis. Eur Heart J. 2012;33(5):614-21.
20) Sabia PJ, Powers ER, Ragosta M, Sarembock IJ, Burwell LR, Kaul S. An association between collateral blood flow and myocardial viability in patients with

recent myocardial infarction. New Eng J Med. 1992;327(26):1825-31.

21) Elsman P, Vant Hof AW, De Boer MJ, Hoorntje JC, Suryapranata H, Dambrink JH, et al. Role of collateral circulation in the acute phase of ST-segment-elevation myocardial infarction treated with primary coronary intervention. Eur Heart J. 2004;25(10):854-8.