Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2025.376706.3913 Volume 31, Issue 7 July. 2025
Manuscript ID: ZUMJ-2504-3913
DOI: 10.21608/zumj.2025.376706.3913
ORIGINAL ARTICLE

Clinical and Technical Predictors of No-reflow during Primary Percutaneous Coronary Intervention of Patients with Acute Myocardial Infarction

Mohamed Salah Abdelbasit[1], Mahmoud Abdelaziz[1], Ahmad Aatif Hasan Saqr[2], Mohamed Samy[1*].
[1]Cardiology Department, Faculty of Medicine, Zagazig University, Egypt.
[2]Cardiology Department, Alsalam Hospital, Port Said, Egypt.

Running Title: No-reflow Predictors in STEMI Patients undergoing PCI.

Mohamed Salah Abdelbasit and Mahmoud Abdelaziz contributed equally in the manuscript as first authors

***Corresponding author:**
Mohamed Samy

E-mail:
mohammedsamyabdulaziz87@gmail.com

Submit Date: 19-04-2025
Revise Date 27-04-2025
Accept Date: 01-05-2025

ABSTRACT

Background: No-reflow is a major problem during primary percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI), which is linked to poor myocardial reperfusion and unfavorable outcomes. Unmasking predictors of no-reflow could guide preventive strategies and enhance patient prognosis. We aimed to assess the no-reflow phenomenon's angiographic, clinical and procedural predictors in STEMI patients receiving primary PCI.

Methods: 66 STEMI patients who received primary PCI at Zagazig University Hospitals between November 2022 and May 2023 were included in this cross-sectional analysis. Patients were divided into two groups according to whether no-reflow occurred during PCI. Angiographic information, baseline clinical features, and procedure specifics were documented. Binary logistic regression analysis was used in the statistical study to determine the independent predictors of no-reflow.

Results: No-reflow occurred in 30.3% (n=20) of patients. Baseline troponin levels were higher in the no-reflow group (684.8±181.6 ng/L vs. 531±196.1 ng/L, p=0.004). The absence of pre-stenting TIMI 3 flow (40% vs. 80.4%, p=0.001), greater number of stents (1.5±0.6 vs. 1.1±0.3, p=0.012) and more frequent use of non-compliant (NC) balloons (80% vs. 23.9%, p<0.001) were more associated with no-reflow. The use of NC balloons was the most robust independent predictor (OR 9.91; 95% CI: 1.90–51.84; p=0.007), followed by pre-stenting absent TIMI 3 flow (OR 0.149; 95% CI: 0.030–0.731; p=0.019).

Conclusion: NC balloons' use and lack of TIMI 3 flow before stenting are independent predictors of no-reflow during primary PCI. Careful procedural planning and early risk identification are essential to minimize this complication and optimize reperfusion outcomes.

Keywords: STEMI; primary PCI; no-reflow; TIMI flow; NC balloon.

INTRODUCTION

Acute ST-segment elevation myocardial infarction (STEMI) continues to be a major cause of death from cardiovascular disease globally. The primary treatment to restore blood flow, known as primary percutaneous coronary intervention (PCI), plays a critical role in reperfusion therapy. This approach significantly helps to preserve heart muscle, prevent mechanical complications, and reduce the risk of developing heart failure after the infarction [1].

Despite the effectiveness of primary PCI, it is not without complications. One of the most critical and difficult challenges encountered during the procedure is the no-reflow phenomenon. This condition is linked to higher rates of in-hospital complications, increased incidence of major adverse cardiac events

(MACE) after discharge, and unfavorable long-term clinical outcomes [2].

Previous literature has identified several non-invasive factors that may predict the occurrence of the no-reflow phenomenon. These include the presence of diabetes mellitus, active smoking habits, elevated levels of high-sensitivity C-reactive protein (hs-CRP), and impaired left ventricular systolic function [3].

Despite significant technological progress in PCI and the routine use of pharmacological agents like glycoprotein IIb/IIIa inhibitors, the no-reflow phenomenon continues to occur [4]. This ongoing challenge underscores the urgent need to more accurately identify predictors of no-reflow to improve procedural safety and enhance patient outcomes.

This study aimed to investigate the clinical, angiographic, and technical factors predicting the no-reflow phenomenon among patients of STEMI receiving primary PCI. We hypothesize that specific pre-procedural clinical factors (e.g., diabetes mellitus, and impaired left ventricular function) and procedural characteristics (e.g., thrombus burden, time to reperfusion, and use of specific devices or medications) are independently associated with an increased risk of no-reflow.

METHODS

Ethical approval

The Institutional Review Board of Zagazig University Hospitals in Sharkia, Egypt, granted ethical approval for this study (Approval No: ZU-IRB #9747/11-10-2022). All of the participants gave their written informed consent before being included in the study.

Study design and population

This comparative cross-sectional study was accomplished in the Cardiology Department of Zagazig University Hospitals over a span of seven months, from November 2022 to May 2023. It involved all STEMI patients who obtained primary PCI during this timeframe. Participants were stratified into two groups based upon the occurrence of the no-reflow phenomenon observed during PCI: the no-reflow group and the normal reflow (control) group. Patients who had received thrombolytic therapy prior to admission, those experiencing cardiogenic shock, those presenting beyond the recommended time window for STEMI, or had an aborted myocardial infarction were excluded from the study.

After applying the exclusion criteria, the estimated population over the study duration was 100 patients, As the incidence of noreflow was estimated to be 31.4% in previous studies [5], the sample size was estimated to be at least 62 patients with confidence interval 95% and design effect 0.8 using open EPI-INFO software.

Clinical and Angiographic Data Collection

Detailed clinical information was gathered, encompassing demographic data and cardiovascular risk factors such as age, gender, dyslipidemia, hypertension, diabetes mellitus, peripheral arterial disease and previous stroke. Additionally, parameters related to the acute clinical presentation were documented, including symptom-to-door time, admission blood pressure, Killip classification, initial serum creatinine and troponin levels, as well as the electrocardiographic location of the STEMI.

The *no-reflow* phenomenon was defined as inadequate myocardial perfusion despite successful mechanical opening of the infarct-related artery (IRA), in the absence of angiographic evidence of dissection, spasm, or significant residual stenosis. Specifically, no-reflow was diagnosed when the final Thrombolysis in Myocardial Infarction (TIMI) flow grade was ≤2, with or without myocardial blush grade (MBG) ≤1, despite the restoration of vessel patency [6]. Angiographic assessments were performed by two independent interventional cardiologists who were blinded to the patients' clinical data. In cases of disagreement, a third senior cardiologist adjudicated the final decision.

Further angiographic assessment included identification of the culprit vessel, frequency of balloon inflations, use of aspiration devices, thrombus load, TIMI flow grade before stent

placement, the number of stents deployed, and the application of non-compliant (NC) balloons following stenting.

Statistical Analysis

Frequencies and percentages were used to summarize categorical variables and mean ± SD was used to summarize continuous variables. Depending on the data distribution, the Student's t-test or the Mann-Whitney U test were used to compare groups for continuous data. The Chi-square test was used to compare categorical variables, and when the sample size was too small for the Chi-square test to be reliable, Fisher's Exact test was used. To find predictors for no-reflow, univariate and multivariate logistic regression analyses were performed. Potential multicollinearity among key procedural variables—such as number of stents used and the application of NC balloons—was assessed using the Variance Inflation Factor (VIF). Variables with VIF values exceeding 5 were carefully reviewed to avoid distortion in the regression model. Potential confounders were addressed by including variables with a p-value <0.10 in the univariate analysis into the multivariate model; a p value <0.05 was deemed significant. SPSS V.25.0 was used to analyze the data (IBM Corp., New York, USA).

RESULTS

A total of 66 patients diagnosed with STEMI who underwent primary PCI were prospectively included in the study. Among these, 20 patients (30.3%) developed the no-reflow phenomenon and were assigned to the no-reflow group, while the remaining 46 patients (69.7%) exhibited preserved post-procedural coronary flow and constituted the control group.

Baseline Clinical and Demographic Characteristics

Table 1 summarizes the baseline clinical and demographic parameters of the study cohort. There were no statistically significant differences between the two groups with respect to age, gender, or major cardiovascular risk factors, including hypertension, smoking history, dyslipidemia, prior cerebrovascular events, or peripheral arterial disease. The mean age was 58.4 ± 11.2 years in the no-reflow group and 60.2 ± 9.1 years in the control group. However, patients in the no-reflow arm demonstrated significantly elevated baseline troponin levels in contrast to the control arm (684.8 ± 181.6 ng/L vs. 531 ± 196.1 ng/L, p = 0.004), indicating more extensive myocardial injury. Other clinical indices on presentation, including heart rate, systolic blood pressure, Killip classification, and renal function (serum creatinine), were comparable in the groups.

Angiographic and Procedural Characteristics

As detailed in Table 2, notable discrepancy was observed in several procedural and angiographic variables. The utilization of aspiration catheters was significantly lower in the no-reflow group compared to controls (20% vs. 50%, p = 0.023). Attainment of TIMI grade 3 flow prior to stenting was also markedly less frequent among patients in the no-reflow group (40% vs. 80.4%, p = 0.001). Furthermore, the no-reflow group required a higher average number of stents (1.5 ± 0.6 vs. 1.1 ± 0.3, p = 0.012), and the post-dilatation with NC balloons was significantly more common (80% vs. 23.9%, p < 0.001). A two-stent strategy for bifurcation lesions was also more frequently employed in the no-reflow cohort (15% vs. 2.2%, p = 0.045). Other angiographic features, including the culprit artery, thrombus burden, lesion calcification, and the number of balloon inflations, did not show significant intergroup differences.

Predictors of the No-Reflow Phenomenon

Univariate logistic regression analysis identified several significant predictors of the no-reflow phenomenon, including elevated baseline troponin levels (OR 1.004; 95% CI: 1.001-1.007; p=0.007), failure to achieve pre-stenting TIMI 3 flow (OR 0.162; 95% CI: 0.051-0.514; p=0.002), a greater number of stents deployed (OR 6.367; CI: 1.834-22.108; p=0.004), and use of NC balloons (OR 2.727; 95% CI: 3.510-46.152; p<0.001). In the multivariate logistic regression model,

independent predictors of no-reflow included the use of NC balloons (OR 9.912; 95% CI: 1.895–51.844; p=0.007) and failure to achieve optimal TIMI 3 flow prior to stenting (OR 0.149; 95% CI: 0.030–0.731; p=0.019), **Figure 1**. Baseline troponin levels exhibited a trend for association with the no-reflow phenomenon in the adjusted model (OR 1.004; 95% CI: 0.999-1.009; p=0.090). Furthers details are listed in **Table 3**.

Table 1: baseline characteristics of the study groups

		No-reflow N=20	Control N=46	Test value	P value
Age (years)		58.4 ± 11.2	60.2 ± 9.1	-0.673	0.504
Gender	Male	14 (70%)	23 (50%)	2.264	0.132
	Female	6 (30%)	23 (50%)		
Hypertension		13 (65%)	26 (56.5%)	0.414	0.520
Smoking		12 (60%)	20 (43.5%)	1.523	0.217
Stroke		0 (0.0%)	4 (8.7%)	1.851	0.174
Peripheral artery disease		1 (5%)	0 (0.0%)	2.335	0.126
Dyslipidemia		5 (25%)	16 (34.8%)	0.615	0.433
Onset of chest pain (hours)		5.4 ± 3.3	4 ± 3.6	1.420	0.161
Site of myocardial infarction	Anterior	10 (50%)	28 (60.9%)	4.889	0.087
	Lateral	2 (10%)	0 (0.0%)		
	Inferior	8 (40%)	18 (39.1%)		
Baseline troponin (ng/L)		684.8 ±181.6	531 ± 196.1	2.991	0.004
Systolic blood pressure (mmHg)		131.0 ± 19.2	132.9 ±13.3	-0.388	0.701
Diastolic blood pressure (mmHg)		81.5 ±16.3	79.1 ±13.1	0.625	0.534
Heart rate		83.7 ±15.8	86.5 ±18.3	-0.603	0.549
Killip class		1.1 ± 0.3	1.1 ± 0.3	-0.167	0.868
Baseline creatinine (mg/dl)		1.1 ± 0.2	1.0 ± 0.2	1.873	0.066

Data presented as mean ± standard deviation or number and percentage. N, number.

Table 2: Angiographic and intervention data of the study groups:

		No-reflow N=20	Control N=46	Test value	P value
Culprit vessel	LAD	10 (50%)	28 (60.9%)	4.889	0.087
	LCX	2 (10%)	0 (0.0%)		
	RCA	8 (40%)	18 (39.1%)		
Calcified culprit vessel		5 (25%)	4 (8.7%)	3.146	0.076
Bifurcation culprit lesion		4 (20%)	6 (13%)	0.525	0.469
Number of balloon inflations		1.3 ± 0.5	1.1 ± 0.3	1.882	0.071
Heavy thrombus burden		5 (25%)	18 (39.1%)	1.226	0.268
Use of aspiration catheter		4 (20%)	23 (50%)	5.190	0.023
Achieving TIMI 3 flow before stenting		8 (40%)	37 (80.4%)	10.505	0.001
Number of stents		1.5 ± 0.6	1.1 ± 0.3	2.728	0.012
Use of non-compliant balloon		16 (80%)	11 (23.9%)	18.140	<0.001
Two-stent bifurcation strategy		3 (15%)	1 (2.2%)	4.028	0.045

Data presented as mean ± standard deviation or number and percentage. LAD, left anterior descending coronary; LCX, left circumflex coronary; RCA, right coronary artery; TIMI, Thrombolysis in Myocardial Infarction.

Table 3: Univariate and multivariate regression analysis of the predictors of the no-reflow in the study population

	Univariate			Multivariate		
Variables	**OR**	**95% CI**	**P value**	**OR**	**95% CI**	**P value**
Baseline troponin	1.004	(1.001-1.007)	0.007	1.004	(0.999-1.009)	0.090
Use of aspiration	0.250	(0.72-0.863)	0.028	0.435	(0.085-2.229)	0.318
Achieving TIMI 3 flow before stenting	0.162	(0.051-0.514)	0.002	0.149	(0.030-0.731)	0.019
Number of stents	6.367	(1.834-22.108)	0.004	1.149	(0.202-6.538)	0.875
Use of NC balloon	12.727	(3.510 – 46.152)	<0.001	9.912	(1.895-51.844)	0.007

CI, confidence interval; NC, non-compliant; OR, Odds Ratio; TIMI, Thrombolysis in Myocardial Infarction

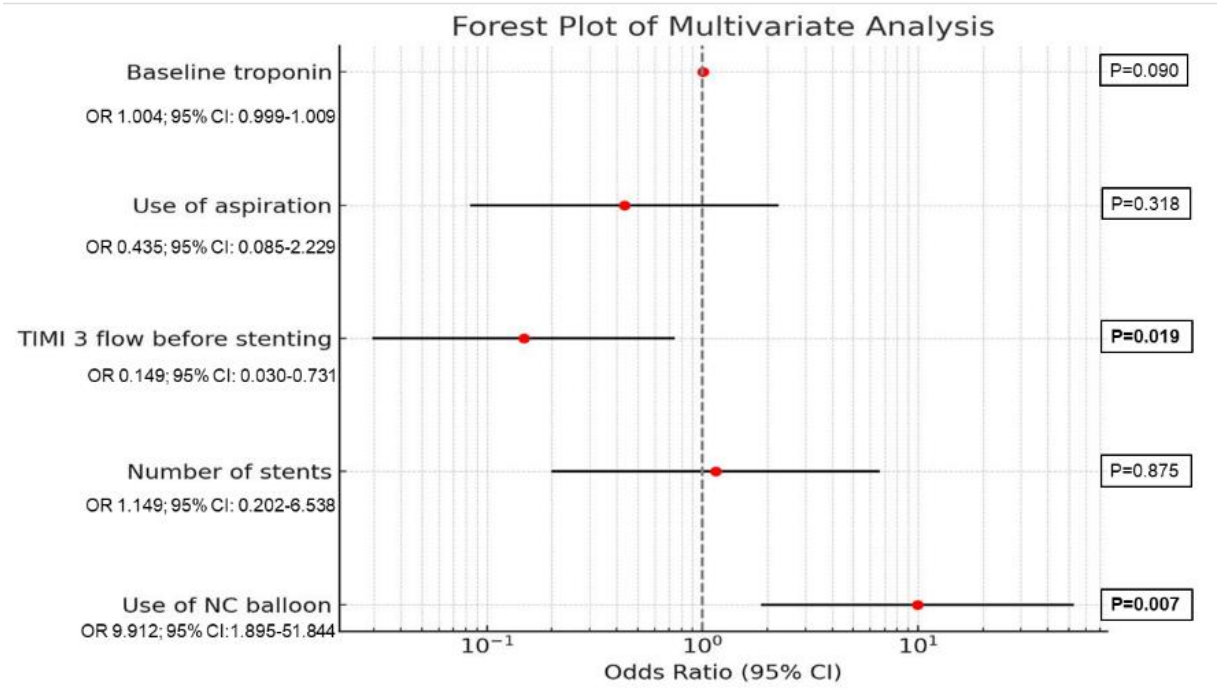

Figure 1. Predictors of no-reflow phenomenon. Forest plot showing multivariate logistic regression results for predictors of the no-reflow phenomenon in STEMI patients undergoing primary PCI. Odds ratios (OR) with 95% confidence intervals (CI) are presented. The vertical dashed line represents the null value (OR = 1).

DISCUSSION

The principal outcomes of this study are:

1. Among STEMI patients adopting primary PCI, elevated baseline troponin concentrations, utilization of NC balloons, and the inability to attain TIMI grade 3 flow prior to stent deployment were significantly associated with the no-reflow phenomenon's existence.
2. Among these, the application of NC balloons was identified as the principal independent predictor of no-reflow, with suboptimal pre-stenting TIMI flow

representing the second most predicting factor.

The no-reflow phenomenon persists as a significant and complex complication for such patients, despite advancements in both interventional and pharmacological approaches. It continues to hinder optimal myocardial reperfusion and is associated with negative clinical outcomes in both the short and long term [2].

Recognizing the clinical, angiographic, and procedural predictors of no-reflow is crucial for risk assessment and improving patient care. Given its prognostic implications, no-reflow has been widely studied [3], and our findings add to the existing body of evidence by identifying important clinical and procedural factors linked to its occurrence.

Clinical Burden

In the current study, the no-reflow phenomenon was observed in 30.3% of STEMI patients performing primary PCI, a rate that is consistent with previously published data. Comparable studies have reported incidences ranging from approximately 25.9% to just over 30%, with some noting rates exceeding 30% following reperfusion therapies such as thrombolysis and primary PCI [5,7]. These findings position our results within the higher end of the reported range, supporting their alignment with current literature.

Predictors

Prior research has demonstrated a relationship between the extent of myocardial necrosis—commonly assessed through cardiac biomarkers like troponin—and the incidence of the no-reflow phenomenon. Elevated baseline troponin levels have been linked to larger infarct size and more severe microvascular dysfunction, thereby predisposing patients to no-reflow [8]. This finding is consistent with our results, which showed significantly higher initial troponin concentrations in the no-reflow group, indicating greater myocardial damage prior to revascularization

The relationship between suboptimal pre-stenting TIMI flow and the no-reflow phenomenon observed in our study aligns with findings from the INFUSE-AMI trial, which identified reduced pre-procedural TIMI flow as an independent predictor of compromised myocardial perfusion following PCI [9]. This association may be attributed to a significant thrombotic burden or distal embolization, both of which can lead to microvascular obstruction—a fundamental mechanism underlying the no-reflow phenomenon.

Interestingly, our study uniquely highlights the use of NC balloons as the strongest independent predictor of no-reflow. While balloon post-dilation is common for optimal stent expansion, aggressive use of NC balloons, especially in thrombus-laden or inflamed plaques, may exacerbate distal embolization and endothelial injury. Zhang et al. similarly found that NC balloon post-dilation was associated with increased microvascular dysfunction in STEMI patients undergoing PCI, suggesting the necessity for more cautious approach when treating high-risk lesions [10].

Recent studies and meta-analyses highlight that the impact of NC balloon use during PCI varies significantly depending on lesion type. While ultra-high-pressure NC balloons have demonstrated high procedural success in predominantly calcified lesions with low complication rates [11], their use in thrombus-rich STEMI lesions is associated with increased risks of no-reflow and microvascular dysfunction. Physiological studies indicate that aggressive NC balloon post-dilation can elevate microvascular resistance and promote distal embolization in these high-risk lesions, contrasting with the benefits seen in calcified plaques. These findings suggest that lesion-specific strategies are essential, challenging the conventional approach of maximal stent expansion and underscoring the need for caution when applying NC balloon techniques in thrombotic settings [12].

Conversely, thrombus aspiration catheter use was more prevalent in the control group. Trials such as TAPAS [13] and EXPIRA [14] supported routine thrombectomy during primary PCI. However, larger studies, including

the TOTAL and TASTE trials [15,16], found no mortality benefit from routine aspiration and raised concerns about increased stroke risk and longer procedural times. Our results suggest that selective use of aspiration in specific patients may still offer advantages, potentially by reducing thrombotic burden and enhancing coronary flow before stenting.

Ultimately, our data indicate no significant association between the lesion location (e.g., LAD vs. RCA), bifurcation involvement, or calcification and no-reflow, which is in partial contrast to earlier reports emphasizing LAD-related infarctions and complex lesions as higher-risk substrates [17]. The lack of statistical difference in these parameters in our cohort may be due to limited sample size or procedural techniques minimizing the influence of lesion anatomy.

Strength points and limitations

The strength points of the current study stem from its focused analysis of both clinical and procedural variables, offering a comprehensive perspective on the predictors of no-reflow in real-world STEMI patients. The prospective nature of data collection and adherence to consistent angiographic assessments could enhance internal validity as well.

Several limitations of this study must be yet considered. The relatively small sample size, particularly within the no-reflow group (n = 20), may have limited the statistical power to identify certain associations. Nevertheless, the overall rate of missing data was low and did not significantly impact the statistical power of the study. The single-center nature of the study may restrict the broader applicability of the findings as well. The variability of operator experience across the cases might have added another limiting factor to our study.

The absence of adjunctive imaging techniques, such as intravascular ultrasound or optical coherence tomography, also limits the ability to further investigate lesion characteristics and procedural mechanisms. Moreover, the lack of biomarker analysis (e.g., hs-CRP or NT-proBNP) prevented additional risk stratification, which could have provided further insights into the clinical implications.

Future directions

Future research should prioritize multicenter, large-scale studies to validate these findings across more diverse patient populations. Furthermore, the no-reflow risk should be studied in the future in the context of lesion complicity and thrombus burden. The incorporation of advanced intracoronary and perfusion imaging may offer deeper insights into the mechanistic interplay between procedural techniques and microvascular injury. Moreover, further investigation into the therapeutic potential of pharmacologic agents—such as adenosine or other vasodilators—in mitigating no-reflow in high-risk patients is warranted. Lastly, procedural strategies should be optimized to achieve effective lesion preparation while minimizing the risk of distal embolization, particularly in cases involving thrombus-rich lesions.

CONCLUSION

No-reflow remains a serious complication during primary PCI of STEMI patients. The application of NC balloons and the lack of pre-stenting TIMI 3 flow, independently predicted the no-reflow. Recognizing and modifying these factors may help reduce no-reflow incidence and improve clinical outcomes.

Conflict of Interest: All authors have no relevant financial or non-financial interests to disclose.

Funding: The authors did not receive support from any organization for the submitted work.

REFERENCES

1. Byrne R A, Rossello X, Coughlan J, Barbato E, Berry C, Chieffo A, et al. 2023 ESC guidelines for the management of acute coronary syndromes: developed by the task force on the management of acute coronary syndromes of the European Society of Cardiology (ESC). European Heart Journal: Acute Cardiovascular Care. 2024; 13(1), pp.55-61.

2. Rao S, Bhardwaj R, Negi PC, Nath RK. No reflow phenomenon in CAD patients after percutaneous coronary intervention: A prospective hospital based observational study. Indian Heart Journal. 2023; 75(2):156-9.

3. Hong YJ, Jeong MH, Choi YH, Ko JS, Lee MG, Kang WY, et al. Predictors of no-reflow after percutaneous coronary intervention for culprit lesion with

plaque rupture in infarct-related artery in patients with acute myocardial infarction. Journal of cardiology. 2009; 54(1):36-44.
4. Pelliccia F, Niccoli G, Zimarino M, Andò G, Porto I, Calabrò P, et al. Pathophysiology and Treatment of the No-Reflow Phenomenon in ST-Segment Elevation Myocardial Infarction: Focus on Low-Dose Fibrinolysis during Primary Percutaneous Intervention. Reviews in Cardiovascular Medicine. 2023; 24(12):365.
5. Nizami AA, Ahmad S, Hanif B. Predictors of No-Reflow in Patients undergoing Primary Percutaneous Coronary Intervention for Acute ST Elevation Myocardial Infarction at Tabba Heart Institute Karachi. Pakistan Journal of Medical & Health Sciences. 2022; 16(07):195.
6. Niccoli G, Burzotta F, Galiuto L, Crea F. Myocardial no-reflow in humans. J Am Coll Cardiol. 2009; 54(6):281–92.
7. Wang JW, Zhou ZQ, Chen YD, Wang CH, Zhu XL. A Risk Score for No Reflow in Patients With ST-Segment Elevation Myocardial Infarction After Primary Percutaneous Coronary Intervention. Clinical cardiology. 2015; 38(4):208-15.
8. Ahmed TA, Sorgdrager BJ, Cannegieter SC, van der Laarse A, Schalij MJ, Jukema W. Pre-infarction angina predicts thrombus burden in patients admitted for ST-segment elevation myocardial infarction. EuroIntervention. 2012; 7(12):1396-405.
9. Brener SJ, Dambrink JH, Maehara A, Chowdhary S, Gershlick AH, Genereux P, et al. Benefits of optimising coronary flow before stenting in primary percutaneous coronary intervention for ST-elevation myocardial infarction: insights from INFUSE-AMI. EuroIntervention. 2014; 9(10):1195-201.
10. Zhang ZJ, Marroquin OC, Stone RA, Weissfeld JL, Mulukutla SR, Selzer F, et al. Differential effects of post-dilation after stent deployment in patients presenting with and without acute myocardial infarction. American heart journal. 2010; 160(5):979-86.
11. Kumar S, Jalli S, Sandoval Y, Alaswad K, Patel NJ, Henry T, Doshi D, Pershad A, Seto A, Kumar G, Burke MN. Systematic Review and Meta-Analysis of the Super High-Pressure Balloon (SIS-OPN) for Percutaneous Coronary Intervention. Catheterization and Cardiovascular Interventions. 2025.
12. Senoz O, Yurdam FS. The effect of postdilatation on coronary blood flow and inhospital mortality after stent implantation in st-segment elevation myocardial infarction patients. Int J Cardiovasc Acad. 2021; 7:132-9.
13. Vlaar PJ, Svilaas T, van der Horst IC, Diercks GF, Fokkema ML, de Smet BJ, et al. Cardiac death and reinfarction after 1 year in the Thrombus Aspiration during Percutaneous coronary intervention in Acute myocardial infarction Study (TAPAS): a 1-year follow-up study. The Lancet. 2008; 371(9628):1915-20.
14. Sardella G, Mancone M, Bucciarelli-Ducci C, Agati L, Scardala R, Carbone I, et al. Thrombus aspiration during primary percutaneous coronary intervention improves myocardial reperfusion and reduces infarct size: the EXPIRA (thrombectomy with export catheter in infarct-related artery during primary percutaneous coronary intervention) prospective, randomized trial. Journal of the American College of Cardiology. 2009; 53(4):309-15.
15. Jolly SS, Cairns JA, Yusuf S, Meeks B, Pogue J, Rokoss MJ, et al. Randomized trial of primary PCI with or without routine manual thrombectomy. New England Journal of Medicine. 2015; 372(15):1389-98.
16. Fröbert O, Lagerqvist B, Olivecrona GK, Omerovic E, Gudnason T, Maeng M, et al. Thrombus aspiration during ST-segment elevation myocardial infarction. New England Journal of Medicine. 2013; 369(17):1587-97.
17. Rezkalla SH, Kloner RA. No-reflow phenomenon. Circulation. 2002; 105(5):656-62.

Citation

Abdelbasit, M., Abdelaziz, M., Saqr, A., Samy, M. Clinical and Technical Predictors of No-reflow during Primary Percutaneous Coronary Intervention of Patients with Acute Myocardial Infarction. Zagazig University Medical Journal, 2025; (2824-2831): -. doi: 10.21608/zumj.2025.376706.3913