



ORIGINAL ARTICLE

The Clinical and Angiographic Outcomes of Routine Post-Dilatation after Primary Percutaneous Coronary Intervention in Patients with Acute ST-elevation Myocardial Infarction

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Submit Date: 12-05-2025

Revise Date: 04-08-2025

Accept Date: 28-10-2025

ABSTRACT

Background: Primary percutaneous coronary intervention (PCI) remains the guideline-recommended reperfusion strategy for patients presenting with acute ST-segment elevation myocardial infarction (STEMI), as it promptly restores blood flow by reopening the infarct-related coronary artery, thereby limiting myocardial damage. This study aimed to evaluate the impact of routine post-dilatation on clinical and angiographic outcomes, including major adverse cardiovascular events (MACE) and final TIMI flow, in STEMI patients undergoing primary PCI.

Methods: This Prospective cohort study included 120 patients were diagnosed with acute STEMI and underwent to PCI they were randomized into two groups: Group I (PDgroup) included 60 patients who underwent post-dilatation after primary PCI. Group II (NPD group) included 60 patients who underwent primary PCI with no post-dilatation.

Results: MACE occurred in 3.3% of patients in the PD group compared to 16.7% in the NPD group, with no reported mortality in either group. Target vessel revascularization (TVR) and acute stent thrombosis occurred in 1.7% and 1.7% of patients in the PD group versus 8.3% and 1.7% in the NPD group, respectively. Although differences in TVR were not statistically significant, the overall incidence of MACE showed a statistically significant reduction in the PD group ($P=0.03$). For secondary outcomes, acute coronary syndrome occurred in 25% of patients in the NPD group compared to 8.3% in the PD group ($P = 0.03$). Angiographic analysis revealed a statistically significant difference in final TIMI flow, with no-reflow (TIMI 0–1) observed in 11.7% of NPD patients compared to 1.7% in the PD group ($P = 0.046$).

Conclusion: The post-dilatation strategy in primary PCI in patients with STEMI tends to reduce MACE as primary outcome, reduce the ACS events after PCI as secondary outcome and reduce the incidence of no reflow/TIMI 0-1 as angiographic outcome.

Keywords: ST-elevation myocardial infarction, TVR, Percutaneous coronary intervention

INTRODUCTION

Acute ST-elevation myocardial infarction (STEMI) is a severe form of transmural myocardial ischemia that results in myocardial injury and necrosis. It occurs due to the acute occlusion of one or more

coronary arteries, which supply oxygenated blood to the myocardium. This occlusion is most commonly caused by the rupture, erosion, or fissuring of an atherosclerotic plaque, or arterial dissection, leading to the formation of a thrombus. Approximately 38%

of hospitalized patients with acute coronary syndrome (ACS) present with STEMI [1].

Primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy for STEMI, as it restores coronary blood flow by reopening the infarct-related artery. Compared to fibrinolytic therapy, primary PCI has been shown to yield superior clinical outcomes [2,3].

However, one of the procedural challenges during PCI is stent under-expansion, which is typically defined as a minimal in-stent diameter less than 80% of the reference vessel diameter. Clinical evidence suggests that stent under-expansion (26%) and malapposition (48%) are major contributors to acute and subacute stent thrombosis. Additionally, malapposition is the leading cause of late and very late stent thrombosis, accounting for approximately 31% of cases [4].

Another significant cause of restenosis is inadequate stent expansion. To ensure optimal stent deployment and full apposition of the stent struts to the arterial wall, post-dilatation is commonly performed using a high-pressure, non-compliant (NC) balloon [5]. This adjunct technique is believed to enhance stent expansion, improve apposition, and potentially reduce restenosis rates. Despite these theoretical benefits, the impact of routine post-dilatation on coronary blood flow and clinical outcomes particularly during primary PCI remains controversial [6].

While some studies have suggested that post-dilatation reduces the need for target vessel revascularization (TVR) [7], others have reported increased risks, including higher mortality rates [8]. As such, the role of post-dilatation in the setting of primary PCI for STEMI patients remains a subject of ongoing debate. This study aimed to evaluate the impact of routine post-dilatation on clinical and angiographic outcomes, including major adverse cardiovascular events (MACE) and final TIMI flow, in STEMI patients undergoing primary PCI.

METHODS

This prospective cohort study included 120 patients who were diagnosed with acute ST-elevation myocardial infarction (STEMI) and underwent primary percutaneous coronary

intervention (PCI). All patients were admitted to the Cardiology Department at Zagazig University Hospitals between January 2024 to January 2025. Patients were randomized into two equal groups: Group I (PD group): 60 patients underwent post-dilatation after primary PCI. The mean age $\pm$ SD was 60.7 ± 7.96 years (range: 40–79 years), with 80% males and 20% females. Group II (NPD group): 60 patients underwent primary PCI without post-dilatation. The mean age $\pm$ SD was 62 ± 7.19 years (range: 42–75 years), with 91.7% males and 8.3% females.

The study was approved by the Ethics Committee of the Faculty of Medicine, Zagazig University (IRB#11420-3/1-2024). Written informed consent was obtained from all participants. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Patients were randomized using a computer-generated randomization list in a 1:1 allocation ratio, utilizing block randomization with a block size of four to ensure balanced group sizes. Allocation concealment was maintained through the use of sealed, opaque envelopes, which were opened only at the time of the procedure by an independent staff member not involved in patient recruitment or outcome assessment.

Angiographic and echocardiographic outcome evaluations were independently performed by two experienced cardiologists who were blinded to the patients' group assignments. This blinding was maintained throughout the analysis phase to minimize observer bias and ensure objectivity in clinical and imaging assessments.

Sample size:

The sample size was determined based on the expected number of eligible cases presenting during the study period. Assuming that all patients meeting the inclusion and exclusion criteria would be recruited over a 6-month period, with an estimated admission rate of 20 STEMI cases undergoing PCI per month, a total of 120 patients were included as a comprehensive sample. These patients were then randomized into two equal groups: Group I (PD group): Post-dilatation was performed following primary PCI. Group II

(NPD group): No post-dilatation was performed after primary PCI.

The inclusion criteria for the study encompassed patients over 18 years of age, regardless of sex, who were admitted with acute STEMI and scheduled to undergo PCI with a stent length of 20 mm or greater, provided they gave written informed consent. The exclusion criteria included individuals with a history of previous CABG, those experiencing cardiogenic shock, patients with a history of bleeding disorders or known coagulopathies, individuals with permanent pacemakers, patients diagnosed with atrial fibrillation, users of anticoagulant medications, and those who declined to participate in the study.

Age, sex, BMI, and related comorbidities (diabetes, hypertension, chronic renal disease, and others) were all meticulously documented, along with any prior ACS, PCI, or family history of coronary artery disease.

Procedure:

Before being sent to the catheterization laboratory for stenting, all patients in the emergency room got the standard care Ibanez et al. [2], which comprised loading doses of atorvastatin (80 mg), clopidogrel (600 mg), and ASA (325 mg). An interventional cardiologist used the usual approach to do PPCI via the radial or femoral arteries. After stent deployment, post-dilatation was performed using an additional non-compliant balloon to achieve the target stent diameter under high-pressure inflation. During the percutaneous coronary intervention (PCI), any occurrence of coronary slow-flow or no-reflow phenomena was managed with intracoronary administration of adenosine and/or nitroprusside, as clinically indicated.

Definitions and Outcomes:

Major adverse cardiovascular events were the main clinical outcome of interest. Acute stent thrombosis, target vessel revascularization (TVR), and mortality were combined to form MACE. A coronary stent thrombotic occlusion was referred to as acute stent thrombosis. All-cause mortality, which included deaths from any cause included both cardiac and non-cardiac fatalities, was the definition of mortality. In the same vessel or

vessels treated during the index PCI, revascularization via PCI or CABG is referred to as TVR [9].

Secondary outcomes included target vessel revascularization and the occurrence of acute coronary syndrome. At least one clinical symptom suggestive of ischemia and an increase or fall in one or more cardiac biomarkers with at least one value over the 99th percentile of the upper reference limit were considered indicators of myocardial infarction: 1) ischemia symptoms; 2) bundle branch block or new substantial abnormalities of the ST-segment or T-wave; 3) pathological ECG Q-waves; 4) imaging revealing altered wall motion or loss of viable cardiac tissue; and 5) Intracoronary thrombus was identified either during coronary angiography or at autopsy. All measured variables adhered to the standards and definitions established by the American College of Cardiology guidelines. Definitions and essential data components for evaluating the clinical care and results of patients with acute coronary syndromes [10].

The angiographic result was the coronary thrombolysis in myocardial infarction (TIMI) flow grade. TIMI flow grades 0, 1, 2, and 3 denoted total occlusion, penetration without perfusion, partial perfusion, and normal perfusion, respectively. In this study, procedural success was classified as TIMI flow Grade-3. The inability to achieve TIMI 3 flow was referred to as slow reflow, while TIMI 0-1 flow was categorized as no reflow.

Statistical analysis

Data were collected, processed, coded, and entered using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Variables with a normal distribution were expressed as mean $\pm$ standard deviation (SD) and analyzed using the independent samples t-test. Variables that were not normally distributed were expressed as median with interquartile range (IQR) and analyzed using the Mann-Whitney U test. The Wilcoxon signed-rank test was used for comparisons between paired groups. Categorical variables were compared using

the Chi-square test or Fisher's exact test when the assumption that "less than 20% of cells have expected counts less than 5" was not met. To adjust for potential confounders such as age, diabetes, hypertension, and other relevant comorbidities, multivariate logistic regression analysis was planned. A p-value of less than 0.05 was considered statistically significant.

RESULTS

This study included 120 patients diagnosed with acute STEMI and underwent PCI; they were divided into two groups: Group (I) (PD group) comprised 60 individuals who had primary PCI followed by post-dilatation. Their mean $\pm$ SD was 60.7 ± 7.96 , and their ages ranged from 40 to 79. There were 20% females and 80% males. Their mean $\pm$ SD was 18 ± 37 , and their BMI varied from 18 to 37 kg/m². 3.3% had a previous ACS, and 20% had a positive family history of ACS. Group (II) (NPD group) comprises 60 individuals who did not receive post-dilatation after first PCI. Their mean $\pm$ SD was 62 ± 7.19 , and their ages varied from 42 to 75 years. There were 8.3% females and 91.7% males. Their mean $\pm$ SD was 25.9 ± 3.01 , and their BMI varied from 18 to 33 kg/m². 8.3% had a previous ACS, and 11.7% had a positive family history of ACS. With respect to demographic data, Table 1 demonstrates that no statistically significant differences were observed between the study groups ($P > 0.05$). Furthermore, there were no notable differences between the groups regarding relevant comorbidities ($P > 0.05$).

Table (2) indicates that there was no statistically significant difference between the two groups in terms of pre- and post-PPCI troponin T levels and ejection fraction (EF) ($P > 0.05$). However, both the PD and NPD groups showed a statistically significant increase in troponin T following PCI ($P < 0.001$ and $P = 0.001$, respectively).

Table (3) shows that, apart from the incidence of MACE (observed in 16.7% of patients in

the NPD group versus 3.3% in the PD group, $P > 0.05$), there were no significant differences between the groups with respect to the primary outcome.

Table (4) demonstrates that there was a statistically significant difference in secondary outcomes between the study groups. In the NPD group, acute coronary syndrome occurred in 25% of patients, compared to 8.3% in the PD group ($P = 0.03$). Furthermore, target vessel revascularization (TVR) was significantly more frequent in the NPD group than in the PD group.

Table 5 highlights the final TIMI flow results, showing a significant difference between groups. The no-reflow phenomenon (TIMI flow grade 0–1) was observed in 1.7% of patients in the PD group versus 11.7% in the NPD group ($P = 0.046$).

Case Demonstration: STEMI with No-Reflow Phenomenon Following Post-Dilatation

This case describes a 49-year-old male with dyslipidemia who presented with typical retrosternal chest pain and vomiting. Upon admission, vital signs were stable, and ECG revealed anterior STEMI. Echocardiography showed borderline LV end-systolic dimensions with mildly impaired ejection fraction (45%), along with akinesia of the apex proper, apical anterior wall and septum, and hypokinesia of the mid anterior septum. Laboratory tests showed elevated troponin I (376 pg/ml) and CK-MB (138 ng/ml). Coronary angiography (Figure 1A) revealed total occlusion of the proximal LAD. Primary PCI was performed using a 3×48 mm Xience Xpedition DES deployed at 14 atm (Figure 1B), achieving TIMI III flow. Post-stenting balloon dilatation was then performed using a 3.5×15 mm Sprinter Legend non-compliant balloon at 14 atm (Figure 1C). However, the patient developed a no-reflow phenomenon post-dilatation (Figure 1D), which was managed successfully with GP IIb/IIIa inhibitor infusion.

Table (1): Demographic Characteristics and Risk Factors among the studied groups

Variables		PD Group (n=60)	NPD Group (n=60)	P. Value
Age (years)	Mean ± SD	60.7 ± 7.96	62 ± 7.19	0.34 ¹
	Range	(40 – 79)	(42 – 75)	
Sex (n. %)	Male	48 (80%)	55 (91.7%)	0.11 ²
	Female	12 (20%)	5 (8.3%)	
BMI (kg/m ²)	Mean ± SD	25.5 ± 4.34	25.9 ± 3.01	0.64 ¹
	Range	(18 – 37)	(18 –33)	
Family history (n. %)	Absent	48 (80%)	53 (88.3%)	0.32 ²
	Present	12 (20%)	7 (11.7%)	
Prior ACS (n. %)	Absent	58 (96.7%)	55 (91.7%)	0.44 ²
	Present	2 (3.3%)	5 (8.3%)	
Previous PCI (n. %)	Absent	60 (100%)	60 (100%)	1.00 ²
	Present	0 (0%)	0 (0%)	
Associated comorbidities				
Diabetes mellitus		17 (28.3%)	15 (25%)	0.68 ¹
Hypertension		23 (38.3%)	19 (31.7%)	0.44 ¹
Chronic kidney disease		6 (10%)	2 (3.3%)	0.27 ²
Hyperlipidemia		20 (33.3%)	16 (26.7%)	0.43 ¹
Smoking		23 (38.3%)	27 (45%)	0.46 ¹

¹Student T-test, ²Fisher exact test, ³Chi-square test, Non-significant: P >0.05, Significant: P ≤0.05

Table (2): Comparison of pre-and post-operative EF and troponin among the studied groups

Variables		PD Group (n=60)	NPD Group (n=60)	P Value
EF (%) pre PPCI	Mean $\pm$ SD	44.3 $\pm$ 6.32	43.1 $\pm$ 4.26	0.23 ¹
	Range	(35 – 70)	(35 – 55)	
EF (%) post PPCI	Mean $\pm$ SD	44.8 $\pm$ 6.15	43.1 $\pm$ 4.26	0.08 ¹
	Range	(35 – 70)	(35 – 55)	
**P value		0.06 ³	1.00 ³	
Troponin T at admission (pre PPCI)	Median (IQR)	320 (260)	285 (268)	0.59 ²
	Range	(126 – 843)	(150 – 1058)	
Troponin T (post PPCI)	Median (IQR)	506 (269)	505 (276)	0.61 ²
	Range	(140 – 4000)	(160 – 10000)	
**P value		<0.001 ⁴	0.001 ⁴	

¹Student T-test, ²Mann-Whitney U test, ³Paired sample T-test, ⁴Wilcoxon signed rank test, Non-significant: P >0.05, Significant: P ≤0.05

*P value=Comparison between the PD & NPD groups, **P value=Comparison within the same group

Table (3): Primary outcome among the studied groups.

Variables (n. %)		PD Group (n=60)	NPD Group (n=60)	P Value
MACE	Absent	58 (96.7%)	50 (83.3%)	0.03
	Present	2 (3.3%)	10 (16.7%)	
Mortality	Absent	60 (0%)	60 (100%)	1.00
	Present	0 (0%)	0 (0%)	
TVR	Absent	59 (98.3%)	55 (91.7%)	0.21
	Present	1 (1.7%)	5 (8.3%)	
Acute stent thrombosis	Absent	59 (98.3%)	55 (91.7%)	0.21
	Present	1 (1.7%)	5 (8.3%)	

*Fisher exact test, Non-significant: $P > 0.05$, Significant: $P \leq 0.05$

Table (4): Secondary outcome among the studied groups.

Variables (n. %)		PD Group (n=60)	NPD Group (n=60)	P Value
ACS	Absent	55 (91.7%)	45 (75%)	0.03
	Present	5 (8.3%)	15 (25%)	
	Unstable angina	3 (5%)	7 (11.7%)	0.12
	NSTEMI	1 (1.7%)	4 (6.7%)	
	STEMI	1 (1.7%)	4 (6.7%)	
TVR	Absent	58 (96.7%)	48 (80%)	0.004
	Present	2 (3.3%)	12 (20%)	

*Fisher exact test, Non-significant: $P > 0.05$, Significant: $P \leq 0.05$

Table (5): Angiographic outcome among the studied groups.

Variables (n. %)		PD Group (n=60)	NPD Group (n=60)	P Value
Final TIMI flow	No reflow and no response to ttt (TIMI 0 - 1)/1	1 (1.7%)	7 (11.7%)	0.046
	No reflow response to ttt (TIMI III)/2	7 (11.7%)	3 (5%)	
	TIMI II/3	0 (0%)	0 (0%)	
	TIMI III without ttt/4	52 (86.7%)	50 (83.3%)	

*Fisher exact test, Non-significant: $P > 0.05$, Significant: $P \leq 0.05$



Figure 1A. RAO caudal view showing proximal LAD total occlusion.



Figure 1B. RAO cranial view showing stenting with final TIMI III flow.



Figure 1C. Post-stenting balloon dilatation with 3.5 x 15 mm NC balloon.



Figure 1D. RAO view showing no-reflow phenomenon post-dilatation

DISCUSSION

In the current study, no statistically significant differences were observed between the post-dilatation (PD) and no post-dilatation (NPD) groups in terms of baseline demographic and clinical characteristics, including age ($P=0.34$), sex ($P=0.11$), body mass index (BMI) ($P=0.64$), family history of coronary artery disease ($P=0.32$), or comorbidities such as diabetes mellitus ($P=0.46$), hypertension ($P=0.44$), hyperlipidemia ($P=0.43$), and smoking status ($P=0.68$). These findings indicate that the randomization process was effective in creating well-balanced groups, reducing the risk of selection bias and enabling a more reliable assessment of the impact of post-dilatation on clinical and angiographic outcomes. As both groups represent patients with the same underlying condition (STEMI), the absence of significant differences in baseline features was expected and desirable, ensuring that observed outcome differences can be attributed to the intervention itself rather than to confounding factors.

Regarding the primary outcome following PCI, the groups being studied differed in a way that was statistically significant. More specifically, severe adverse cardiovascular events occurred in 16.7% of individuals in the NPD group. MACE, compared to 3.3% of patients in the PD group ($P=0.03$). This implies that after PCI, post-dilatation may have a preventive role against significant adverse cardiovascular events. Improved stent expansion and stability may have contributed to the observed decrease in MACE by improving coronary perfusion and lowering restenosis rates.

In a related study, a retrospective cohort analysis carried out in 2022 sought to assess how post-dilatation affected coronary blood flow and MACE in patients with ST-segment elevation both during hospitalization and a year after first PCI. QCA, the corrected TIMI frame count (CTFC), and the frequency of no-reflow/slow-flow at different procedure phases were among the characteristics that were measured in the study. While no statistically significant differences in MACE events were observed during hospitalization,

the post-dilatation group exhibited a lower frequency of target vessel revascularization and target lesion revascularization compared to the non-post-dilatation group during the one-year follow-up. Age, diabetes history, and post-dilatation were found to be independently linked with long-term follow-up of MACE using a multivariable logistic regression model [12].

Conflicting findings have been recorded by certain research, though. For example, a study by Li et al. [8] discovered that although post-dilatation was linked to a lower risk of MACE (OR = 0.67, 95% CI: 0.45–1.00; $P=0.05$), the impact was not statistically significant when patients were categorized according to their STEMI status or length of follow-up. Likewise, a study of Karjalainen et al. [13] discovered that while there was no discernible change in MACE rates between the post-dilatation and non-post-dilatation groups, the post-dilatation group saw lower non-fatal MI rates over the course of long-term follow-up. These differences imply that a number of variables, including as patient characteristics, lesion complexity, and procedural methods, may affect how post-dilatation affects MACE. Our results revealed no significant difference between the groups under study in terms of TVR, which was 1.7% in the PD group and 8.3% in the NPD group ($P=0.21$). Post-dilatation may not have a clear effect on the long-term requirement for revascularization, as evidenced by the lack of a statistically significant difference in TVR rates between the two groups. Although the PD group's incidence of TVR was statistically lower (1.7%) than that of the NPD group (8.3%), the P-value of 0.21 suggests that this difference may be the result of chance rather than a real clinical effect. Factors including lesion characteristics, patient comorbidities, and PCI procedure procedures may have an impact on this finding.

However, after post-dilatation, target vessel revascularisation had dropped by 44%, according to the experiment known as CRUISE (Can Routine Ultrasound Influence Stent Expansion) [14].

On the other hand, Yamaji, et al. [15] discovered that 25 (25.5%) lesions treated

with everolimus-eluting metallic stents (EES) and 48 (50.5%) lesions treated with bioresorbable scaffolds (BRS) had post-dilatation primary percutaneous coronary intervention. Neither the post-procedural minimal lumen diameter nor the baseline parameters of the groups showed any discernible changes. In contrast to the EES group, lesions treated with post-dilatation had a tendency toward a reduced minimal lumen area at 6 months and a considerably higher angiographic late lumen loss in the BRS group. Both the post-dilatation and non-post-dilatation groups had similarly low neointimal healing ratings. Post-dilatation did not lead to better arterial healing or a larger lumen area at follow-up in a subgroup of patients with ST-segment elevation myocardial infarction who received initial PCI with BRS or EES.

Our research showed a statistically significant difference between the groups under examination in terms of the secondary outcome after PCI. Specifically, 25% of patients in the NPD group experienced acute coronary syndrome, compared to 8.3% of patients in the PD group ($P=0.03$). These findings imply that post-dilatation can lower the incidence of acute coronary syndrome by optimising stent expansion and vascular stabilisation. This operation improves coronary perfusion, reduces the likelihood of residual stenosis, and minimises the risk of microvascular dysfunction, which could otherwise lead to ischaemia outcomes.

The few studies that are currently available on post-dilatation primarily concentrate on patients who have calcified lesions, lengthy lesions, or stable coronary artery disease. Acute coronary syndrome (ACS) patients were not included in the majority of these observational studies. According to the results of a new meta-analysis (conference abstract) of seven observational studies involving patients with coronary artery disease, post-dilatation did not lower the incidence of myocardial infarction, MACE, all-cause mortality, or target vessel revascularization. According to the meta-analysis, post-dilatation might help certain patients but not all PCI recipients [16].

In the study by Gao et al. the use of intracoronary vasodilator medications

resulted in reduced immediate TIMI flow and more complex target lesions in the post-dilatation group. However, the final TIMI flow was found to be identical between the two groups. According to subgroup analysis, this strategy could help patients with ACS of any kind, as seen by a decrease in target vessel revascularization within a year after post-dilatation [17].

The angiographic result showed that the groups in our study differed in terms of final TIMI flow in a statistically significant way. Patients in the NPD group had no reflow and no-reflow to treatment (TIMI 0 - 1)/1 in 11.7% of cases, whereas patients in the PD group had this in 1.7% of cases ($P=0.046$). These results demonstrate the potential advantage of post-dilatation in enhancing coronary perfusion through improved stent expansion and decreased microvascular blockage. In order to maintain sufficient coronary blood flow during PCI, post-dilatation may help minimize plaque displacement, optimize stent apposition, and decrease distal embolization.

Unlikely, a study was performed by Soyulu, et al. [18], this comprises patients who presented with STEMI and thereafter had PPCI. Two groups were randomly selected from among the patients: those who had post D done ($n = 62$) or those who did not ($n = 62$). The TIMI frame count (TFC) and TIMI flow were used to assess coronary blood flow. Regarding MI localization, culprit coronary artery, and baseline TIMI flow, there was no difference. The postD group's final adjusted TFC and slow-reflow rate, however, were noticeably greater. This disparity could be explained by variations in lesion characteristics, operative methods, and patient selection. One proposed rationale is that aggressive post-dilatation may elevate the risk of slow reflow by promoting distal embolization or inducing microvascular injury. The observed results may also have been affected by differences in balloon size, inflation pressure, and procedural techniques. Additionally, post-dilatation was found to be an independent predictor of sluggish reflow (OR 11.566, $P=0.014$) by Soyulu et al. [18], indicating that excessive mechanical stress on the artery wall

may occasionally impair microvascular integrity rather than enhance perfusion. These contradictory findings highlight the necessity of a patient-specific strategy for post-dilatation that weighs the risk of microvascular problems against any potential advantages.

However, a retrospective cohort study conducted in 2022 by Xu et al. [12] aimed to assess the effect of post-dilation on coronary blood flow in patients with ST-segment elevation myocardial infarction undergoing primary PCI. The incidence of no-reflow or slow-flow at different procedure stages, final quantitative coronary angiography (QCA), and corrected TIMI frame count (CTFC) were among the characteristics assessed in the study. The results showed that there was no discernible difference between the post-dilation and non-post-dilation groups in the ultimate incidence of no-reflow/slow-flow.

These findings should be cautiously evaluated, though. First off, the operator's wishes and the unique circumstances of each patient will largely determine whether or not post-dilatation method is administered following stent deployment. Secondly, in addition to stent under-expansion, several periprocedural factors such as thrombus aspiration, intracoronary administration of thrombolytic or vasodilator agents, and patient adherence to antiplatelet therapy are associated with PCI-related complications. Furthermore, although multiple studies have reported on final coronary TIMI flow grades following PCI, few have specifically investigated the occurrence of slow or no-reflow phenomena between post-dilation and non-post-dilation groups. Consequently, more randomized controlled trials are necessary to confirm the possible advantages of post-dilation in this particular therapeutic context. According to the CRUISE trial, intravascular ultrasound guiding resulted in a 14% increase in the final minimal stent area following post-dilatation [14].

In our investigation, there was a statistically significant rise in troponin T levels in both the PD and NPD groups ($P < 0.001$ and $P = 0.001$, respectively). This increase is anticipated after PCI because myocardial damage may

result from microvascular blockage, distal thrombotic substance embolization, or mechanical manipulation of the coronary artery. However, the degree of troponin elevation and its association with long-term outcomes determine the clinical importance of this rise. Excessive troponin increase may be a sign of more widespread ischemia damage or microvascular dysfunction, even if some myocardial injury is inevitable during PCI.

Similarly, an investigation conducted by Samir et al. [19] investigated the clinical implications of post-PCI troponin increase. According to the study, elevated troponin levels after PCI may be a proxy for the degree of myocardial damage, depending on a number of procedural and patient-related variables.

Our study uniquely contributes to the evolving evidence on the role of routine post-dilatation in patients with ST-segment elevation myocardial infarction (STEMI), a population often underrepresented in previous research. While many prior studies focused on elective PCI or stable coronary artery disease (CAD) cases, the acute inflammatory and thrombotic milieu in STEMI presents distinct challenges affecting stent deployment and clinical outcomes.

Recent investigations (2022–2024) have highlighted mixed results regarding the efficacy of post-dilatation in improving clinical endpoints in STEMI. For instance, Xu et al. [12] reported no significant difference in no-reflow rates or major adverse cardiac events (MACE) with post-dilatation in STEMI patients, emphasizing the complexity of lesion morphology and microvascular injury in this setting. Conversely, Gao et al. [17] suggested that selective use of intracoronary vasodilators alongside post-dilatation could reduce target vessel revascularization, pointing to potential benefits when tailored to patient characteristics.

Our findings of significantly reduced MACE and improved TIMI flow in the post-dilatation group provide supportive evidence for routine post-dilatation as a beneficial adjunct in STEMI PCI, contributing to better coronary

perfusion and clinical outcomes. This underscores the need for further randomized controlled trials specifically targeting STEMI populations to optimize post-PCI strategies.

Practical Implications:

The findings of this study have important clinical relevance for optimizing PCI outcomes in patients with STEMI. Routine post-dilatation after stent deployment appears to improve stent expansion, reduce microvascular obstruction, and decrease adverse cardiac events such as MACE and acute coronary syndrome.

Limitations:

This study has several limitations that warrant consideration. The relatively small sample size and single-center design may limit the generalizability and statistical power to detect differences in some clinical outcomes. Post-dilatation was performed at the operator's discretion, which could introduce selection bias despite randomization. The absence of routine intravascular imaging (such as IVUS or OCT) restricts detailed assessment of stent expansion and apposition, potentially limiting mechanistic insights. Additionally, the follow-up duration may be insufficient to capture long-term events such as restenosis or late stent thrombosis. Variability in procedural techniques and adjunctive therapies was not standardized, which could affect the consistency of outcomes. Finally, microvascular function was assessed primarily by angiographic parameters without advanced physiological measurements, possibly overlooking subtle differences in microvascular perfusion. Future multicenter randomized studies with larger populations and standardized protocols including intravascular imaging and extended follow-up are needed to confirm these findings.

CONCLUSION

In patients with STEMI, the post-dilatation technique during primary PCI tends to decrease MACE as the major outcome, ACS occurrences following PCI as the secondary outcome, and the incidence of no reflow/TIMI 0-1 as the angiographic outcome.

Conflict of interest: The authors declare no conflict of interest.

Financial Disclosures: This study was not supported by any source of funding.

Sources of funding: No specific grant was obtained for this research from governmental, private, or nonprofit funding organizations.

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Citation

Adel, A., El-Shaer, M., Mahmoud, F., Eldesouky Nashy, B. The Clinical and Angiographic Outcomes of Routine Post-Dilatation after Primary Percutaneous Coronary Intervention in Patients with Acute ST-elevation Myocardial Infarction. *Zagazig University Medical Journal*, 2025; (5697-5708): -. doi: 10.21608/zumj.2025.384310.3946