RESEARCH ARTICLE

DOI: 10.4274/ijca.2025.20982
Int J Cardiovasc Acad 2025;11(3):133-146

Effect of Stent Post Dilatation Versus No Post Dilatation in Patients with STEMI Treated by Primary PCI on No Reflow and in-hospital Outcome

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Abstract

Background and Aim: Percutaneous coronary intervention (PCI) remains the primary modality of choice for achieving reperfusion in patients with acute coronary syndrome. However, complications such as no-reflow phenomenon can occur. Stent post-dilatation (SPD) is common in non-infarct settings, however its use during acute myocardial infarction is controversial. To investigate impact of SPD versus no SPD in ST elevation myocardial infarction (STEMI) cases undergoing primary PCI on incidence of no-reflow phenomenon and in-hospital outcomes.

Materials and Methods: A prospective, single-center, randomized controlled trial was conducted on 300 STEMI cases who presented to Ain Shams University Hospitals between February 2024 and August 2024. Following successful stent implantation, confirmed thrombolysis in myocardial infarction (TIMI) III flow, and adequate stent expansion, cases were randomly stratified into two groups: group I (n=150), which underwent SPD, and group II (n=150), which did not receive SPD. Post-procedural TIMI flow and myocardial blush grade (MBG) were assessed and compared between the two groups. In-hospital clinical outcomes were likewise evaluated.

Results: Cases who underwent SPD (group I) showed significantly lower TIMI flow and MBG than cases in group II; consequently, group I showed elevated prevalence of transient no-reflow (43.3% vs. 4% in group II). There is a relation between SPD and occurrence of no-reflow. In-hospital major adverse cardiac events rates were comparable between two groups, with no substantial variation detected. Longer chest pain duration, higher non-compliant (NC) balloon inflation pressure, higher NC balloon to stent size ratio, and longer stent length were strongly and negatively correlated with TIMI flow and MBG outcomes, leading to poorer post procedural results.

Conclusion: Post-stent dilatation during primary PCI in STEMI patients was associated with a higher incidence of transient no-reflow immediately following the procedure. However, this did not translate into a significant difference in short-term-in-hospital clinical outcomes-likely due to prompt intra-procedural management of no-reflow.

Keywords: STEMI, percutaneous coronary intervention, no-reflow phenomenon, TIMI flow, myocardial blush grade, post-dilatation, in-hospital outcomes

INTRODUCTION

Since the advent of coronary stents, percutaneous coronary intervention (PCI) has become a reliable and effective therapeutic strategy, widely adopted as a primary treatment modality for ST elevation myocardial infarction (STEMI).[1]

Nonetheless, a subset of cases undergoing primary PCI fails to achieve effective reperfusion. The no-reflow phenomenon, first identified in humans by Ito et al.[2] in 1992, refers to the absence of adequate myocardial perfusion despite successful reopening of the epicardial coronary artery.

To cite this article: Gamal S, Shaalan H, Abu Arab TM, Iskandar MM. Effect of stent post dilatation versus no post dilatation in patients with STEMI treated by primary PCI on no reflow and in-hospital outcome. Int J Cardiovasc Acad. 2025;11(3):133-146

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Received: 26.05.2025
Accepted: 07.08.2025
Publication Date: 15.09.2025

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Angiographic no-reflow is characterized by thrombolysis in myocardial infarction (TIMI) flow less than 3, or TIMI 3 flow with myocardial blush grade (MBG) 0 or 1 without mechanical obstruction. The development of the no-reflow phenomenon following primary PCI in STEMI patients represents a critical indicator of unfavorable prognosis.[3]

Stent mal-apposition and under-expansion are thought to be major factors contributing to adverse clinical outcomes caused by stent thrombosis and in-stent restenosis in the drug-eluting stent (DES) era.[4]

The role of stent post-dilatation (SPD) during primary PCI in STEMI remains controversial. While SPD is traditionally performed to ensure optimal stent expansion and apposition, concerns have emerged regarding its potential to provoke distal embolization, microvascular obstruction, or mechanical injury, potentially leading to the no-reflow phenomenon and adverse outcomes.[5]

Given this ongoing debate, our study aimed to address the following research question:

"Does SPD during primary PCI in STEMI patients increase the incidence of the no-reflow phenomenon and influence the in-hospital clinical outcomes compared to no post-dilatation?"

We hypothesized that SPD, although intended to optimize stent deployment, may paradoxically increase the risk of no-reflow due to microvascular compromise or embolization in the highly thrombotic setting of STEMI. This study sought to prospectively evaluate the impact of SPD versus no-SPD on no-reflow and in-hospital clinical outcomes.

METHODS

This study is a prospective, single-center, randomized controlled trial, which included 300 STEMI cases presented to emergency departments of Ain Shams University Hospitals, for the period from February 2024 to August 2024 where all cases were randomly divided after successful primary PCI with stent implantation, confirmed TIMI III flow, and adequate stent expansion, into 2 groups in a 1:1 ratio. Group I included 150 cases who underwent SPD; group II included 150 cases who did not undergo SPD. Patients with stent under-expansion of 20% or more or TIMI flow less than III were excluded from the study, prior to randomization (Figure 1).

The sample size was calculated, based on prior data suggesting that there is a potential difference of approximately 20% in no-reflow incidence between groups. With an alpha error of 0.05 and power of 80%, a minimum of 135 patients per group was required. To account for potential dropouts, 150 patients were enrolled in each arm.

The study protocol was approved by Ain Shams University Faculty of Medicine Scientific and Ethical Committee (protocol no: FMASU MS82/2024, date: 06.02.2024). Written informed consent was secured from all participants, with strict adherence to ethical standards ensuring protection of privacy and confidentiality of personal data.

Cases were eligible for inclusion if they presented with STEMI within 12 hours of symptom onset, meeting established criteria for primary PCI [symptoms indicative of myocardial ischemia, such as persistent chest pain, accompanied by electrocardiography (ECG) changes consistent with STEMI or its equivalents]. Additional inclusion criteria required that cases have undergone primary PCI with a successfully placed angiographic stent, defined by achieving less than 20% residual stenosis and restoration of TIMI grade III flow. Cases were excluded if they presented more than 12 hours after symptom onset, received fibrinolytic therapy, did not undergo stent implantation, required bifurcation stenting, or presented with cardiogenic shock. Additional exclusion criteria included severe stent under-expansion necessitating urgent post-dilatation, a heavy thrombus burden (mainly grade 4 with thrombus size double vessel diameter), requirement for aggressive pre-dilatation with large non-compliant balloons (>2.5 mm), or enrollment in other study protocols targeting no-reflow. These criteria were designed to ensure a homogeneous study population for evaluating the specific effect of SPD. However, it is important to note that these exclusions inherently limit the generalizability of our findings to more complex STEMI patients. Relevant clinical data on cases were collected and tabulated.

All enrolled cases were managed according to a standardized protocol. The protocol was initiated by obtaining written informed consent. This was followed by thorough history taking, physical examination, and acquisition of a 12-lead ECG within 10 minutes of presentation, and within 10 minutes after intervention. Laboratory assessments included blood glucose, complete blood count, lipid profile, renal function tests, and cardiac biomarkers. Antiplatelet therapy comprised an oral loading dose of 300 mg acetylsalicylic acid, along with 180 mg ticagrelor; alternatively, 600 mg clopidogrel was administered in cases where ticagrelor was contraindicated or unavailable.

Diagnostic coronary angiography was done using radial or femoral access, and the culprit vessel was identified. TIMI thrombus score and TIMI flow grade were recorded, and lesion was classified according to American College of Cardiology / American Heart Association classification.[6] Primary PCIs were done according to the recommendations of current European Society of Cardiology guidelines. DESs were utilized in all primary PCI procedures, with stent dimensions and placement sites selected according to the operator's clinical decision.

Unfractionated heparin was administered intra-procedurally at a dose ranging from 70 to 100 IU/kg. In cases exhibiting a substantial thrombus burden, the use of manual thrombus aspiration and bailout glycoprotein (GP) IIb/IIIa inhibitors was determined based on the operator's clinical decision. Pre-dilatation might be done if needed using a small balloon under low atmospheric pressure.

Following stent placement, clear stent imaging (Philips Stent Boost, GE Stent Viz) was used in all cases to evaluate stent expansion. Cases that needed urgent post-stent dilatation (stent under-expansion 20% or more), were excluded. TIMI flow was assessed and cases with TIMI flow less than III were excluded from the study.

Subsequently, patients who achieved TIMI III flow and demonstrated adequate stent expansion, defined as stent under-expansion of less than 20% confirmed by clear stent imaging, were randomized into two groups. Group I consists of cases who underwent SPD. Group II includes cases who didn't undergo SPD, using a random sequence generated via the Rand function in Excel 2023, created prior to study initiation. Allocation was concealed using sealed opaque envelopes, which were sequentially numbered and opened only after TIMI III flow was confirmed. This ensured that treatment assignment was not influenced by procedural characteristics or operator preference prior to randomization.

Randomization was intentionally performed after achieving TIMI III flow and confirming adequate stent expansion to ensure that the impact of SPD would be evaluated specifically on microvascular perfusion and myocardial tissue-level outcomes. This approach was chosen to eliminate confounding factors related to epicardial flow restoration and mechanical stent optimization, thereby isolating the effects of SPD on TIMI flow and MBG.

Post-dilatation was performed at 14-18 atmospheric pressure, with an NC balloon of a size 0.25-0.5 mm larger in diameter than stent size, based on the operator's assessment of the proximal and distal reference vessel diameters and visual evaluation of stent apposition. The balloon length typically matched the stented segment to ensure uniform expansion. Immediately after SPD, in the first group TIMI flow and MBG were reassessed. In cases where no reflow was detected post-SPD, operators employed adjunctive pharmacologic or mechanical maneuvers to restore flow. These included intracoronary vasodilators (adenosine, verapamil), aspiration thrombectomy when embolization was suspected, low-pressure ballooning, and the administration of GP IIb/IIIa inhibitors. The choice and combination of these therapies was applied according to operator discretion.

All procedures were performed by a pool of four experienced interventional cardiologists, each with extensive expertise in primary PCI. Operators followed a standardized procedural protocol for both PCI and SPD, including guidance on balloon sizing and inflation pressures. Despite this standardization, inter-operator variability in procedural technique cannot be entirely excluded.

Due to the procedural nature of SPD, operator blinding was not possible. However, post-procedural TIMI flow and MBG were independently assessed by two experienced interventional cardiologists who were blinded to the treatment allocation. In cases of discrepancy between the two assessors, a third senior cardiologist, also blinded to group assignment, provided adjudication to reach consensus.

No-reflow was defined as either a TIMI flow grade of less than III, or a TIMI III flow accompanied by a MBG of 0 or 1, in the absence of angiographic evidence of vessel dissection, distal embolic cutoff, or mechanical obstruction.[1]

The primary angiographic endpoint was occurrence of no-reflow, assessed immediately after SPD in the intervention group or at the end of the procedure in the no-SPD group.

All cases were admitted to coronary care unit and were kept on dual antiplatelet therapy and anti-ischemic medications. The cases' vital data, ECG, and cardiac enzymes were followed up. An echocardiogram was performed on all cases.

The duration of hospital stay was calculated. Adverse events such as heart failure (HF), occurrence of ventricular aneurysms, ST re-elevation, cardiac death, and non-cardiac death were counted.

The primary clinical endpoint was in-hospital major adverse cardiovascular events (MACE), defined as a composite of cardiac death, re-infarction, target vessel revascularization (TVR), stroke, cardiogenic shock requiring inotropic support, and life-threatening ventricular arrhythmias.

The secondary endpoints included the incidence of post-PCI chest pain, defined as recurrent chest discomfort >15 minutes within 24 hours post-PCI, impaired left ventricular (LV) systolic function, mechanical complications, LV thrombus, major bleeding, prolonged hospital stay.

The data analysis followed an intention-to-treat principle, where all randomized patients were analyzed according to their assigned group, regardless of any procedural deviations or post-randomization events.

Statistical Analysis

Data were collected, revised, coded, and entered into the Statistical Package for the Social Sciences (IBM SPSS) version 27 IBM Corp. was released in 2020. IBM SPSS Statistics for Windows,

Version 27.0. Armonk, NY: IBM Corp. The quantitative data were presented as means, standard deviations, and ranges. Also, qualitative variables were presented as numbers and percentages. The one-sample Kolmogorov-Smirnov test can be used to test whether a variable is normally distributed. The comparison between groups regarding qualitative data was done using the chi-square test. The comparison between two independent groups with quantitative data was done using an independent t-test. Spearman correlation coefficients were used to assess the correlation between two quantitative parameters in the same group. Univariate and multivariate logistic regression analysis were used to assess predictors of no reflow. A *P*-value of <0.05 was considered statistically significant. Where applicable, 95% confidence intervals (CI) were calculated to assess the precision of the estimated effects.

RESULTS

This study encompassed 300 patients with STEMI, all of whom were treated with primary PCI involving stenting of the culprit vessel. Patients who achieved TIMI grade III flow were subsequently randomized into two groups: group I (n=150), who underwent SPD, and group II (n=150), who did not undergo post-dilatation.

No substantial variations were detected between two groups in terms of demographic characteristics, cardiovascular risk factors, clinical presentation, angiographic findings, or interventional data as shown in Tables 1 and 2.

As shown in Table 3, comparison of TIMI flow and MBG between two groups revealed that patients in group I, who underwent SPD, had significantly lower TIMI flow and MBG, with a higher incidence of no-reflow immediately following SPD (22% vs. 0% for TIMI flow less than III and 43.3% vs. 4% for MBG less than II, as shown in Figure 2a and 2b). This indicates a significant relation between SPD and no-reflow occurrence, as shown in Figure 3. However, the majority of these instances were transient, with successful restoration of TIMI III flow following vasodilators, GP IIb/IIIa, or additional ballooning maneuvers. Final angiographic flow was comparable between groups, despite the higher transient no-reflow rate in the SPD group. Group I experienced a higher incidence of post-PCI chest pain, lasting over 15 minutes, within the first 24 hours (14.6% compared to 5.3% in group II). Among those in group I, eight patients (36.3%) showed transient ECG changes such as ST-segment shifts or T wave inversions, while this was observed in only two patients (25%) in group II. An elevation in cardiac troponin levels indicating myocardial injury was

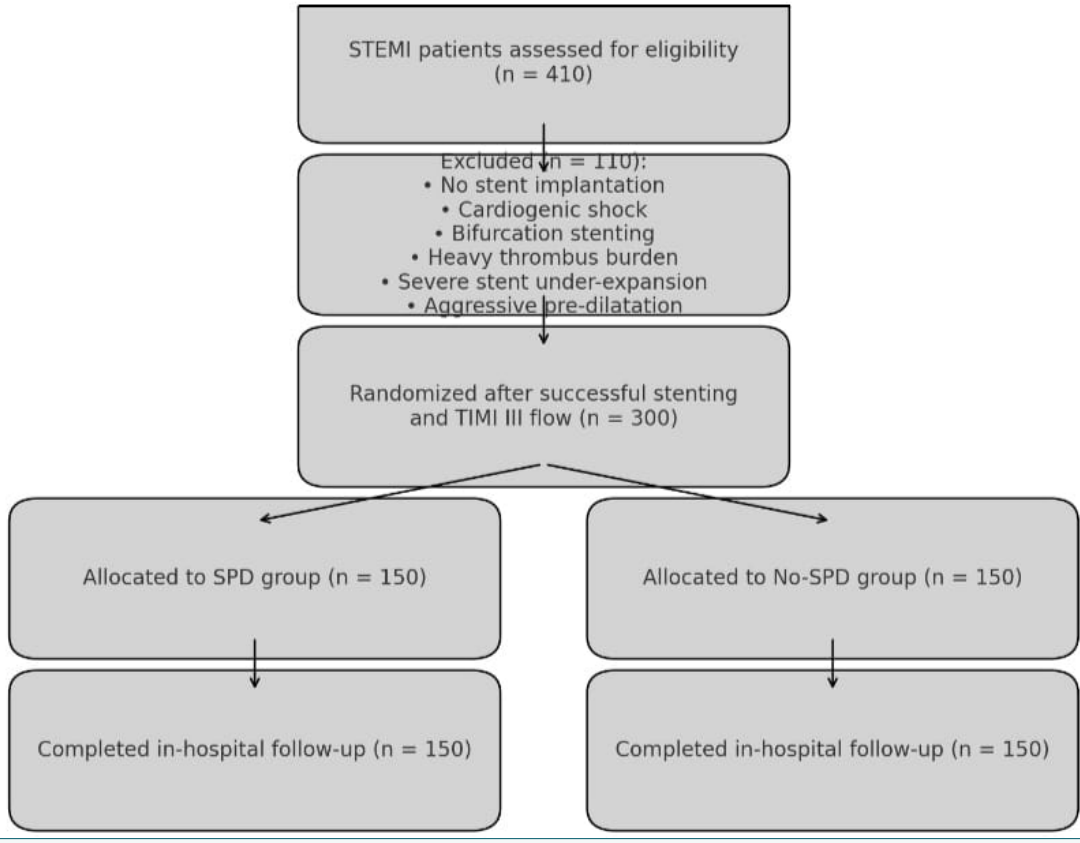


Figure 1: CONSORT flow diagram illustrating patient enrollment, exclusion, randomization, allocation, and follow-up
STEMI: ST elevation myocardial infarction, TIMI: Thrombolysis in myocardial infarction SPD: Stent post-dilatation

Table 1: Comparison between group I and group II regarding demographic data, risk factors and presentation of the studied patients

No. =150		Group I No. =150	Group II	Test value	*P*-value	Sig.
Demographic data						
Gender	Female	35 (23.3%)	33 (22.0%)	0.076*	0.783	NS
	Male	115 (76.7%)	117 (78.0%)			
Age	Mean ± SD	54.61±10.43	55.95±10.43	-1.118•	0.265	NS
	Range	23-77	28-76			
Risk factors						
Diabetes mellitus		69 (46.0%)	60 (40.0%)	1.102*	0.294	NS
Hypertension		70 (46.7%)	62 (41.3%)	0.866*	0.352	NS
Smoking		95 (63.3%)	98 (65.3%)	0.131*	0.718	NS
Dyslipidemia		110 (73.3%)	103 (68.7%)	0.793*	0.373	NS
Family history		65 (43.3%)	53 (35.3%)	2.012*	0.156	NS
CKD		13 (8.7%)	15 (10.0%)	0.158*	0.691	NS
Presentation						
ECG	Anterior STEMI	71 (47.3%)	75 (50.0%)	10.176*	0.179	NS
	Infroposterior STEMI	23 (15.3%)	27 (18.0%)			
	Infroposterolateral STEMI	3 (2.0%)	2 (1.3%)			
	Inferior STEMI	43 (28.7%)	38 (25.3%)			
	Lateral STEMI	0 (0.0%)	4 (2.7%)			
	Anterolateral STEMI	6 (4.0%)	1 (0.7%)			
	Posterior STEMI	0 (0.0%)	1 (0.7%)			
	Inferolateral STEMI	4 (2.7%)	2 (1.3%)			
Killip class	I	135 (90.0%)	145 (96.7%)	5.595*	0.061	NS
	II	11 (7.3%)	3 (2.0%)			
	III	4 (2.7%)	2 (1.3%)			
	IV	0	0			
Preloading	Ticagrelor	135 (90.0%)	130 (86.7%)	0.809*	0.369	NS
	Clopidogrel	15 (10.0%)	20 (13.3%)			
Chest pian duation (hours)	Mean ± SD	6.6±2.68	6.49±2.68	0.345•	0.731	NS
	Range	1-12	1-12			
Time to wire crossing (minutes)	Mean ± SD	34.67±10.03	35.57±10.34	-0.765•	0.445	NS
	Range	20-60	20-60			

P-value >0.05: Non significant, *P*-value <0.05: Significant, *P*-value <0.01: Highly significant

*: Chi-square test, •: Independent t-test, SD: Standard deviation, NS: Non-significant, CKD: Chronic kidney disease, STEMI: ST elevation myocardial infarction, ECG: Electrocardiography

found in 5 patients with chest pain (22.7%) in group I and in 1 patient (12.5%) in group II. However, none of these cases fulfilled the criteria for reinfarction. The in-hospital clinical outcomes were largely comparable between the SPD and no-SPD groups. Mortality occurred in 2 patients (1.3%) in the SPD group versus 1 patient (0.6%) in the no-SPD group, a difference that was not statistically significant ($P = 0.330$). Reinfarction, urgent revascularization, and cerebrovascular stroke (CVS) did not occur in either group (0%). The incidence of cardiogenic shock requiring inotropic support was slightly higher in the SPD group (2.7%) compared to the no-SPD group (1.3%), though this difference was not statistically significant ($P = 0.410$). Similarly, ventricular tachycardia was noted in 2.0% of SPD cases and 1.3% of no-SPD cases ($P = 0.652$), and major bleeding was rare and not significantly different (0.7% vs. 0%, $P = 0.317$). Group I had a longer hospital stay (median 2 (2-4) days) compared to Group II (median 2 (1.5-3) days).

The analysis reveals that higher pressure during NC balloon inflation and a larger difference between NC balloon and stent

Table 2: Comparison between group I and group II regarding angiographic and interventional data of the studied patients

Angiographic data						
Approach	Femoral	147 (98.0%)	150 (100.0%)	3.030*	0.082	NS
	Radial	3 (2.0%)	0 (0.0%)			
Culprit lesion	LAD	77 (51.3%)	75 (50.0%)	8.378*	0.079	NS
	LCX	8 (5.3%)	15 (10.0%)			
	RCA	59 (39.3%)	54 (36%)			
	Diagonal	0 (0.0%)	4 (2.7%)			
	OM	6 (4.0%)	2 (1.3%)			
Culprit lesion site	Proximal	76 (51.4%)	89 (59.8%)	7.232*	0.204	NS
	Distal	7 (4.7%)	11 (7.4%)			
	Mid	62 (41.9%)	49 (32.9%)			
	Ostial	3 (2.0%)	0 (0.0%)			
Type of lesion	A	26 (17.3%)	27 (18.0%)	0.789*	0.674	NS
	B	34 (22.7%)	40 (26.7%)			
	C	90 (60.0%)	83 (55.3%)			
Vessel diameter (QCA)	Mean ± SD	3.19±0.34	3.2±0.42	-0.218•	0.828	NS
	Range	2.25-4.5	2-5			
Lesion length	Short (<20 mm)	60 (40.0%)	67 (44.7%)	0.669*	0.413	NS
	Long (>20 mm)	90 (60.0%)	83 (55.3%)			
TIMI flow (pre)	0	70 (46.7%)	76 (50.7%)	3.078*	0.380	NS
	I	24 (16.0%)	14 (9.3%)			
	II	38 (25.3%)	42 (28.0%)			
	III	18 (12.0%)	18 (12.0%)			
Thrombus grade	0	4 (2.7%)	0 (0.0%)	6.025*	0.110	NS
	I	62 (41.3%)	53 (35.3%)			
	II	42 (28.0%)	44 (29.3%)			
	III	42 (28.0%)	53 (35.3%)			
Interventional data						
Thrombus aspiration	Yes	0 (0.0%)	2 (1.3%)	2.013*	0.156	NS
GP IIb/IIIa	Yes	44 (29.3%)	49 (32.7%)	0.390*	0.533	NS
Pre-dilatation	Yes	84 (56.0%)	77 (51.3%)	0.657*	0.418	NS
Pre-dilatation balloon size	Mean ± SD	2.131±0.281	2.173±0.308	0.913•	0.362	NS
	Range	1.5-2.5	1.5-2.5			
Pressure of pre-dilatation	Mean ± SD	17.62±1.8	17.41±2.31	0.658•	0.511	NS
	Range	12-20	10-20			
Stent	Promus	42 (28.0%)	38 (25.3%)	2.352*	0.309	NS
	Xience	71 (47.3%)	63 (42.0%)			
	Onyx	37 (24.7%)	49 (32.7%)			
Stent diameter	Mean ± SD	3.157±0.357	3.197±0.410	0.901•	0.368	NS
	Range	2.25-4	2.25 -5			
Stent length	Mean ± SD	30.373±5.654	29.127±6.222	1.816•	0.070	NS
	Range	18-38	12-38			
Stent pressure	Mean ± SD	13.59±1.68	13.55±1.48	0.183•	0.855	NS
	Range	11-18	11-16			
NC balloon length	Mean ± SD	14.627±2.646	-	-	-	-
	Range	8-20	-			
NC balloon pressure	Mean ± SD	16.43±1.27	-	-	-	-
	Range	14-18	-			
Difference between NC & stent size	Mean ± SD	0.39±0.12	-	-	-	-
	Range	0.25-0.5	-			

P-value >0.05: Non significant, *P*-value < 0.05: Significant, *P*-value < 0.01: Highly significant

*: Chi-square test; •: Independent t-test

TIMI: Thrombolysis in myocardial infarction, LAD: left anterior descending artery, LCX: Left circumflex artery, RCA: Right coronary artery, OM: Obtuse marginal artery, QCA: Quantitative coronary angiography, GP: Glycoprotein, NC: Non-compliant, SD: Standard deviation, NS: Non-significant, GP: Glycoprotein

Table 3: Comparison between group I and group II regarding post-procedural results and in-hospital outcome of the studied patients

No. =150		Group I No. =150	Group II	Test value	*P*-value	Sig.
Results						
TIMI flow post	0	0 (0.0%)	0 (0.0%)	37.079*	0.000	HS
	I	4 (2.7%)	0 (0.0%)			
	II	29 (19.3%)	0 (0.0%)			
	III	117 (78.0%)	150 (100.0%)			
MBG	0	42 (28.0%)	0 (0.0%)	76.058*	0.000	HS
	I	23 (15.3%)	6 (4.0%)			
	II	27 (18.0%)	21 (14.0%)			
	III	58 (38.7%)	123 (82.0%)			
No reflow (TIMI flow < III or TIMI III flow with MBG grade of 0, 1).		65 (43.3%)	6 (4%)	64.201*	0.000	HS
Systolic BP	Mean ± SD	117.8±15.1	115.4±14.87	1.387•	0.166	NS
	Range	90-160	90-150			
Diastolic BP	Mean ± SD	73.93±8.74	73.87±10.54	0.060•	0.952	NS
	Range	60-100	60-100			
Heart rate	Mean ± SD	76.21±12.3	80.61±12.27	-3.101•	0.002	HS
	Range	45-110	50-110			
Post PCI chest pain		22 (14.6%)	8 (5.3%)	2.73	0.0064	HS
Echocardiography						
EF %	Mean ± SD	42.34±7.77	42.42±8.33	-0.086•	0.932	NS
	Range	30-66	29-65			
LVEDD (mm)	Mean ± SD	52.28±3.35	51.83±4.89	0.923•	0.357	NS
	Range	42-59	38-62			
LVESD (mm)	Mean ± SD	34.47±3.23	34.57±4.63	-0.203•	0.840	NS
	Range	27-45	24-55			
Mechanical complications	No	150 (100%)	150 (100.0%)	-	-	-
	Yes	0 (100%)	0 (0.0%)			
LV thrombus	No	147 (98.0%)	148 (98.7%)	0.203*	0.652	NS
	Yes	3 (2.0%)	2 (1.3%)			
In hospital MACE						
Mortality	No	148 (98.7%)	149 (99.4%)	0.330*	0.566	NS
	Yes	2 (1.3%)	1 (0.6%)			
Reinfarction	No	150 (100.0%)	150 (100.0%)	-	-	-
	Yes	0 (0.0%)	0 (0.0%)			
Urgent revascularization	No	150 (100.0%)	150 (100.0%)	-	-	-
	Yes	0 (0.0%)	0 (0.0%)			
CVS	No	150 (100.0%)	150 (100.0%)	-	-	-
	Yes	0 (0.0%)	0 (0.0%)			
Cardiogenic shock requiring IV supports	No	146 (97.3%)	148 (98.7%)	0.680*	0.410	NS
	Yes	4 (2.7%)	2 (1.3%)			
Malignant ventricular arrythmia	No	147 (98.0%)	148 (98.7%)	0.203*	0.652	NS
	Yes	3 (2.0%)	2 (1.3%)			
Major bleeding	No	149 (99.3%)	150 (100.0%)	1.003*	0.317	NS
	Yes	1 (0.7%)	0 (0.0%)			
Hospital stay (days)	Median (IQR)	2 (2-4)	2 (1.5-3)	3.204	0.001	HS

P-value >0.05: Non significant, *P*-value <0.05: Significant, *P*-value <0.01: Highly significant

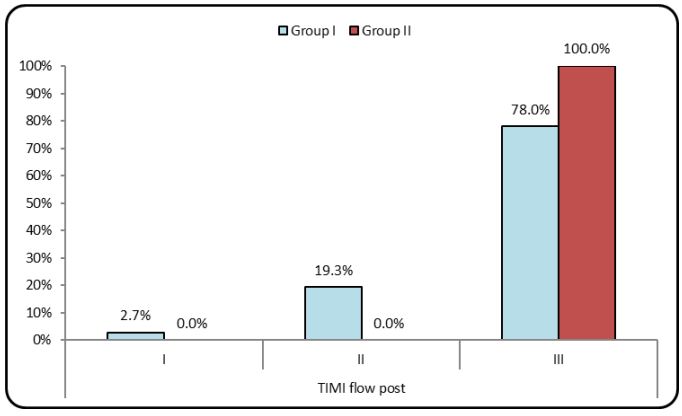
*: Chi-square test, •: Independent t-test, TIMI: Thrombolysis in myocardial infarction, BP: Blood pressure, CVS: Cerebrovascular stroke, EF: Ejection fraction, LVEDD: Left ventricular end diastolic diameter, LVESD: Left ventricular end systolic diameter, MACE: Major adverse cardiovascular events, NS: Non-significant, LV: Left ventricular, IQR: Interquartile range, SD: Standard deviation

sizes are both strongly associated with poorer TIMI flow post-procedure and MBG outcomes, indicating poorer results with increased pressure and size discrepancies. Additionally, longer stents correlate negatively with both TIMI flow and MBG, suggesting that longer stents may lead to worse outcomes with SPD. The duration of chest pain is also negatively correlated with both TIMI flow and MBG, indicating that prolonged chest pain is linked to poorer post-procedural results as shown in Table 4 and Figures 4-7.

Conversely, stent diameter and NC balloon length do not significantly affect TIMI flow or MBG.

Although multiple clinical and procedural variables were included in the regression analysis, only the factors presented in Table 5 showed statistically significant associations with no-reflow. In univariate analysis, several variables were significantly associated with no-reflow, including stent-post dilatation, hypertension, chronic kidney disease (CKD), high SPD pressure (>16 atm), and a size discrepancy >0.25 mm between the NC balloon and stent. Among these, CKD and SPD pressure >16 atm remained independent predictors in multivariate analysis.

(a)



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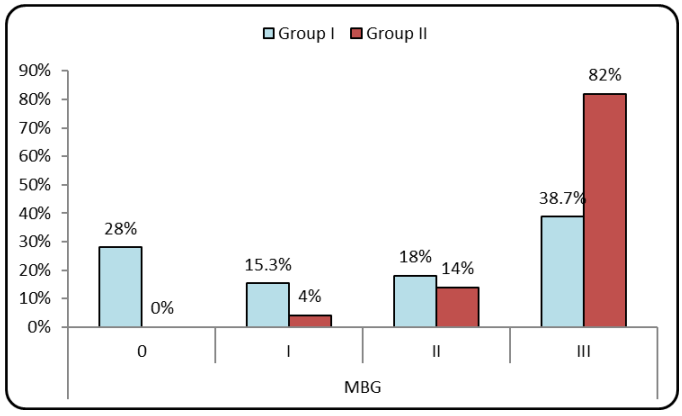

Figure 2: Comparative assessment of post-procedural TIMI flow (a) and myocardial blush grade (MBG) (b) between group I and group II among studied cases

TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade

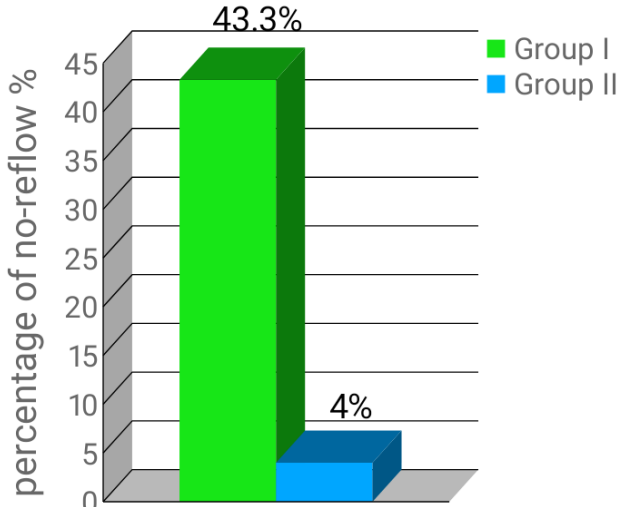

Figure 3: Comparison between group I and group II regarding post-procedural no-reflow of studied cases

Table 4: Correlations between various parameters (chest pain duration, stent diameter, length, NC balloon length, size and pressure) and TIMI Flow & MBG post operative

	TIMI flow post		MBG	
	r	*P*-value	r	*P*-value
Pressure of NC balloon inflation	-0.380**	0.000	-0.387**	0.000
Difference between NC & stent size	-0.319**	0.000	-0.299**	0.000
NC balloon length	0.137	0.093	0.020	0.809
Stent diameter	0.099	0.227	0.105	0.200
Stent length	-0.262**	0.001	-0.186*	0.023
Chest pain duration	-0.279**	0.001	-0.169*	0.039

*: Significant; **: Highly significant, Spearman correlation coefficient, TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade, NC: Non-compliant

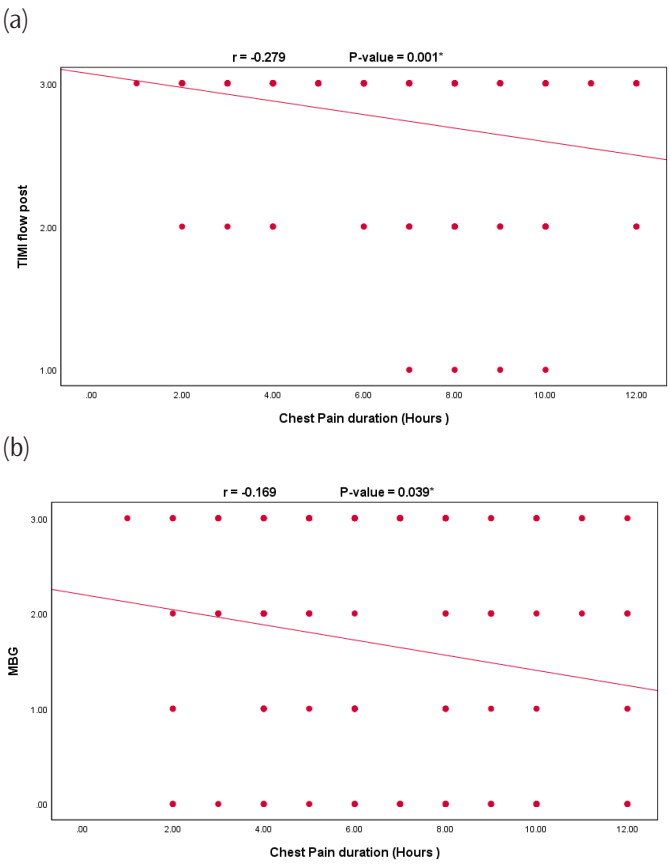

Figure 4: Correlation between TIMI flow (a) & MBG (b) and chest pain duration

**: Significant, TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade*

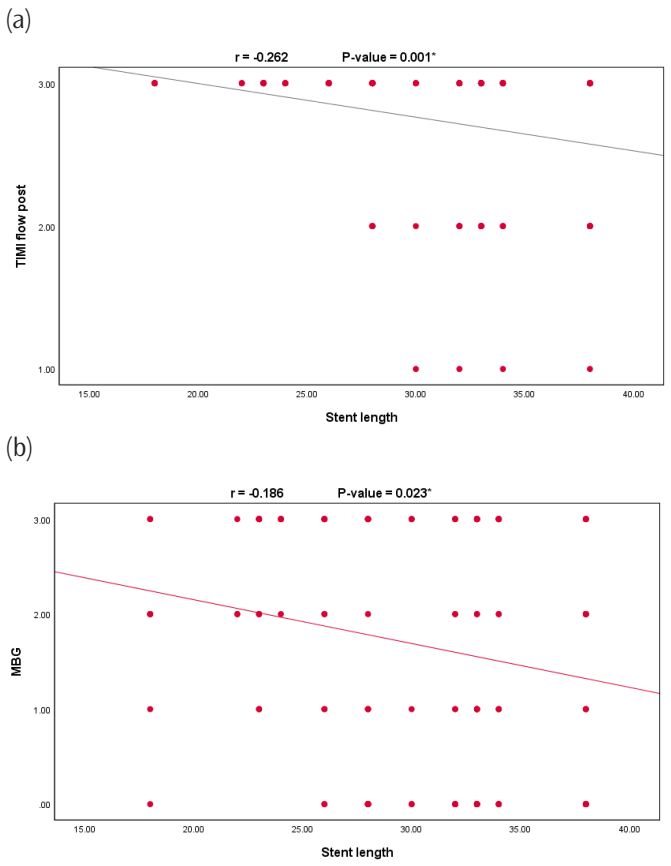

Figure 5: Correlation between TIMI flow (a) & MBG (b) and stent length

**: Significant, TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade*

DISCUSSION

SPD has been acknowledged to enhance both angiographic results and clinical outcomes in patients with stable coronary artery disease. However, its application in the context of acute myocardial infarction (AMI) remains a subject of concern due to potential risks associated with its use in this acute setting.[7]

The clinical value of post-dilatation during stenting for AMI has been debated, with prior studies reporting conflicting outcomes. Zhang et al.[8] were first to demonstrate that employing post-dilatation in this setting may elevate the long-term risk of mortality or recurrent MI.

Conversely, other research has indicated that post-stent dilatation in the setting of AMI may be linked to a reduced risk of TVR and overall MACEs during a clinical follow-up period of up to five years.[9]

Our study was conducted at Ain Shams University Hospitals, which is considered a high-volume tertiary PCI center that provides primary PCI service 24/7, with an average time-to-wire crossing of 30-45 minutes; this was reflected in our study (mean time-to-wire crossing was 34.67±10.03 minutes in group I and 35.57± 0.34 minutes in group II).

In our study, patients in group I, who underwent SPD, exhibited significantly lower TIMI flow and MBG compared to patients in group II, who did not undergo SPD. As a result, group I had a higher incidence of no-reflow.

As per the study protocol, only patients who achieved TIMI III flow after stenting were included; thus, all patients in group II had TIMI III flow. However, in group I, TIMI III flow was affected after SPD, so only 117 patients (78%) persisted to have TIMI III flow. Additionally, 4 patients had TIMI I flow, and 29 patients had TIMI II flow.

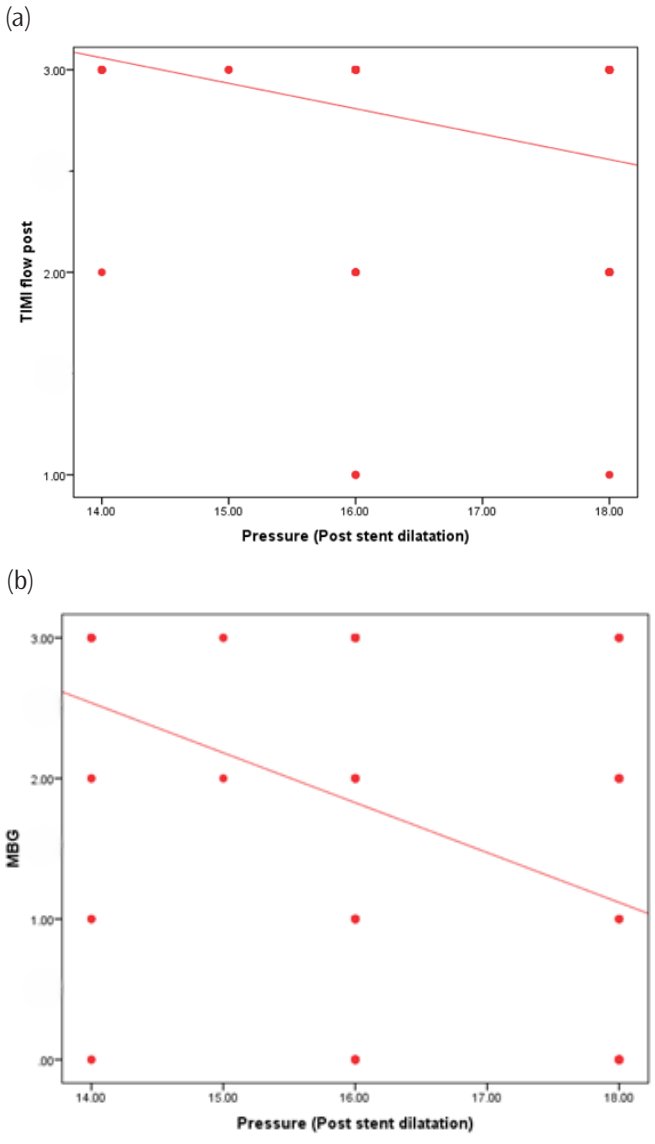

Figure 6: Correlation between TIMI flow (a) & MBG (b) and pressure of NC balloon inflation

TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade, NC: Non-compliant

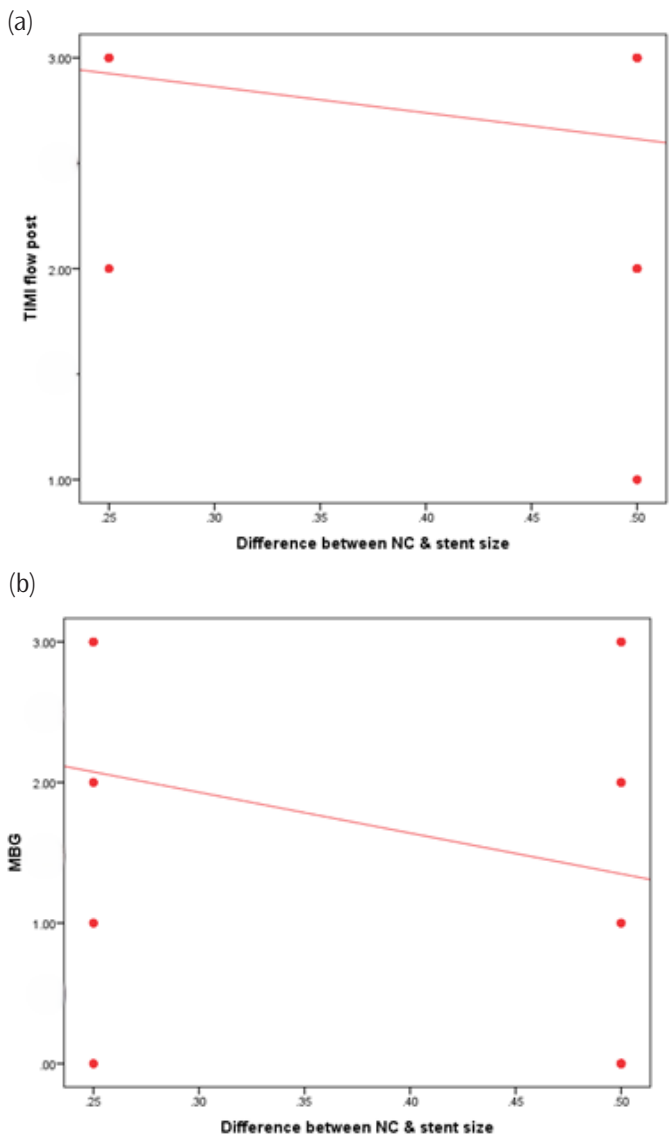

Figure 7: Correlation between TIMI flow (a) & MBG (b)and difference between NC and stent size

TIMI: Thrombolysis in myocardial infarction, MBG: Myocardial blush grade, NC: Non-compliant

Table 5: Univariate and multivariate logistic regression analysis to assess factors associated with no reflow

	Univariate				Multivariate			
	P-value	OR	95% CI for OR		*P*-value	OR	95% CI for OR	
			Lower	Upper			Lower	Upper
Stent-post dilatation	**0.000**	18.353	7.626	44.167	-	-	-	-
Hypertension	**0.039**	1.763	1.029	3.021	-	-	-	-
Dyslipidemia	0.051	1.905	0.997	3.640	-	-	-	-
CKD	**0.046**	2.286	1.016	5.143	**0.009**	8.134	1.695	39.041
SPD inflation pressure >16 atm	**0.004**	2.786	1.384	5.606	**0.007**	2.717	1.322	5.587
Difference between NC & stent size >0.25 mm	**0.021**	2.199	1.128	4.286	-	-	-	-

OR: Odds ratio, CI: Confidence interval, NC: Non-compliant, CKD: Chronic kidney disease, SPD: Stent post-dilatation

Regarding MBG, most patients (96%) in group II with no SPD achieved MBG II (21 patients) and III (123 patients); only 6 patients had MBG I. However, in group I with SPD, 42 patients had MBG 0, 23 patients had MBG I, and only 85 patients (56.7%) achieved MBG II (27 patients) and MBG III (58 patients).

As a result, no-reflow by its definition (TIMI flow less than III or TIMI III flow with MBG grade of 0, 1) occured in 65 patients in group I versus 6 patients in group II (43.3% vs. 4%).

We concluded that there's a significant relationship between SPD in the setting of primary PCI and the occurrence of no reflow. It is important to note that all patients achieved TIMI III flow immediately post-stenting prior to randomization. Therefore, the observed reduction in TIMI flow grade and MBG within the SPD group reflects the direct procedural impact of SPD itself. This methodological aspect is a critical consideration when interpreting the relationship between SPD and post-procedural microvascular outcomes in our study. Our finding that SPD was associated with lower TIMI flow, reduced MBG, and a higher incidence of no-reflow, is paradoxical and challenges conventional beliefs regarding SPD's benefits in primary PCI. Several mechanisms may explain this phenomenon. First, SPD may promote distal embolization by dislodging thrombotic material or plaque debris, even in patients with apparent TIMI III flow post-stenting. Second, the high-pressure inflation with an oversized NC balloon could induce microvascular spasm or direct microvascular injury. Third, while no angiographically visible dissections were recorded, subtle dissections or intimal tears below the detection threshold may have occurred, adversely affecting perfusion. Fourth, SPD might exacerbate microvascular compression or contribute to myocardial edema, aggravating microvascular obstruction. Collectively, these potential mechanisms suggest that SPD, in the context of STEMI, may have unintended deleterious effects on microvascular integrity, warranting further investigation with advanced imaging modalities and microcirculatory assessments. Our study's higher observed no-reflow rates in the SPD group reflect a transient deterioration in coronary flow immediately post-SPD. This indicates a temporary issue, rather than permanent microvascular damage, as evidenced by final angiographic equivalence between groups after adjunctive management. This distinction is critical, as it suggests that SPD may acutely provoke microvascular compromise, possibly via the mentioned mechanisms, but that such effects are often reversible with appropriate intra-procedural strategies. Therefore, while SPD may transiently impair myocardial perfusion, careful technique and adjunctive measures can mitigate these risks. Our regression analysis highlights that, while the application of SPD itself did not independently predict no-reflow after adjustment, the use of high inflation pressures during SPD (>16 atm) was an independent predictor. This implies that procedural technique, specifically the degree of mechanical stress on the vessel wall, may play a more critical role in microvascular injury.

The results of our study were harmonized with those reported in a retrospective study by Senoz and Yurdam[1] which evaluated 255 STEMI patients who underwent primary PCI. Of these, 115 patients received SPD, while 140 did not. The incidence of no-reflow (TIMI 0-2) at baseline and immediately after stent implantation was comparable between SPD and non-SPD groups (94.4% vs. 95.4%, $P = 0.757$; and 23.1% vs. 20.4%, $P = 0.621$, respectively). However, final no-reflow rate was markedly elevated among patients in the SPD group (22.2% vs. 9.3%, $P = 0.009$).

To note that our study is different from that of Senoz and Yurdam[1] as we did a prospective randomized trial that excluded patients with angiographic evidence of heavy thrombus burden, patients with less than TIMI III flow after stenting and those who needed aggressive pre-dilatation using large NC balloons >2.5 mm as these factors could increase risk of no-reflow and potentially affect results.

Even though the group of SPD in our study experienced no-reflow more than other group.

Our results were further aligned with a meta-analysis by Putra et al.[10] which encompassed ten studies and reported that SPD was performed in 40.7% of patients. The analysis revealed a significant association between post-dilatation and an elevated risk of no-reflow during primary PCI, with an odds ratio of 1.33 (95% CI: 1.12-1.58; $P = 0.001$).

Our study was discordant with the post-dilatation STEMI trial, [11] a prospective observational study, which found that SPD with NC balloons in STEMI patients undergoing primary PCI significantly improved stent expansion, apposition, and post-PCI FFR, though it had no overall significant effect on coronary microcirculation.

The discrepancy between our findings and those of the post-dilatation STEMI trial may, in part, be explained by our exclusion of patients with marked stent underexpansion, who required immediate post-dilatation. In contrast, the post-dilatation STEMI trial permitted operator-driven decision-making regarding post-dilatation, and patients deemed not to require it were excluded from the study population.

As mentioned, in our study, no-reflow assessment was done immediately post-dilatation and not in the final one, as operators had managed no-reflow with different measures to improve flow. This was successful in most of the patients, explaining why both groups had a similar in-hospital outcome.

As for the in-hospital outcome, there was substantial variation between the two groups in post-PCI chest pain, which was more frequent in group I.

Echocardiographic assessment performed on the day following primary PCI revealed no substantial variations between study groups in terms of ejection fraction, cardiac chamber dimensions, or mechanical complications.

In a study by Morishima et al.[12] 120 consecutive patients experiencing their first AMI and treated with percutaneous transluminal coronary angioplasty (PTCA), in the absence of flow-limiting lesions, were evaluated. Based on post-PTCA cineangiographic findings, patients were categorized into a no-reflow group (n=30) and a reflow group with TIMI grade 3 flow (n=90). Over a mean follow-up period of 5.8±1.2 years, survivors in the no-reflow group exhibited significantly higher LV end-diastolic and end-systolic volume indices, elevated plasma brain natriuretic peptide levels, and reduced LV ejection fractions compared to those in the reflow group. These findings suggest that the no-reflow phenomenon may contribute to adverse LV remodeling.

So good initial post PCI echocardiography may not indicate good late outcome and long term follow up is mandatory.

In-hospital MACE was generally similar between the SPD and no-SPD groups. Mortality rates were low and not significantly different (1.3% vs. 0.6%, $P = 0.330$). No cases of reinfarction, urgent revascularization, or CVS occurred in either group. Incidences of cardiogenic shock (2.7% vs. 1.3%, $P = 0.410$), ventricular arrhythmia (2.0% vs. 1.3%, $P = 0.652$), and major bleeding (0.7% vs. 0%, $P = 0.317$), were slightly higher in the SPD group, but none of these differences reached statistical significance.

This is consistent with findings of Gao et al.[4] who conducted a prospective study involving 336 AMI patients, of whom 199 (59.2%) underwent post-stent dilation. There were no substantial variations between the two groups in terms of HF, ventricular aneurysm, stent thrombosis, cardiac death, non-cardiac death, or severe hemorrhage ($P > 0.05$). Follow-ups at 30 days post-procedure showed no differences in stent thrombosis, TVR, or MACE.

Although our study did not find notable variations in clinical outcomes between two groups due to relatively short follow-up period, and may be due to good final flow after measures taken by operators to deal with no-reflow.

Additional research has emphasized long-term implications of the no-reflow phenomenon. In a study by Kim et al.[13] data from 4,329 patients with AMI enrolled in a Korean multicenter registry were analyzed. Among these, 4,071 patients exhibited no evidence of no-reflow, while 213 experienced transient no-reflow, and 45 had persistent no-reflow following PCI.

Over a three-year follow-up period, patients in the persistent no-reflow group demonstrated significantly higher rates of all-cause mortality (HR: 1.98; 95% CI: 1.08-3.65; $P = 0.028$) and cardiac mortality (HR: 3.28; 95% CI: 1.54-6.95; $P = 0.002$) relative to the normal reflow group. Transient no-reflow was associated with an elevated risk of all-cause mortality only relative to the normal reflow group (HR: 1.58; 95% CI: 1.11-2.24; $P = 0.010$). Additionally, when comparing transient to persistent no-reflow, the latter was linked to a markedly higher all-cause mortality rate (46.7% vs. 24.4%, log-rank $P = 0.033$).

In a study by Choo et al.[14] a total of 2,017 patients with STEMI who underwent primary PCI were consecutively enrolled in the Korean multi-center AMI registry. The primary endpoint was all-cause mortality, and the no-reflow phenomenon was identified in 262 patients, representing 13.0% of the cohort. Patients exhibiting no-reflow phenomenon, demonstrated a markedly elevated mortality rate relative to those with successful reflow (30.2% vs. 18.3%, $P < 0.001$). Multivariate analysis using the Cox proportional hazards model identified no-reflow as an independent predictor of long-term mortality (adjusted hazard ratio: 1.45; 95% CI: 1.12-1.86; $P = 0.004$).

Additionally, in our study, correlations between chest pain duration and procedural parameters, including stent diameter, stent length, NC balloon length, inflation size and pressure, and TIMI flow and MBG post-operative, revealed that longer chest pain duration, higher NC balloon inflation pressure, higher NC balloon to stent size ratio, and longer stent length were strongly and negatively correlated with TIMI flow and MBG outcomes, leading to poorer post-procedural results.

Conversely, stent diameter and NC balloon length didn't significantly affect TIMI flow or MBG.

In summary, routine SPD in the setting of primary PCI, after excluding the urgent need for this technique, is usually associated with transient no-reflow and may result in a poor outcome. However, this was not proven in our study due to several factors, such as no-reflow being managed by operators in most of the patients and the very short in-hospital follow-up period.

Future research should incorporate mechanistic studies using advanced intracoronary imaging modalities, such as optical coherence tomography or intravascular ultrasound (IVUS), to precisely evaluate stent expansion, residual plaque burden, and potential plaque disruption induced by SPD. Additionally, techniques such as microvascular resistance measurements, index of microcirculatory resistance, or coronary flow reserve could provide functional insights into microvascular integrity post-SPD.

Randomized trials integrating these imaging and physiological assessments could help delineate the direct relationship between SPD, distal embolization, microvascular dysfunction, and clinical outcomes in STEMI. Such studies would also guide the development of refined SPD protocols that balance stent optimization with microvascular protection.

Study Limitations

This study is limited by its single-center design and short in-hospital follow-up duration. Additionally, assessment of myocardial perfusion relied solely on TIMI flow and MBG, whereas more advanced modalities such as MRI or myocardial contrast echocardiography could provide more comprehensive evaluations. Furthermore, IVUS-guided PCI was not available during the study period. A key limitation of our study is that randomization occurred only after successful stent placement and restoration of TIMI III flow. This excludes patients with stent under-expansion or poor initial flow, reducing the generalizability of the results. Additionally, high-risk STEMI patients, such as with cardiogenic shock, heavy thrombus, bifurcation lesions, or needing aggressive pre-dilatation, were not included. Since these patients are at greater risk for no-reflow and may respond differently to SPD, our findings mainly apply to lower-risk STEMI patients with optimal initial outcomes and should not be extended to more complex patients without further research. In addition, the study was conducted by a team of multiple operators, which may have introduced inter-operator variability, despite following standardized protocols for PCI and SPD. Especially within the SPD group, procedural variation occurred as procedural variation balloon size was ultimately chosen based on individual operator judgment, potentially leading to minor inconsistencies in SPD across patients. Also, despite randomization, there remains potential for residual confounding from unmeasured variables that may have influenced the incidence of no-reflow. Finally, blinding was not feasible for operators due to the nature of the intervention.

CONCLUSION

Post-stent dilatation during primary PCI in STEMI patients was associated with a higher incidence of transient no-reflow immediately following the procedure. However, this did not translate into a significant difference in short-term in-hospital clinical outcomes, likely due to prompt intra-procedural management of no-reflow.

Our study does not support routine stent-post dilatation in the setting of primary PCI, especially with long chest pain duration, long stents, large difference between stent and NC balloon size, and with high NC pressure as these factors had negative correlation with TIMI flow and MBG. However, given the study's single-center nature, operator-dependent variability, lack of blinding, and short follow-up, larger studies are needed before definitive practice recommendations can be made.

Ethics

Ethics Committee Approval: The study protocol was approved by Ain Shams University Faculty of Medicine Scientific and Ethical Committee (protocol no: FMASU MS82/2024, date: 06.02.2024).

Informed Consent: Written informed consent was secured from all participants, with strict adherence to ethical standards ensuring protection of privacy and confidentiality of personal data.

Footnotes

Authorship Contributions

Surgical and Medical Practices: H.S., T.M.A.A., M.M.I., Concept: H.S., T.M.A.A., Design: S.G., T.M.A.A., Data Collection or Processing: S.G., H.S., T.M.A.A., M.M.I., Analysis or Interpretation: S.G., H.S., T.M.A.A., M.M.I., Literature Search: S.G., M.M.I., Writing: S.G., M.M.I.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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