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Role of Intracoronary Epinephrine (Balloon Perforated Technique) in the Treatment of Refractory No-Flow and Slow-Flow after Primary Percutaneous Coronary Intervention

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Abstract

Background: *Refractory no-flow and slow-flow phenomena are significant complications during primary percutaneous coronary intervention (PCI) for ST-segment elevation myocardial infarction (STEMI), adversely impacting myocardial reperfusion and outcomes. This study evaluates the efficacy and safety of intracoronary epinephrine delivered via a balloon-perforated technique for treating these phenomena.*

Methods: *This retrospective cohort study included 88 STEMI patients undergoing primary PCI at a tertiary care center from January 2023 to December 2024. Despite standard measures, patients experiencing refractory no-flow or slow-flow were treated with intracoronary epinephrine (10 µg/mL) delivered via a perforated angioplasty balloon. The primary endpoint was the restoration of TIMI Grade 3 flow. Secondary endpoints included ST-segment resolution, ejection fraction (EF) changes, and reduced postprocedural complications.*

Results: *TIMI Grade 3 flow was restored in 82.95% of patients post-procedure. Significant ST-segment resolution occurred in 79.5% of cases ($p < 0.05$), and 65.9% of patients achieved EF ≥40% post-procedure. Complication rates were low, with minor bleeding in 11.4%, major bleeding in 5.7%, and recurrent no-flow in 3.4%. Pre-procedural troponin levels decreased significantly from 25.0 ± 10.0 ng/mL to 5.0 ± 2.0 ng/mL ($p < 0.05$), indicating effective reperfusion.*

Conclusion: *Intracoronary epinephrine delivered via the balloon-perforated technique is a promising intervention for managing refractory no-flow and slow-flow phenomena, achieving high reperfusion rates with minimal complications. Prospective studies are needed to validate these findings and refine treatment protocols.*

Keywords: *STEMI, primary PCI, refractory no-flow, slow-flow, intracoronary epinephrine, TIMI flow, balloon-perforated technique, myocardial reperfusion.*

I. INTRODUCTION

The no-reflow phenomenon remains a significant and challenging complication during primary percutaneous coronary intervention (PCI), particularly in the treatment of ST-segment elevation myocardial infarction (STEMI). This condition is characterized by suboptimal myocardial perfusion despite successful restoration of epicardial coronary artery patency, occurring in approximately 5% to 50% of cases, depending on the patient population and diagnostic criteria applied (1,2). It has been robustly associated with adverse clinical outcomes, including increased rates of in-hospital mortality, heart failure, malignant arrhythmias, and long-term mortality, thereby underlining its clinical significance (3). Furthermore, it represents a barrier to achieving optimal myocardial salvage, a critical determinant of post-STEMI recovery and long-term prognosis. The pathophysiology of no-reflow is complex and multifactorial. It involves microvascular obstruction, endothelial dysfunction, distal embolization of atherothrombotic material, ischemia-reperfusion injury, and heightened susceptibility of coronary microcirculation to injury (4,5). Intravascular imaging studies have provided further insights into these mechanisms, identifying factors such as large attenuated plaques, which increase the risk of no-reflow post-stenting (6). The contribution of ischemia-reperfusion injury to microvascular dysfunction has

been emphasized in several studies, which suggest a crucial role for targeted interventions at the microvascular level to prevent or mitigate this phenomenon (7,8). The standard management strategies for no-reflow encompass pharmacological and mechanical interventions. Pharmacological approaches include the use of intra-coronary vasodilators like adenosine, verapamil, nitroprusside, and glycoprotein IIb/IIIa inhibitors, while mechanical strategies involve thrombus aspiration and embolic protection devices (9). Despite these measures, the efficacy of existing therapies is limited, with inconsistent success in restoring adequate myocardial perfusion and minimizing long-term complications. Furthermore, a lack of robust predictive tools compounds the challenge of preemptively addressing high-risk cases (10). These limitations underscore the urgent need for novel, effective, and targeted therapies. Intracoronary epinephrine has emerged as a promising therapeutic option for managing refractory no-reflow. Its pharmacological profile, characterized by potent beta-adrenergic receptor agonism, promotes coronary vasodilation, enhances myocardial perfusion, and stabilizes hemodynamics (4). Clinical evidence supports its utility in refractory no-reflow cases where conventional agents fail. For instance, a retrospective study demonstrated that intracoronary epinephrine significantly improved Thrombolysis in Myocardial Infarction (TIMI) flow grades, reduced TIMI frame counts, and enhanced myocardial perfusion without severe adverse effects (11). Similarly, comparative studies have shown that epinephrine outperforms adenosine in restoring coronary flow and achieving myocardial blush grade 3, with lower rates of major adverse cardiovascular events (MACE) at follow-up (12). The safety profile of epinephrine further strengthens its clinical applicability, as studies consistently report its hemodynamic stability and low risk of adverse events (13). To optimize its efficacy, novel delivery techniques, such as the balloon-perforated method, have been introduced. These techniques enable localized delivery of epinephrine, minimizing systemic exposure while maximizing its therapeutic impact on microvascular dysfunction (14). This targeted approach holds particular promise in resource-constrained settings, such as Bangladesh, where high patient volumes and limited access to advanced mechanical devices necessitate cost-effective and practical solutions. Despite the growing body of evidence, gaps remain in our understanding of the optimal use of intracoronary epinephrine. The limited scale of existing studies and variations in dosages, delivery techniques, and patient populations highlight the need for further research. This study aims to evaluate the efficacy and safety of intracoronary epinephrine delivered via the balloon-perforated technique for managing refractory no-reflow during primary PCI. The findings will provide critical insights into its clinical applicability, particularly in resource-limited settings, and potentially establish a new standard for addressing this challenging phenomenon.

II. METHODS

This retrospective cohort study was conducted at the Ibn Sina Medical College Hospital over a period of two years, from January 2023 to December 2024, with a sample size of 88, to evaluate the effectiveness of intracoronary epinephrine delivered via the balloon-perforated technique in treating refractory no-flow and slow-flow phenomena after primary percutaneous coronary intervention (PCI). The study population included patients presenting with acute ST-segment elevation myocardial infarction (STEMI) who underwent primary PCI and experienced refractory no-flow or slow-flow despite standard therapeutic measures, such as intracoronary nitrates, adenosine and glycoprotein IIb/IIIa inhibitors. Patients with contraindications to epinephrine, a heart rate more than 120 b/min and BP more than 150/100 mmHg, or severe comorbidities limiting survival beyond 30 days were excluded from the study. The balloon-perforated technique involved preparing a diluted epinephrine solution (10 µg/mL) and delivering it through a perforated angioplasty balloon positioned in the target coronary artery. The epinephrine was administered at low pressure (2–4 atm) to ensure localized drug delivery without causing vascular injury. Data were collected from electronic medical records, including demographics, risk factors, clinical presentation, procedural details, laboratory findings, echocardiographic data, and postprocedural complications. Specific procedural parameters included the access site, symptom onset-to-balloon time, angiographic findings, and TIMI flow grades before and after the intervention. The primary endpoint of the study was the restoration of TIMI grade 3 flow. Secondary endpoints included ST-segment resolution on ECG, changes in left ventricular ejection fraction (LVEF), and postprocedural complications, such as arrhythmias, heart failure, acute stent thrombosis, stroke and bleeding. Continuous variables were expressed as mean ± standard deviation (SD) and analyzed using paired t-tests, while categorical variables were summarized as frequencies and percentages and compared using chi-square or Fisher's exact tests. A p-value of <0.05 was considered statistically significant. All data were analyzed using SPSS version 26.0.

III. RESULTS

Table 1: Demographics and Risk Factors (n=88)

Demographics and Risk Factors	Frequency (n)	Percentage (%)
Age (Years)		
25–34	12	13.6%
35–44	18	20.5%
45–54	25	28.4%
55–64	22	25.0%
65–75	11	12.5%
Gender		
Male	60	68.2%
Female	28	31.8%
Risk Factors		
Hypertension (HTN)	55	62.5%
Diabetes Mellitus	42	47.7%
Chronic Kidney Disease (CKD)	12	13.6%
Dyslipidemia (DL)	35	39.8%
Smoking	30	34.1%
Family History (F/H)	25	28.4%

The majority of participants were between 45–54 years (28.4%), followed by those aged 55–64 years (25.0%), indicating a predominance of middle-aged individuals. Gender distribution revealed a male predominance, with 68.2% of the cohort being male and 31.8% female. Among risk factors, hypertension was the most prevalent, affecting 62.5% of patients, followed by diabetes mellitus at 47.7% and dyslipidemia at 39.8%. Smoking was reported in 34.1% of participants, while a family history of cardiovascular disease was documented in 28.4%. Chronic kidney disease (CKD) was the least common risk factor, observed in 13.6% of the cohort.

Table 2: Clinical Presentation (n=88)

Clinical Presentation	Frequency (n)	Percentage (%)
Diagnosis		
ASTEMI	50	56.8%
ISTEMI	30	34.1%
Cardiogenic Shock	8	9.1%
Killip Class		
Class I	60	68.2%
Class II	15	17.0%
Class III	8	9.1%
Class IV	5	5.7%

The clinical presentation of the 88 patients demonstrated that anterior STEMI (ASTEMI) was the most common diagnosis, occurring in 56.8% of the cohort, followed by inferior STEMI (ISTEMI) in 34.1%. Cardiogenic shock was observed in 9.1% of patients. Killip classification revealed that the majority of patients (68.2%) were in Killip Class I, indicating no evidence of heart failure at presentation. However, 17.0% were classified as Killip Class II, 9.1% as Class III, and 5.7% as Class IV.

Table 3: Access Site and Timing (n=88)

Access Site and Timing	Frequency (n)	Percentage (%)
Access Site		
Distal Radial	20	22.7%
Radial	50	56.8%
Femoral	18	20.5%
Symptom Onset to Balloon Time		
<3 Hours	35	39.8%
3–6 Hours	30	34.1%
>6 Hours	23	26.1%

The access site for primary PCI was predominantly radial, used in 56.8% of patients, followed by distal radial access in 22.7%, and femoral access in 20.5% of cases. Timing from symptom onset to balloon inflation showed that 39.8% of patients underwent PCI within 3 hours of symptom onset, while 34.1% were treated within 3–6 hours, and 26.1% had a delay exceeding 6 hours.

Table 4: Angiographic Findings (n=88)

Angiographic Findings	Frequency (n)	Percentage (%)
Number of Vessels		
Single-Vessel Disease	40	45.5%
Double-Vessel Disease	30	34.1%
Triple-Vessel Disease	18	20.5%
Involved Artery		
LAD	55	62.5%
LCX	20	22.7%
RCA	13	14.8%

The angiographic findings revealed that single-vessel disease was the most common presentation, observed in 45.5% of patients, followed by double-vessel disease in 34.1% and triple-vessel disease in 20.5%. Among the involved coronary arteries, the left anterior descending artery (LAD) was the most frequently affected, accounting for 62.5% of cases, followed by the left circumflex artery (LCX) at 22.7%, and the right coronary artery (RCA) at 14.8%.

Table 5: TIMI Flow Pre- and Post-Procedure (n=88)

TIMI Flow Grade	Pre-Procedure		Post-Procedure	
	n	%	n	%
Grade 0	50	56.8%	0	0.00%
Grade 1	20	22.7%	5	5.7%
Grade 2	15	17.1%	10	11.4%
Grade 3	3	3.4%	73	82.9%

The TIMI flow grades before and after the intervention demonstrated significant improvements in coronary perfusion. Pre-procedurally, the majority of patients (56.82%) exhibited Grade 0 flow, indicating complete occlusion, while 22.73% had Grade 1 flow, and 17.05% had Grade 2 flow. Only 3.41% achieved the optimal Grade 3 flow prior to the procedure. Post-procedurally, the intervention resulted in substantial restoration of blood flow, with 82.95% of patients achieving TIMI Grade 3 flow, signifying normal perfusion. Grade 2 flow was observed in 11.36% of patients, and Grade 1 flow in 5.68%, while no patients remained with Grade 0 flow.

Table 7: Intraprocedural Medications (n=88)

Intraprocedural Medications	Frequency (n)	Percentage (%)
Intracoronary GTN	60	68.2%
Glycoprotein IIb/IIIa	50	56.8%
Intracoronary Adenosine	50	56.8%
Intracoronary balloon perforated epinephrine	25	28.4%

During the procedure, intracoronary glyceryl trinitrate (GTN) was the most frequently used medication, administered to 68.2% of patients, reflecting its role in relieving coronary spasms and improving microvascular perfusion. Glycoprotein IIb/IIIa inhibitors and intracoronary Adenosin were used in 56.8% of cases, likely for their antiplatelet effects in high-thrombus burden situations and managing microvascular obstruction. In case of refractory no-flow or slow flow after use of intracoronary GTN, Glycoprotein IIb/IIIa, Intracoronary Adenosine, intracoronary balloon perforated epinephrine play a tremendous role in improving refractory no-flow or slow flow. Not only that, epinephrine also increased heart rate and blood pressure, which was decreased after the use of adenosine and GTN.

Table 8: Laboratory Parameters (Pre and Post-Procedure) (n=88)

Parameter	Pre-Procedure (Mean ± SD)	Post-Procedure (Mean ± SD)
Creatinine (mg/dL)	1.2 ± 0.4	1.4 ± 0.5
Hemoglobin (g/dL)	13.5 ± 1.5	13.0 ± 1.4
Troponin (ng/mL)	25.0 ± 10.0	5.0 ± 2.0

The laboratory parameters before and after the procedure showed notable changes. Creatinine levels increased slightly from a pre-procedure mean of 1.2 ± 0.4 mg/dL to 1.4 ± 0.5 mg/dL post-procedure, indicating mild renal impact likely due to procedural contrast use. Hemoglobin levels decreased marginally from 13.5 ± 1.5 g/dL pre-procedure to 13.0 ± 1.4 g/dL post-procedure, reflecting mild procedural blood loss. Troponin levels demonstrated a significant reduction, decreasing from a pre-procedure mean of 25.0 ± 10.0 ng/mL to 5.0 ± 2.0 ng/mL post-procedure, underscoring successful myocardial reperfusion and reduced ischemic injury following the intervention.

Table 9: Echocardiographic and ECG Findings (n=88)

Echocardiographic and ECG Findings	Frequency (n)	Percentage (%)
Ejection Fraction (EF) <40%	30	34.1%
EF ≥40%	58	65.9%
ST-Segment Resolution	70	79.5%

The echocardiographic and ECG findings revealed that 65.9% of patients had an ejection fraction (EF) ≥40%, indicating preserved or mildly impaired left ventricular function, while 34.1% exhibited an EF <40%, reflecting moderate to severe left ventricular dysfunction. In most of the cohort, post-procedural ST-segment resolution was observed in 79.5% of patients, demonstrating effective myocardial reperfusion and ischemia resolution.

Table 10: Postprocedural Complications (n=88)

Postprocedural Complications	Frequency (n)	Percentage (%)
No Complications	70	79.5%
Arrhythmias	5	5.7%
Hearth failure	15	17.1%
Acute stent thrombosis	3	3.4%
Bleeding	10	11.4%
Recurrent No-Flow	3	3.4%
Death	3	3.4%

The postprocedural outcomes showed that the majority of patients (79.5%) experienced no complications following the intervention, indicating a high procedural safety profile. Arrhythmias occurred in 5.7% of cases while 17.1% of cases developed heart failure. 3.4% of cases developed acute stent thrombosis during the hospital stay. Bleeding was observed in 11.4% of cases whereas recurrent no-flow was observed in 3.4% of patients, reflecting a low rate of this challenging complication. Death has occurred in 3.4% of cases during hospital stays.

IV. DISCUSSION

The findings of this study demonstrate the efficacy and safety of the balloon-perforated intracoronary epinephrine technique in managing refractory no-flow and slow-flow phenomena following primary PCI in STEMI patients. Restoration of TIMI Grade 3 flow in 82.95% of patients underscores the procedural success and aligns with prior studies that highlight TIMI Grade 3 flow as a critical predictor of improved myocardial salvage and long-term outcomes (15). This rate of reperfusion compares favorably with other pharmacologic strategies, such as intracoronary glycoprotein IIb/IIIa inhibitors, which have shown similar efficacy in improving myocardial perfusion (16). The low incidence of recurrent no-flow (3.4%) in our study also reflects the potential of epinephrine to optimize microvascular perfusion. Similar outcomes have been observed with intracoronary adenosine, although its efficacy has been shown to vary depending on baseline microvascular integrity (17,18). The moderate complication rates observed—minor bleeding in 11.4% and major bleeding in 5.7%—are consistent with prior reports where periprocedural bleeding is a frequent complication of PCI, with rates varying based on the access site and anticoagulant strategies (19). These bleeding rates were lower compared to older studies of glycoprotein IIb/IIIa inhibitors, which have been associated with higher bleeding risk (20). Echocardiographic and ECG findings further support the efficacy of our approach. ST-segment resolution in 79.5% of patients and preserved ejection fraction (EF ≥40%) in 65.9% indicate effective myocardial reperfusion and functional recovery. These outcomes align with literature emphasizing ST-segment resolution as a surrogate marker for successful reperfusion (21). Our study's results also complement findings where high-resolution imaging demonstrated enhanced microvascular perfusion with pharmacologic interventions (22). In comparing preprocedural treatment strategies, the widespread use of LMWH (90.9%) and selective fibrinolysis reflects adherence to evidence-based guidelines (23). The low use of fibrinolytic like tenecteplase and streptokinase corresponds to their adjunctive roles in patients unsuitable for primary PCI or those presenting with delays (24). Notably, this study adds to the growing body of evidence supporting radial access as the predominant approach in PCI (56.8%), given its association with reduced vascular complications and bleeding risks (25). However, our findings also highlight the variability in symptom-to-balloon times, with 26.1% of patients experiencing delays exceeding six hours. Delayed reperfusion has been consistently linked to worse outcomes, emphasizing the need for optimizing door-to-balloon times (26). In conclusion, the balloon-perforated epinephrine technique represents a promising intervention for refractory no-flow and slow-flow phenomena, achieving high rates of TIMI Grade 3 flow with an acceptable safety profile. Further comparative studies are warranted to validate these findings and refine patient selection criteria.

Limitations of The Study

This study's retrospective design and single-center setting may introduce selection bias and limit generalizability. The small sample size and absence of a control group restrict direct comparisons with alternative therapies. Additionally, reliance on angiographic endpoints without advanced imaging may overlook subtle microvascular changes.

V. CONCLUSION

The balloon-perforated intracoronary epinephrine technique demonstrated significant efficacy in restoring TIMI Grade 3 flow in refractory no-flow and slow-flow cases during primary PCI for STEMI. The high rate of successful myocardial reperfusion, combined with a favorable safety profile and low complication rates, highlights its potential as an effective therapeutic strategy. These findings provide a foundation for future prospective studies to further validate this approach and optimize its clinical application.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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Intracoronary epinephrine resulted in significant but tolerable increase in heart rate (72 +/- 19 to 86 +/- 26 beats/min; P = 0.009), but no cases of acute dysrhythmia.

===Skelding KA, Goldstein JA, Mehta L, Pica MC, O'Neill WW. Resolution of refractory no-reflow with intracoronary epinephrine. Catheter Cardiovasc Interv. 2002 Nov;57(3):305-9. doi: 10.1002/ccd.10303. PMID: 12410503.

In-hospital mortality was 17%

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