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ORIGINAL ARTICLE

Deferred versus Immediate Stenting in Patients Presenting with Aborted Myocardial Infarction

Mohamed Salah Abdelbasit[1*], Mohamed Hosainy Salama[2], Mohamed Saad El-Shetry[1]
[1] Cardiology department, Faculty of Medicine, Zagazig University, Egypt
[2] Zagazig university hospitals, Egypt

[*] **Corresponding author**:
Mohamed Salah Abdelbasit
Email:
msa_20122002@yahoo.com

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ABSTRACT

Background: No-reflow is challenging during primary percutaneous coronary intervention (PCI) of acute myocardial infarction (MI) patients. In stable patients with spontaneously aborted MI, no-reflow could worsen the patients' outcomes. Deferring stenting in such patients could decrease the risk of no-reflow and improve outcomes.

Methods: This prospective cohort study included all patients with aborted MI defined as complete resolution of chest pain and ST segment elevation, and TIMI 3 flow on the initial angiography of the culprit vessel, presenting within 48 hours of chest pain onset. We compared patients who underwent immediate stenting with those with deferred PCI after 48 hours of glycoprotein IIb/IIIa inhibitors infusion regarding risk of no-reflow, in-hospital and one-year outcomes.

Results: This study involved 316 patients with aborted myocardial infarction. Deferred PCI (106 patients) had a lower incidence of no-reflow (20.8% vs 37.1%; P = 0.003), in-hospital heart failure (17% vs 31%; P = 0.007), and one-year all-cause mortality (2.8% vs 9%; P = 0.04) without increase in bleeding risk or in-hospital re-infarction. Regression analysis revealed that lesion length was the most independent predictor of no-reflow (OR: 1.120; P <0.001). BNLTI (**B**ifurcation culprit lesion, **N**umber of stent inflations, lesion **L**ength, **T**hrombus, and **I**mmediate PCI) factor is a novel parameter with cut-off value ≥0.389 predicts no-reflow in patients with aborted MI with sensitivity 80% and specificity 77.3% (AUC: 0.838; P <0.001).

Conclusion: Deferred stenting in patients presenting with aborted MI is associated with lower incidence of no-reflow, better in-hospital and one-year outcomes without increased risk of re-infarction.

Keywords: No-reflow; Myocardial infarction; Primary PCI; Deferred stenting.

INTRODUCTION

Primary percutaneous coronary intervention (PCI) remains the cornerstone in treating acute ST-segment elevation myocardial infarction (STEMI), significantly reducing cardiovascular mortality when promptly administered [1,2]. Nonetheless, PCI in the acute STEMI setting presents notable challenges, predominantly due to a high thrombotic burden and the increased risk of the no-reflow phenomenon.

This phenomenon is defined as impaired epicardial perfusion of the coronary artery (TIMI flow grade 0 or 1) despite successful revascularization and angiographic patency post-stent deployment. This takes place in about 10% of STEMI conditions undergoing primary PCI [3], often due to microvascular obstruction (MVO) as a result of distal

embolization, vasospasm, or micro-thrombosis. This phenomenon is more prevalent among patients with extensive thrombus burden, advanced age, or delayed presentation [3–7].

Direct stenting has demonstrated superiority over balloon angioplasty in reducing the risk of no-reflow [8]. However, it may be associated with technical limitations such as stent under-sizing or mal-apposition due to vasospasm or thrombus burden, increasing subsequent stent thrombosis and restenosis incidence [9,10].

Optimal timing of stent implantation, whether immediate or deferred, remains a subject of ongoing debate. Deferred stenting, initiated after restoration of TIMI 3 flow, is hypothesized to preserve microvascular integrity and reduce thrombus embolization, thus mitigating no-reflow risk. Several studies, including recent meta-analyses, suggest that this strategy improves myocardial blush grade and reduces cardiac death, albeit without consistent statistical significance [11,12]. However, deferral carries the potential risk of in-hospital re-infarction [13–15], and its impact on future left ventricular (LV) function and heart failure remains uncertain [16]. Notably, thrombus burden ongoing with TIMI 3 flow shall aid determine patients that take best advantage of stenting deferral [17]. Conversely, immediate stenting has not consistently demonstrated clinical benefit in such patients [8,18]. In selected scenarios, stent implantation may even be avoidable when residual stenosis is absent after pharmacologic therapy [19]. Cardiac magnetic resonance (CMR)-based investigations have not established superiority of either strategy [20,21].

We hypothesized that in presented cases alongside aborted myocardial infarction (AMI), characterized with complete resolution to chest pain and ST-segment elevation, along with TIMI 3 flow in the infarct-related artery, deferred stenting may be associated with reduction in the incidence of no-reflow and improvement myocardial salvage relative to immediate stenting.

METHODS

Study Design

A prospective cohort study has been performed at the cardiac catheterization laboratory of Zagazig University Hospitals between March 2022 and March 2025. The method has been confirmed by the Institutional Review Board of the Faculty of Medicine, Zagazig University (IRB #11357-26-12-2023), and written informed consent has been gained from the participants.

Study Population

The researchers screened all STEMI cases within 48 hours of chest pain onset who underwent primary PCI during study period. STEMI has been defined per standard ECG characteristics: ST-segment elevation ≥1 mm in two contiguous leads, with specific thresholds for leads V2–V3 based on age and sex [22]. Patients with new-onset left bundle branch block were also eligible.

Inclusion criteria focused on patients with aborted MI, defined by the complete termination of symptoms and ECG finding, and confirmed TIMI 3 flow in the culprit vessel at the same time of initial angiography. Based on operator discretion, cases have been categorized into immediate PCI (Group I) or deferred PCI (Group II), the latter receiving a 48-hour intravenous glycoprotein IIb/IIIa inhibitor infusion prior to stenting. Being observation study, the decision of deferred or immediate stenting was left to the operator choice.

Patients with indications for emergent PCI, persistent symptoms, ongoing ischemia, hypotension, cardiogenic shock, ventricular arrhythmias, or TIMI flow <3, were excluded.

Data Collection and Assessments

Demographic and clinical variables, including heart rate, blood pressure, Killip class, infarction site, loading medications, and baseline laboratory and echocardiographic parameters (LVEDV, LVESV, and LVEF via modified Simpson's biplane method [23]) were recorded.

The whole cases were given standard loading doses of aspirin (300 mg), clopidogrel (600 mg) or ticagrelor (180 mg), atorvastatin (80 mg), and unfractionated heparin (100 IU/kg). Coronary angiography was performed within 60 minutes of emergency department presentation, with vascular access and procedural decisions left to the operator.

In the deferred group, patients received continuous tirofiban infusion (0.15 mg/kg/min for 48 hours) and subcutaneous enoxaparin (1 mg/kg/12 h). Angiographic and procedural variables included lesion characteristics, thrombus burden, stenting technique, use of intracoronary nitroglycerin, balloon inflations,

contrast volume, and post-stenting interventions.

No-reflow was defined as TIMI flow grade 0 or 1 post-stenting despite angiographic stent patency. In-hospital events (reinfarction, heart failure, bleeding, mortality) and 1-year outcomes (all-cause and cardiovascular mortality, reinfarction, repeat revascularization) were recorded.

Statistical Analysis

Continuous parameters were depicted as arithmetic mean ± standard deviation and contrasted employing the Student's t-assessment. Discrete parameters were presented as absolute frequencies and relative proportions, with comparative analyses executed utilizing the chi-square examination. Multivariable logistic modeling was utilized to determine autonomous determinants of no-reflow phenomenon. Receiver operating characteristic (ROC) curve evaluation was undertaken for statistically significant determinants to ascertain optimal cutoff values. A probability value < 0.05 was interpreted as statistically meaningful. Statistical procedures were executed using SPSS version 25.0 (IBM Corporation, Chicago, IL, USA).

RESULTS

A total of 316 patients with aborted MI were enrolled between March 2022 and March 2025. All presented pain-free with TIMI 3 flow in the culprit artery. Of these, 210 patients underwent immediate PCI (Group I), and 106 underwent deferred PCI after 48 hours of glycoprotein IIb/IIIa inhibitor infusion (Group II).

Baseline demographic and clinical profiles were statistically analogous between the cohorts, with no statistical significant differences in MI location, Killip class, P2Y12 inhibitor use, lab results, or echocardiographic parameters (Table 1, table 2).

Angiographic features were similarly matched, including vessel diameter, lesion length, thrombus presence, and bifurcation lesions. Time-to-wire was significantly longer in the deferred group (12.1 ± 6.9 h vs. 59.5 ± 6.8 h; Table 3).

Procedural parameters were generally balanced. However, the deferred group demonstrated superior post-wiring TIMI 3 flow (93.4% vs. 46.7%, p < 0.001), required fewer stents, and exhibited a lower incidence of no-reflow (20.8% vs. 37.1%, p = 0.003; Table 2).

Predictors of No-Reflow

Univariate analysis identified immediate PCI (OR: 2.256; p = 0.004), lesion length (OR: 1.120; p < 0.001), thrombus presence (OR: 1.817; p = 0.018), bifurcating lesions (OR: 2.385; p = 0.004), number of PTCA inflations (OR: 1.322; p = 0.007), and number of stent balloon inflations (OR: 2.518; p < 0.001) as significant predictors. In multivariable analysis, the autonomous prognostic factors identified were lesion length, thrombus burden, bifurcating lesion, immediate PCI, and stent balloon inflations (Table 4, Table 5).

ROC analysis revealed lesion length ≥ 37.75 mm as the best predictor of no-reflow (AUC = 0.765, sensitivity 72%, specificity 88.4%).

A composite risk score, the BNLTI factor (Bifurcation, Number of stent inflations, Lesion Length, Thrombus, Immediate PCI), was derived:

$$BNLTI = \sum [(0.184 \times bifurcation^{*}) + (0.137 \times stent\ inflations) + (0.016 \times lesion\ length) + (0.140 \times thrombus) + (0.175 \times \text{immediate PCI}^{*}) - 0.639]$$

(*yes = 1; no = 0)

BNLTI score demonstrated strong predictive accuracy (AUC = 0.838; p < 0.001) with a cutoff ≥ 0.389 (sensitivity 80%, specificity 77.3%).

In-Hospital and 1-Year Outcomes

In-hospital mortality was higher in the immediate PCI group, though not statistically significant (4.8% vs. 2.8%; p = 0.414). Heart failure incidence was significantly higher in the immediate group (31% vs. 17%; p = 0.007). Re-infarction and bleeding events were similar between groups (Table 5).

At one-year follow-up, all-cause mortality was significantly lower in the deferred PCI group (2.8% vs. 9%; p = 0.04). No differences were observed in cardiovascular death, reinfarction, or revascularization rates (Table 6).

Table 1: Basic characteristics of study population

Variables		PPCI N= 210	Deferred PCI N=106	Test value	P value
Age (years)		59.3 ± 9.1	61.2 ± 8.9	-1.735[t]	0.084
Gender	Male	150 (71.4%)	84 (79.2%)	2.240[x]	0.134
	Female	60 (28.6%)	22 (20.8%)		
Smoking		139 (66.2%)	59 (55.7%)	3.338[x]	0.068
Hypertension		64 (30.5%)	36 (34%)	0.396[x]	0.529
Diabetes Mellitus		110 (52.4%)	64 (60.4%)	1.820[x]	0.177
Family history of premature CAD		54 (25.7%)	36 (34%)	2.353[x]	0.125

t: student's t-test; x: chi-squared test, PPCI: primary percutaneous coronary intervention, PCI: percutaneous coronary intervention, CAD: coronary artery diseas

Table 2: Admission clinical, laboratory, electrocardiographic, and echocardiographic data of the study population

Variables		PPCI N= 210	Deferred PCI N=106	Test value	P value
Killip Class	I	200 (95.2%)	103 (97.2%)	0.666[x]	0.414
	II	10 (4.8%)	3 (2.8%)		
Admission Systolic blood pressure (mmHg)		122.2 ± 15.3	121.8 ± 15.4	0.244[t]	0.807
Admission Heart rate (beat per minute)		71.1 ± 12.0	71.3 ± 12.0	-0.158[t]	0.875
Site of myocardial infarction	Inferior	25 (11.9%)	10 (9.4%)	0.437[x]	0.804
	Lateral	27 (12.9%)	14 (13.2%)		
	Anterior	158 (75.2%)	82 (77.4%)		
P2Y12 inhibitor	Clopidogrel	40 (19%)	28 (26.4%)	2.264[x]	0.132
	Ticagrelor	170 (81%)	78 (73.6%)		
Chronic medications	BB	45 (21.4%)	24 (22.6%)	0.061[x]	0.805
	ACEI/ARBS	54 (25.7%)	32 (30.2%)	0.712[x]	0.399
Culprit vessel	RCA	23 (11%)	9 (8.5%)	0.469[x]	0.791
	LCX	29 (13.8%)	15 (14.2%)		
	LAD	158 (75.2%)	82 (77.4%)		
Haemoglobin level		12.8 ± 1.4	12.6 ± 1.4	0.997[t]	0.319
Creatinine level		1.2 ± 0.6	1.3 ± 0.7	-1.754[t]	0.080
Baseline LVEDV		106.6 ± 32.4	110.3 ± 3.2	-0.988[t]	0.324
Baseline LVESV		54.8 ± 26	53.7 ± 21.8	0.360[t]	0.719
Baseline LVEF		50.4 ± 9.3	52.6 ± 7.4	-2.135[t]	0.034

t: student's t-test; x: chi-squared test, PPCI: primary percutaneous coronary intervention, PCI: percutaneous coronary intervention, LVEDV: left ventricular end diastolic volume, LVESV: left ventricular end systolic volume, LVEF: left ventricular ejection fraction.

Table 3: PCI procedural data of the study population

Variables		PPCI N= 210	Deferred PCI N=106	Test value	P value
Time to wire cross (hours)		12.1 ± 6.9	59.5 ± 6.8	-57.488[t]	<0.001
Visualized thrombus		121 (57.6%)	60 (56.6%)	0.030[x]	0.863
Vessel ref. diameter (mm)		3.4 ± 0.4	3.3 ± 0.4	1.344[t]	0.180
Lesion length (mm)		29.5 ± 9.3	31.5 ± 8.6	-1.851[t]	0.065
Bifurcation lesion		39 (18.6%)	17 (16%)	0.310[x]	0..578
Pre-stent Nitro-glycerine		129 (61.4%)	56 (52.8%)	2.146[x]	0.143
TIMI flow after wiring	TIMI 0	24 (11.4%)	2 (1.9%)	65.581[x]	<0.001
	TIMI 1	31 (14.8%)	2 (1.9%)		
	TIMI 2	57 (27.1%)	3 (2.8%)		
	TIMI 3	98 (46.7%)	99 (93.4%)		
Number of PTCA balloon inflations		1.8 ± 1.2	1.7 ± 1.2	0.765[t]	0.445
Attempt Direct stenting		64 (30.5%)	28 (26.4%)	0.563[x]	0.453
Successful direct stenting		30 (14.3%)	10 (9.4%)	1.500[x]	0.221
Stent number	One stent	109 (51.9%)	41 (38.7%)	6.334[x]	0.042
	Two stents	99 (47.1%)	65 (61.3%)		
	Three stents	2 (1%)	0 (0.0%)		
Number of stent balloon inflations		1.9 ± 1.0	1.8 ± 0.8	0.952[t]	0.342
NC balloon use		100 (47.6%)	47 (44.3%)	0.305[x]	0.581
Post stent Nitro-glycerine use		158 (75.2%)	77 (72.6%)	0.249[x]	0.618
Volume of contrast		183.4 ± 35.1	186.7 ± 39.8	-0.717[t]	0.474
No-reflow		78 (37.1%)	22 (20.8%)	8.746	0.003

t: student's t-test; x: chi-squared test, PPCI: primary percutaneous coronary intervention, PCI: percutaneous coronary intervention, TIMI: thrombolysis in myocardial infarction, PTCA: percutaneous trans-catheter coronary angioplasty, NC: non-compliant

Table 4: Univariate and multivariate regression analysis to pick up predictors of no-reflow

Predictors of No-reflow	Univariate Odds ratio	P value	Multivariate Odds ratio	P value
Immediate PCI	2.256 (1.306-3.897)	0.004	3.481 (1.770-6.846)	<0.001
Lesion length	1.120 (1.083-1.159)	<0.001	1.108 (1.064-1.154)	<0.001
Lesion thrombus	1.817 (1.107-2.980)	0.018	2.382 (1.282-4.426)	0.006
Bifurcation culprit lesion	2.385 (1.322-4.302)	0.004	3.180(1.449-6.979)	0.004
Number of PTCA inflations	1.322 (1.078-1.622)	0.007	1.260 (0.974-1.630)	0.078
Number of stent balloon inflations	2.518 (1.901-3.334)	<0.001	2.002 (1.403-2.856)	<0.001
NC balloon post dilatation	4.510 (2.696-7.543)	<0.001	1.780 (0.919-3.450)	0.088

PCI: percutaneous coronary intervention, PTCA: percutaneous trans-catheter coronary angioplasty, NC: non-compliant

Table 5: Step wise forward condition regression analysis to pick up best predictors of no-reflow

Predictors of No-reflow	Odds ratio	P value
Lesion length	1.117 (1.073-1.162)	<0.001
Immediate PCI	3.661 (1.870-7.168)	<0.001
Lesion thrombus	2.520 (1.365-4.655)	0.003
Bifurcation culprit lesion	3.435 (1.589-7.427)	0.002
Number of stent balloon inflations	2.273 (1.642-3.146)	<0.001

PCI: percutaneous coronary intervention

Table 6: One-year follow up

	PPCI N= 210	Deferred PCI N=106	Test value	P value
In hospital mortality	10 (4.8%)	3 (2.8%)	0.666[x]	0.414
In hospital heart failure	65 (31%)	18 (17%)	7.099[x]	0.007
In hospital re-infarction	2 (0.9%)	3 (2.8%)	1.595[x]	0.207
In hospital major bleeding	1 (0.5%)	2 (1.9%)	1.490[x]	0.222
In hospital minor bleeding	1 (0.5%)	3 (2.8%)	3.123[x]	0.077
1 year all-cause mortality	19 (9%)	3 (2.8%)	4.204[x]	0.040
1 year cardiovascular mortality	10 (4.8%)	1 (0.9%)	3.057[x]	0.080
1 year myocardial infarction	2 (1%)	1 (0.9%)	0.000[x]	0.994
1 year repeat revascularization	3 (1.4%)	1 (0.9%)	0.133[x]	0.716

x: chi-squared test, PPCI: primary percutaneous coronary intervention, PCI: percutaneous coronary intervention

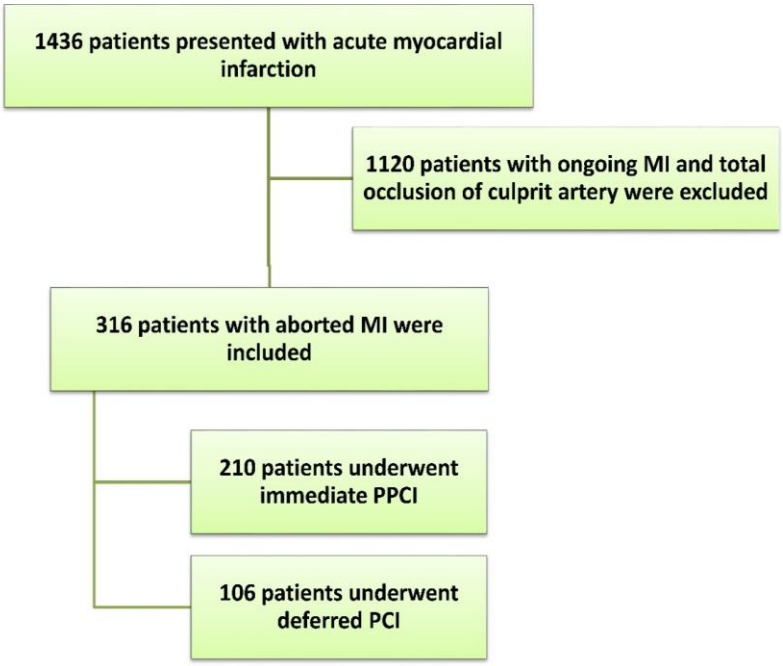

Figure 1: Flow chart of the study population.

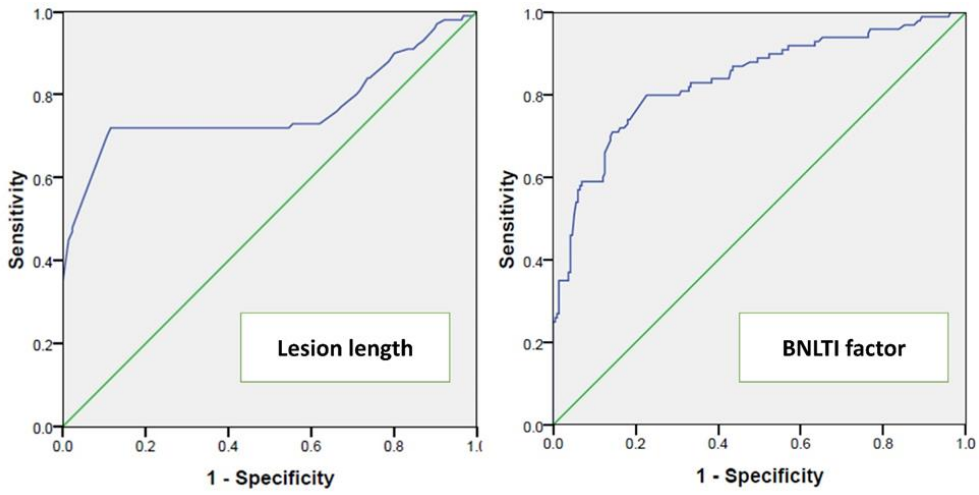

Figure 2: Receiver operating characteristic (ROC) curves for the lesion length and BNLTI factor for prediction of no-reflow in patients with abort

DISCUSSION

The no-reflow phenomenon persists as a significant procedural adverse event among individuals presenting with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI), and is correlated with heightened periprocedural morbidity and mortality [7].

Despite its clinical significance, current guideline recommendations lack clarity regarding the optimal timing of revascularization in patients presenting with aborted myocardial infarction (MI). While expert consensus supports deferring stenting in patients with a high thrombus burden [17], angiographic assessment of thrombus remains subjective and imprecise. Furthermore, accurately predicting the risk of no-reflow during primary PCI continues to pose a clinical challenge.

In this study of patients with aborted MI, deferred stenting performed 48 hours after intensive antithrombotic therapy—specifically glycoprotein IIb/IIIa inhibitor infusion—was associated with a significant reduction in the incidence of no-reflow and in-hospital heart failure, without a corresponding increase in rates of in-hospital reinfarction or major or minor bleeding complications. Multivariate analysis identified lesion length, immediate PCI, the presence of thrombus, bifurcating culprit lesions, and number of stent balloon inflations as independent predictors of no-reflow.

To facilitate individualized risk prediction, these variables were incorporated into a novel composite score—BNLTI (Bifurcation, Number of stent inflations, Lesion length, Thrombus, Immediate PCI)—which demonstrated strong predictive value for no-reflow. A BNLTI score ≥ 0.389 was associated with a sensitivity of 80% and specificity of 77.3% for predicting no-reflow in patients with aborted MI, providing clinicians with a practical and evidence-based tool for early risk stratification.

At one-year follow-up, the deferred PCI strategy was associated with significantly lower all-cause mortality compared with immediate

PCI. However, cardiovascular mortality, reinfarction, and need for repeat revascularization did not differ significantly between the two groups.

These findings align with select prior studies [14,15,24] supporting the efficacy and safety of deferred stenting in STEMI patients by reducing periprocedural complications without increasing reinfarction risk. The success of this strategy may, in part, be attributable to the availability of potent antithrombotic agents, particularly glycoprotein IIb/IIIa inhibitors [25,26]. Conversely, findings from larger trials such as the DANAMI-3–DEFER substudy [21] and MIMI (Minimalist Immediate Mechanical Intervention) trial [20] indicated no significant benefit of routine deferred stenting on infarct size, myocardial salvage index, or microvascular obstruction (MVO) as assessed by cardiac magnetic resonance (CMR). Intriguingly, the MIMI trial even suggested potential harm due to increased MVO in deferred patients.

The heterogeneity in findings across trials may reflect fundamental differences in trial design and patient selection. The DEFER-STEMI trial, for instance, focused on high-risk patients predisposed to no-reflow [24], while the MIMI trial explicitly excluded patients with high thrombus burden due to their increased risk of no-reflow [20]. DANAMI-3–DEFER applied a routine deferred strategy to all STEMI patients, regardless of thrombus burden or flow grade [16].

In contrast, the present study was limited to a well-defined subset of STEMI patients who presented with aborted MI—characterized by TIMI 3 flow on initial angiography and complete resolution of chest pain and ST-segment elevation. Importantly, no mechanical intervention (e.g., wiring, ballooning, thrombectomy) was performed at the index procedure, thereby isolating the effect of pharmacological stabilization and deferred stenting. This design differs fundamentally from trials where initial mechanical reperfusion was routinely employed to stabilize flow.

Our results also underscore the specific association between lesion length and risk of no-reflow. Patients with presumably long culprit lesions exhibited a significantly lower incidence of no-reflow when stent implantation was deferred for ≥48 hours. These findings are consistent with earlier reports identifying long lesions as a critical risk factor for no-reflow and procedural complications [7]. Unlike other trials that utilized interventions such as balloon angioplasty or thrombectomy at the initial presentation, our cohort received no mechanical manipulation prior to stenting. This unique protocol emphasizes the role of time and pharmacological therapy in stabilizing the lesion prior to PCI.

Timing of deferred PCI has varied across studies—from 4–16 hours in DEFER-STEMI, to 24–48 hours in DANAMI-3–DEFER and MIMI, to >48 hours in our protocol. While longer deferral theoretically increases the risk of reocclusion and bleeding, it may also reduce the risk of no-reflow, as large thrombi may require several days to resolve fully [27]. In DEFER-STEMI, deferring stenting reduced the incidence of no-reflow from 28.6% to 5.9%, with improvements in final TIMI flow and a 12% absolute increase in myocardial salvage index as measured by CMR [24]. Our findings mirror these benefits, with a reduction in no-reflow incidence from 37.1% in the immediate PCI group to 20.8% in the deferred PCI group, and an improvement in one-year all-cause mortality (2.8% vs. 9%).

Notably, glycoprotein IIb/IIIa inhibitors were uniformly administered to all patients in the deferred group in our study, offering a model of deferred stenting augmented by aggressive pharmacotherapy. This contrasts with DANAMI-3–DEFER, where use of IIb/IIIa inhibitors was infrequent in both arms, and may partially explain differences in outcomes. However, such aggressive antithrombotic therapy also raises concern for increased bleeding, which should be weighed against potential benefits.

Multiple prior studies support the notion that deferred stenting reduces periprocedural

complications. Carrick et al. demonstrated increased myocardial salvage and reduced no-reflow in high-risk STEMI patients receiving deferred stents [24], consistent with our observations. Despite its routine application, immediate stenting in STEMI has not been shown to improve clinical endpoints compared with deferred strategies [28]. Angiographic studies have consistently shown that deferred stenting may yield superior final TIMI flow and improved myocardial blush grades [29]. Moreover, immediate stenting has been associated with higher risk of no-reflow and intraprocedural thrombotic events in approximately 12% of STEMI patients [30]. The pathophysiology of STEMI involves acute coronary thrombosis following rupture of a vulnerable plaque. Although angiographically invisible in some cases, thrombus is present in the majority of STEMI patients [31]. Optical coherence tomography (OCT) studies have highlighted the prognostic importance of thrombus burden, often exceeding that of the underlying plaque [32]. High thrombus burden has been independently associated with major adverse cardiovascular events (MACE), stent thrombosis, and poor perfusion metrics including low myocardial blush grade and no-reflow [33].

Although thrombus burden was not formally quantified in our study, deferring stenting likely allowed for thrombus regression and vasospasm resolution, facilitating more accurate stent sizing and potentially reducing risks of malapposition. This hypothesis is supported by findings from Kim et al., who demonstrated that deferred PCI results in larger stent diameters and shorter lengths, optimizing procedural outcomes [34].

Study Limitations

This study was a single-center, non-randomized observational study, limiting the generalizability and ability to draw causal inferences. As the majority of patients were treated with primary PCI, the study does not inform outcomes in patients receiving fibrinolytic therapy. Additionally, advanced tools for assessing reperfusion quality—such as CMR or corrected TIMI frame counts—were not employed. Therefore, while the findings are compelling, they warrant confirmation in multicenter, randomized controlled trials using objective perfusion imaging endpoints.

Future recommendations

Based on our findings, we recommend considering deferred stenting in patients with aborted MI and a BNLTI score ≥ 0.389, as these individuals are at particularly high risk for no-reflow and often require greater procedural manipulation. However, validation of this approach through prospective, randomized trials is necessary before broad clinical adoption.

CONCLUSION

In patients presenting with aborted myocardial infarction and angiographic TIMI 3 flow, deferred stenting after 48 hours of glycoprotein IIb/IIIa inhibitor infusion was associated with statistically significant reduction in the risk of no-reflow and in-hospital heart failure, and improvement in one-year survival. The BNLTI factor, developed in this study, offers a practical, evidence-based tool for early identification of patients at high risk of noreflow.

Declarations

Ethics approval and consent to participate: Study approval was obtained from Institutional Review Board at Faculty of Medicine, Zagazig University, Egypt (IRB approval number 11357-26-12-2023). All patients had written informed consent to participate in the study.

Availability of data and materials: The data supporting the findings of this study are available from the corresponding author upon justified and reasonable request.

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Table Legends

Table 1: Basic characteristics of study population.
Table 2: Admission clinical, laboratory, electrocardiographic, and echocardiographic data of the study population
Table 3: PCI procedural data of the study population
Table 4: Univariate and multivariate regression analysis to pick up predictors of no-reflow.
Table 5: Step wise forward condition regression analysis to pick up best predictors of no-reflow.
Table 6: One-year follow up.

Comment [mm1]: Split of tables

Figure Legends

Figure 1: Flow chart of the study population.
Figure 2: Receiver operating characteristic (ROC) curves for the lesion length and BNLTI factor for prediction of no-reflow in patients with abort

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