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Predictors and Outcomes of No Reflow Phenomenon Post-Primary Coronary Intervention in Young Patient

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ABSTRACT

Background: Acute treatment of ST elevation myocardial infarction (STEMI) is restoration of myocardial perfusion by recanalization of the occluded vessel. **Objective:** The aim of the present study is to detect no reflow post primary percutaneous coronary intervention (PCI) in young STEMI patients and to correlate clinical, electrocardiogram, angiographic and procedural variables with no reflow.

Patients and methods: This Cohort study was conducted in the Cardiology Department, Faculty of Medicine, Zagazig University on 106 young patients with acute myocardial infarction treated with PPCI during the period from January 2021 to April 2022. The patients were divided into 2 groups according to myocardial blush; *Group (I)* which included 80 patients with normal flow, and *Group (II)* which included 26 patients with No reflow.

Results: We found that No significant difference between the 2 studied groups regarding Initial Thrombolysis in Myocardial Infarction (TIMI) flow 0 or 1. The admission EF was significantly lower among the No Reflow Group and the no reflow group significantly associated with major adverse cardiovascular events (MACE), mortality, smoking, low EF and anterior wall myocardial infarction (AWMI) were independent predictors for no reflow.

Conclusions: No reflow in young patient with STEMI could be attributed to novel predictors such as Smoking, low EF and AWMI. This phenomenon was associated with MACE and higher mortality.

Keywords: STEMI, Anterior wall myocardial infarction Percutaneous Coronary Intervention, major adverse cardiovascular events, No reflow phenomenon.

INTRODUCTION

Primary percutaneous coronary intervention (PPCI) has been established as the most effective management strategy to restore antegrade blood flow in ST-elevation myocardial infarction (STEMI) [(1)].

The no reflow phenomenon is defined as inadequate myocardial perfusion passing through a given segment of coronary circulation with no angiographic evidence of mechanical vessel obstruction [(2)].

The no reflow phenomenon occurs in a considerable number of patients with acute STEMI (25%) undergoing primary reperfusion therapy [(3)]. Experimental and clinical studies have shown that the no reflow phenomenon is associated with large myocardial necrosis and high mortality [(4)].

Suggested mechanisms for no reflow or slow flow include coronary microcirculation disturbances, such as distal embolization of thrombus and plaque debris, microvascular damage, and reperfusion injury [(5)].

No reflow is associated with larger infarct size, lower left ventricular ejection fraction (LVEF), adverse left ventricular remodeling in the late phase of myocardial infarction (MI), and increased risk of heart failure, risk of cardiac rupture, and risk of death [(6)]. Both short term and long term prognosis of no reflow are poor in humans. Malignant arrhythmias, pump failure, cardiac rupture and re-infarction are potential complications of no reflow during the immediate in-hospital course [(7)].

A number of clinical, serologic, and angiographic parameters have been shown to be associated with no reflow [(2)]. In addition, a number of treatment strategies have been tried with variable results in no reflow. Knowing the predictors or risk factors of no reflow can help prevent this dreaded complication of PPCI [(8)].

In the present study, we aimed to detect no reflow post primary PCI in young STEMI patients and to correlate clinical, ECG, angiographic, procedural variables and with no reflow.

PATIENTS AND METHODS

This Cohort study was conducted in the Cardiology Department, Faculty of Medicine, Zagazig University on 106 young patients with acute myocardial infarction treated with PPCI during the period from January 2021 to April 2022. The patients were divided into 2 groups according to myocardial blush; *Group (I)* which included 80 patients with normal flow, and *Group (II)* which included 26 patients with No reflow.

Inclusion criteria: Consecutive patients with acute myocardial infarction who had undergone PPCI were included in the study, after giving informed consent.

Exclusion criteria:

Rescue PCI. MI Patient with cardiogenic shock. Patients with coronary dissection (whether spontaneous or procedure-related). Patients in whom no stenting will be done for various reasons such as unsuitable anatomy or insignificant lesions in coronary angiogram or high thrombus burden. Previous revascularization.

Diagnosis of no reflow was defined as post PPCI Myocardial blush grade (MBG) <2 infarct related artery.

All patients in the study were subjected to full history taking and full clinical examination. Severity of heart failure was assessed according to the Killip classification. Twelve-lead ECGs (recorded at 25 mm/s

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and 10 mm/mV voltages) was obtained from all patients on admission and 60 min after PPCI and all measurements were obtained from these ECG papers. Transthoracic two-dimensional echocardiography was performed upon admission to the Coronary Care Unit (CCU) to establish left ventricular (LV) function. Laboratory investigations included blood glucose (BG), CRP, Neutrophils/lymphocyte ratio, HgA1c, Lipid profile, Troponin and CKMB level.

Coronary angiography was performed according to the standard criteria. Primary PCI should be considered in all patients with STEMI, particularly high

-risk patients in cardiogenic shock <18 h from symptoms, <75 years old and in whom fibrinolytic therapy is contraindicated or who have already received thrombolytic.

Ethical considerations:

An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistic analysis:

Data collected throughout history, basic clinical examination, laboratory investigations and outcome measures coded, entered and analyzed using Microsoft Excel software. Data were then imported into Statistical Package for the Social Sciences (SPSS version 20.0) software for analysis. According to the type of data, qualitative represented as number and percentage, and quantitative represented by mean ± SD. Difference and association of qualitative variable were examined by Chi square test (X^2). Differences between quantitative independent groups were examined by Student's t test. Independent predictor factors were identified by logistic regression. P-value was set at $\leq$0.05 for significant results and $\leq$0.001 for highly significant result.

RESULTS

Normal flow group included 56 males and 24 females, while no reflow group included 20 males and 6 females. Table 1 shows that there was no significant difference regarding age, sex, BMI, renal insufficiency, hypertension, DM, (P>0.05), while smoking and previous CAD were more frequent in no reflow group (P values 0.00and 0.05, respectively).

Table (1): Basic demographic and co-morbidity distribution among studied groups.

Variable			Normal Flow Group (N=80)	No Reflow Group (N=26)	t/ χ^2	P-value
Age (year) Mean ± SD			44.94 ± 3.62	43.60 ± 3.79	1.856	0.066
BMI (kg/m^2) Mean ± SD			32.07 ± 4.22	32.18 ± 3.76	0.138	0.890
Sex	**Male**	**N (%)**	56 (70%)	20 (76.9%)	0.46	0.51 NS
	Female	**N (%)**	24 (30%)	6 (23.1%)		
Family History		**N (%)**	21 (26.3%)	7 (26.9%)	0.005	0.95 NS
Smokers		**N (%)**	24 (30%)	19 (73.1%)	**15.1**	**<0.001 HS**
Previous CAD		**N (%)**	10 (12.5%)	6 (23.1%)	1.71	0.19 NS
Renal insufficiency		**N (%)**	11 (13.8%)	4 (15.4%)	Fisher	0.84 NS
Hypertension		**N (%)**	33 (41.3%)	12 (46.3%)	0.19	0.66 NS
Diabetes mellitus		**N (%)**	22 (27.5%)	8 (30.8%)	0.103	0.75 NS

χ^2: Chi square test.t: independent sample t test. NS: P-value >0.05 is statistically not significant. HS: P-value <0.001 is highly significant.

Table 2 showed that there is statistically significant difference between studied groups regarding admission EF and WBCs count which were significantly lower among No Reflow Group, also troponin level was statistically significant high among them.

Table (2): Laboratory and ECHO distribution between studied groups.

Variable	Normal Flow Group Mean ± SD	No Reflow Group Mean ± SD	t	P-value
FBG (mg/dL)	108.07±24.35	101.15±24.70	1.306	0.19 NS
PP2 (mg/dL)	164.01±38.61	151.26±7.20	1.120	0.27 NS
HA1C%	5.93±1.20	6.07±1.23	0.596	0.55 NS
HB (g/dL)	12.27±1.77	12.28±1.68	0.028	0.98 NS
WBCs (mcL)	9.72±1.57	12.14±1.89	**4.89**	**<0.001 HS**
PLT (mcL)	188.06±5.79	179.37±8.05	0.813	0.42 NS
HDL (mg/dL)	39.43±5.04	39.84±4.82	0.433	0.67 NS
LDL (mg/dL)	150.39±16.47	152.09±17.28	0.518	0.61 NS
Admission EF	50.81±4.73	43.94±4.09	**7.989**	**<0.001 HS**
Troponin I(ng/ml)	8.25 ± 1.12	9.56 ± 1.23	**2.71**	**0.008 S**

t: independent sample t test. NS: P-value>0.05 is not significant. HS: P-value≤0.001 is highly significant. S: P-value<0.05 is statistically significant. FBG: fasting blood glucose. PP2: two hours postprandial glucose. HbA1c: glycosylated hemoglobin. HB: hemoglobin. WBCs: white blood cells. PLT: platelet count. HDL: high density cholesterol. LDL: low density cholesterol. EF: ejection fraction.

Table 3 showed that total ischemic time and reference luminal diameter were significantly higher among No Reflow group.

Table (3): Angiographic finding of studied groups.

Variable		Normal Flow Group (N=80)	No Reflow Group (N=26)	t/ χ^2	P
Total ischemic time (Mean ± SD)		4.33 ± 0.80	6.22 ± 0.93	10.1	**<0.001 HS**
luminal diameter (Mean ± SD)		3.01 ± 0.26	3.22 ± 0.65	2.38	**0.02 S**
LAD	**N (%)**	25 (31.3%)	14 (53.8%)	4.31	**0.03 S**
Multi vessel CAD	**N (%)**	19 (23.8%)	7 (26.9%)	0.11	0.74 NS
Proximal vessel Lesion	**N (%)**	14 (17.5%)	6 (23.1%)	0.39	0.53 NS
Thrombus burden grade> 3	**N (%)**	11 (13.8%)	11 (42.3%)	9.73	**0.002 S**
Initial TIMI flow	**N (%)**	35 (43.8%)	11 (42.3%)	0.02	0.89 NS
Pre-dilatation					
Balloon angioplasty	**N (%)**	5 (6.3%)	1 (3.8%)	Fisher	0.09
Stenting after pre-dilatation		49 (61.3%)	20 (76.9%)	2.19	0.15
Direct stenting		26 (32.5%)	5 (19.2%)	1.67	0.196 NS
Post dilatation	**N (%)**	11 (13.8%)	7 (26.9%)	2.42	0.12 NS

χ^2: Chi square test. t: independent sample t test. NS: P-value >0.05 is not significant. S: P-value<0.05 is statistically significant. HS: P-value ≤0.001 is statistically highly significant.

Table 4 showed that there is statistically significant difference between the studied groups regarding MACE.

Table (4): MACE distribution among studied groups.

Variable	Group		χ^2	P-value
	Normal Flow Group N=80 (%)	No Reflow Group N=26 (%)		
MACE	11 (13.8%)	12 (46.2%)	12.1	<0.001 HS
Type of MACE				
Arrhythmias	4 (5.0%)	2 (7.7%)		0.63 NS
Acute pulmonary edema	2 (2.5%)	1 (3.8%)	Fisher	0.31 NS
Hypotension	2 (2.5%)	1 (3.8%)		0.31 NS
Death	3 (3.8%)	8 (30.8%)		<0.001 HS

χ^2: Chi square test. ** P≤0.001 is statistically highly significant.

Table 5 showed that on studying factors associated with no reflow among studied patients, smoking, low admission EF <50%, and proportion AWMI, significantly independently increase risk by 9.349, 6.301, and 2.001 folds respectively. While thrombus burden >3 non-significantly independently increase risk by 1.427 folds.

Table (5): Multivariate logistic regression for independent predictors of No reflow among studied patients.

Variable	Wald	P-value	AOR	95% C.I	
				Lower	Upper
Smoking	13.968	0.00**	9.349	6.448	13.139
LOW AdmissionEF<50	14.306	0.00**	6.301	2.162	8.561
Low WBCs count	1.008	0.112	1.25	0.98	2.45
\\High troponin level	3.771	0.03	2.24	1.13	5.22
luminal diameter	5.331	0.02	3.44	2.21	6.23
LAD	12.461	0.00**	2.001	1.214	7.053
Thrombus burden >3	1.437	0.231	2.833	0.516	15.550

AOR: adjusted odds ratio. CI: confidence interval. **P ≤0.001 is statistically highly significant.

DISCUSSION

The current study revealed that age was distributed as 44.94 (SD 3.62) and 43.60 (SD 3.79) respectively between groups with no significant difference between groups.

This agreed with **El Hefnawi *et al.*** [9] who aimed to identify the clinical, electrocardiographic, pre procedural finding that could predict slow flow/ no reflow in STEMI patients treated with PCI and to determine predictors of adverse clinical events during hospital stay and short term in slow flow /no reflow group. They showed statistically non significance difference between two groups (p >0.05) regarding age.

The understanding regarding age-related to no reflow is limited. This mechanism is probably through pre-existing micro vascular dysfunction.

Previous studies documented that increased incidence of no reflow phenomenon was related to risk factors as old age [10].

About 81.1% male patients had no reflow, while female patients were only 18.9% in our study. In previous study, the reported incidence for female patients was 25.3% [11].

This study showed no significant difference founded between studied groups hypertension and diabetes mellitus. While, previous studies documented that increased incidence of no reflow phenomenon was related to risk factors as hypertension, **Fajar *et al.*** [10] and diabetes mellitus [12].

Our results showed no significant difference between studied groups regarding laboratory results.

Refaat *et al.* [13] aimed to detect novel predictors of no reflow phenomenon and the resulting adverse long term outcomes. They confirmed the link between inflammatory mediators and no reflow development. It was noted that fibrinogen level was significantly higher (P <0.001) in no reflow group than in normal flow group.

Abdi *et al.* [14] evaluated clinical predictors of no reflow phenomenon after PCI in patients with acute STEMI, to plan a better treatment of these patients. They reported that WBC count was higher in patients with the no reflow phenomenon, which may be due to increased inflammation and aggregation of WBC in artery and myocardium. This increases the possibility of the no reflow phenomenon.

The current study revealed that total ischemic time was significantly higher among no reflow group. This agreed with **Sabin *et al.*** [15] who analyzed the factors predicting no reflow. They reported that the longer the ischemia, the more severe the no reflow.

After the prolonged cessation of coronary occlusion and the restoration of blood flow to the epicardial coronary arteries, there is sufficient structural damage to the microvasculature to prevent the restoration of normal blood flow to the cardiac myocytes [16]. The structural damage is more pronounced with longer periods of coronary occlusion [17]. No reflow appears to be a process rather than an immediate event that occurs at the moment of reperfusion. Experimental studies showed that the no reflow area increases with time after reperfusion [18].

We found that no reflow group significantly associated with Patients with Killip classes 3 and 4 **Refaat *et al.*** [13] confirmed this finding.

Previous studies documented that increased incidence of no reflow phenomenon was related to risk factors as higher KILLIP class [10]. The current study revealed no significant difference between studied groups regarding proximal vessel Lesion.

Regarding the angiographic predictors, total and proximal occlusions were associated with increased incidence of no reflow [19].

Sabin *et al.* [15] demonstrated that the presence of large lesioned vessels, especially those with an IRA diameter above 4 mm, was associated with the occurrence of no reflow. Patients with lesions that were larger than 20 mm in size were more likely to develop no reflow after primary PCI than those with lesions that were smaller than 20 mm in size. Large vessels are able to contain large amounts of plaque lipid or thrombi; the larger the lesioned vessels, the slower the flow velocity,

and the longer the target lesion, the larger the amount of thrombus and plaque burden. This would explain the high risk for slow/no reflow that was observed in these patients after primary PCI[20].

Our results showed that, no reflow group significantly associated with presence of MORE grade 3 thrombus and no significant difference between studied groups regarding Initial TIMI flow 0 or 1.

Abdi *et al.* [15] reported that, thrombus grade was a predictive factor of the no reflow phenomenon, which was higher in the case group. An increased clot volume causes higher thrombus grade and subsequently increases the possibility of no reflow phenomenon [21].

It has been documented that the pre-revascularization angiographic features of the infarct-related artery (IRA) such as scored thrombus burden can be drawn upon as a simple and efficient clinical tool in the prediction of the slow-flow or no reflow phenomenon after PCI [22].

Although some of the previous investigators have not included the effect of thrombus burden in their study, **Magro *et al.*** [23] indicated that a \higher preprocedural thrombus grade of the IRA affects the flow restoration and increases the risk of the no reflow phenomenon [24].

Alidoosti *et al*. [25] investigated the clinical, angiographic, preprocedural, and procedural characteristics associated with the no reflow phenomenon among patients undergoing primary PCI. They reported that a higher preprocedural thrombus grade was one of the independent correlates for the no-flow or slow-flow phenomenon after primary PCI for acute STEMI.

This study showed that admission EF was significantly lower among No Reflow Group. This was in line with **Refaat *et al.*** [13] who reported that the incidence of no reflow was associated with decreased LVEF on admission ($P < 0.001$).

Similarly, **Abdi *et al.*** [15] reported that LVEF was lower in patients with no reflow phenomenon. This can be explained by the severity of coronary lesion and occlusion, and subsequently, more damage to the myocardium in case group.

This study showed that no reflow group significantly associated with MACE. In agreement with **Zhou *et al.*** [19] **who** showed that no reflow is associated with high incidence of adverse clinical events. **Van Kranenburg *et al.*** [26] concluded that micro vascular obstruction is responsible for no reflow, which is an independent predictor of adverse clinical events and cardiac death at 2 years.

Ahmed Osama *et al.* [9] reported that, regarding short term outcome after 3 months in no reflow group, 2 patients had MI, 3 patients had acute heart failure, 2 patients had CV death, and two patients had stroke and 1 patient with cardiac arrest. In group without no reflow there was one patient had MI, one patient had acute heart failure. There was statistically significant difference in adverse clinical events occurred on follow up after 3 months among both studied groups.

The current study revealed that no reflow group significantly associated with mortality. Also, **Refaat *et al.*** [13] reported that, adverse long term outcomes as higher mortality was more frequent in patients with no reflow. Kaplane Meier curve revealed a significant difference between no reflow and normal groups for 6 months mortality ($P < 0.001$).

The current study revealed that, smoking, low EF and AWMI were independent predictors for no reflow. According to **Refaat *et al.*** [13] reported that old age, history of diabetes mellitus, high troponin levels, and heavy thrombus burden, all were found to be independent predictors of incidence of no reflow in STEMI patients treated with primary PCI. The logistic regression model of the study of **Abdi *et al.*** [15] showed that WBC count and thrombus grade are independent predictive factors of developing no reflow phenomenon.

In conclusion, the no reflow phenomenon after PCI in young patients with STEMI is predicted by simple clinical and angiographic features, in particular, smoking, previous CAD, low EF, high thrombus burden on baseline angiography and patients who have a large luminal diameter increased risk for no reflow development, early reperfusion and lesser period can prove to be an important strategy to decrease the incidence of no reflow.

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