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Clinical and procedural predictors and short-term survival of the patients with no reflow phenomenon after primary percutaneous coronary intervention

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ABSTRACT

Objectives: In the present study, we analysed the incidence of no-reflow phenomenon, its clinical and procedural predictors, and associated in-hospital outcomes for the patients undergoing primary percutaneous coronary intervention (PCI).
Background: No-reflow phenomenon after primary PCI is a procedural complication associated with adverse post-procedure outcomes.
Methods: Data for this study were extracted from global registry, NCDR®, the site of National Institute of Cardiovascular Disease (NICVD), Karachi from July 2017 to March 2018. The demographic, clinical, and procedural characteristics, and in-hospital outcomes were analysed for the patients with and without no-reflow after primary PCI.
Results: Of total of 3255 patients, no-reflow phenomenon was found in 132 (4.1%) patients and it was associated with significantly higher in-hospitality mortality (6.8% vs. 2.9%; $p = 0.01$), cerebrovascular accident (1.5% vs. 0%; $p < 0.001$), post procedure bleeding (2.3% vs. 0.5%; $p = 0.009$), and cardiogenic shock (3.8% vs. 1.2%; $p = 0.011$). The multivariate analysis showed advanced age [odds ratio = 1.63, 95% confidence interval 1.09–2.44, $p = 0.018$], diabetes [1.66, 1.14–2.42, $p = 0.009$], prior history of CABG [8.70, 1.45–52.04, $p = 0.018$], low pre-procedure TIMI flow grade [2.04, 1.3–3.21, $p = 0.002$], longer length of target lesion [1.51, 1.06–2.16, $p = 0.023$], and 10 fold raised troponin I [1.55, 1.08–2.23, $p = 0.018$] were the independent predictors of no-reflow.
Conclusions: In this selected group of patients, the no-reflow phenomenon after primary percutaneous coronary intervention is not that uncommon. It is associated with an increased risk of adverse post-procedure hospital course including mortality. Pathophysiology of the no-reflow phenomenon is complex and opaque, however, it can be predicted based on certain clinical and procedural characteristics.

1. Introduction

The fundamental of ST-segment elevation myocardial infarction (STEMI) treatment strategy is the earliest restoration of myocardial perfusion [1]. The percutaneous coronary intervention (PCI) is the most efficient treatment of choice for STEMI, based on good success rates in restoring blood flow and lower rates of infarction or recurrent ischemia, which significantly improves the quality of life of patients and prevent further necrosis of the myocardium [2,3]. It is also very feasible, cost effective and delivered 'in a timely fashion' [2,3]. However, a considerable proportion of patients having PCI does not necessarily translate the improvement of myocardial perfusion, despite imaging evidence that the target stenosis was revascularized. This phenomenon is known as no-reflow [4]. The no-reflow is considered a complex process characterized by multiple pathogenetic components including ischemic injury, distal atherothrombotic embolization, reperfusion injury, and susceptibility of coronary microcirculation to injury [4,5]. The various trials and studies have identified a number of predisposing factors of no-reflow phenomenon, proposed an explanative mechanisms and strategies to overcome the phenomenon in their clinical setup [6–9]. The impact of no reflow on the clinical outcome has been well documented [10]. However, several characteristics of no-reflow phenomenon remain unclear [10].

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In past studies, the incidence of no reflow varies, ranging from 2 to 44% of all STEMI patients undergoing PCI and has associated with increased mortality, ranging from 7.4 to 30.3% [11]. However, the known incidence or the factors predispose to development of no-reflow among STEMI patients undergoing primary PCI is limited [11]. Prospectively collected primary PCI register of the National Institute of cardiovascular Diseases (NICVD), Karachi, Pakistan (member of National Cardiovascular Data Registry, NCDR) offers a unique opportunity to examine the no-reflow phenomenon in a large sample of local STEMI patients undergoing primary PCI. Our objectives of the present study were to determine incidence of no-reflow phenomenon among patients presented with ST-segment elevation myocardial infarction (STEMI) undergoing primary PCI, adverse post-procedure hospital course associated with no-reflow phenomenon, and finally to determine the clinical and procedural predictors of no-reflow phenomenon.

2. Methods

2.1. Study population

The NCDR CathPCI Registry by American College of Cardiology Foundation and the Society for Cardiovascular Angiography and Interventions, collects clinical data for PCI procedures from >1000 cardiac catheterization facilities across the United States and other parts of the world. The National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan is the member of the NCDR® registry since July 2017 and the largest public sector tertiary care cardiac center in Pakistan, providing acute and specialized cardiac care to >0.78 million patients per year. Data for this study was extracted from a prospectively collected NCDR registry site of Pakistan after institutional ethical review committee approval (approval number: ERC-32/2018). The patients undergoing primary PCI for STEMI from July 1, 2017 to March 31, 2018 were evaluated in the present analysis (n = 3255). All the primary PCI procedures were performed by consultant cardiologist and periprocedural antithrombotic regimen was Aspirin, loading does of Clopidogrel, maintenance does of same drugs onwards, and unfractionated heparin according to the weight of the patient on the table.

2.2. Study variables

The demographic profile, clinical characteristics, procedural characteristics, and post procedure outcomes were obtained. The Demographic profile consists of gender, age (year), and body mass index (kg/m^2). Clinical characteristics consist of patient history of diabetes mellitus, hypertension, smoking, family history of coronary artery disease (CAD), prior myocardial infarction (MI), prior PCI, currently on dialysis, prior heart failure (HF), prior coronary artery bypass grafting (CABG), chronic lung disease, prior peripheral artery disease (PAD), prior cardiogenic shock, and prior cardiac arrest. Procedural characteristics consists of fluoroscopy times (minute), contrast volume (ml), use of intra-aortic balloon pump (IABP), need of mechanical ventilator (MV) support, access for procedure, multivessel diseased, pre-procedural thrombolysis in myocardial infarction (TIMI) flow, lesion complexity, presence of thrombus, bifurcation lesion, and lesion length. Post-procedure outcomes all cause in-hospital mortality, post-procedure MI, cardiogenic shock, heart failure, cerebrovascular accident (CVA), dialysis, transfusion, bleeding, and other vascular complications.

Acquired angiographic films were not long enough to estimate myocardial blush grading (MBG), therefore, no-reflow phenomenon was defined based on visually classified TIMI flow grade on post procedure angiograms. No reflow was defined as post-procedure TIMI flow grade of 0–II in the absence of residual coronary stenosis, dissection or spasm, which persisted at the end of the PCI procedure.

2.3. Statistical analysis

Extracted data were analysed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, US). Frequency percentage were calculated for categorical variables and mean ± standard deviation was calculated for continuous variable. Chi-square test was performed for the assessment of categorical demographic variables, clinical characteristics, procedural characteristics, and post procedure outcomes by patients groups based on no-reflow phenomenon. Normality of distribution on continuous variables was assessed by applying Kolmogorov-Smirnov (KS) test and independent sample *t*-test or Mann-Whitney *U* test was performed to compare between patients groups based on no-reflow phenomenon. Statistically significant demographic variables, clinical characteristics, and procedural characteristics were taken as predictors of no re-flow phenomenon and multivariate logistic regression analysis was performed. Regressors of multivariate logistic regression model include advanced age (>65 years), female gender, diabetes, hypertension, smoking, prior coronary artery bypass grafting, pre-procedure TIMI flow grade (0–I), increased creatinine (≥1.3 mg/dl), longer lesion length (>20 mm), raised troponin I (10 fold), decreased LVEF (<40%), intra-aortic balloon pump (IABP) used, and cardiogenic shock at start of PCI. Predictive strength of each variable expressed in terms of odds ratio (OR) and its 95% confidence interval (CI) and associated *p*-value. *p*-Value < 0.05 was the criteria for statistical significance throughout the analysis.

3. Results

3.1. Demographic and clinical characteristics

Of 3255 patients with STEMI undergoing PCI, male patients were 82.7% (2692) of the total sample, mean ± standard deviation (SD) of patient's age at the time of procedure was 55.2 ± 11.16 years and body mass index (BMI) was 25.95 ± 4.58 kg/m^2. For majority of the patients, 46.2% (1505), as per the Canadian Cardiovascular Society (CCS) grading of angina pectoris, at presentation patients were classified as no symptoms no angina, while, CCS I through IV were observed in 6.3% (206), 12.1% (393), 18.3% (596), and 17.1% (555) respectively. Plain old balloon angioplasty (POBA) was performed in 7.3% (236) patients and type of stent was drug eluting stent (DES) for 50.1% (1631) patients and bare metal stent (BMS) for remaining 42.6% (1388) patients. Pre-procedure TIMI flow grade was 0 in 58.1% (1890), I in 10.3% (334), II in 19.2% (625), and III in remaining 12.5% (406) of the patients. Post-procedure TIMI flow grade was 0 was observed in 0.8% (28), I in 0.8% (27), II in 2.5% (80), and III in remaining 95.9% (3123) of the patients. Post-procedure no-reflow phenomenon was developed in 132 (4.1%) patients.

Patients who developed no-reflow were few years older than the patients with normal flow. No-reflow phenomenon was significantly higher among female, diabetic, hypertensive, non-smokers, and patients with prior CABG. Demographic and clinical characteristics of the patients are presented in Table 1.

3.2. Pre- and peri-procedural characteristics

Pre- and peri-procedural characteristics are presented in Table 2. Mean fluoroscopy times were 14.01 ± 8.39 min and mean amount of contrast used was 138.87 ± 46.81 ml. A majority of the procedures, 64.4% (2097), were performed through femoral access. High/C lesions were observed in 44.9% (1462) patients. No-reflow phenomenon was significantly more common among the patients with initial TIMI flow grade of 0–I, elevated creatinine, 10 fold increased troponin I, longer lesion length, decreased left ventricular ejection fraction, cardiogenic shock, and for the patients in whom IABP was used.

3.3. In-hospital outcomes

Total post-procedure in-hospital mortality rate was 3.0% (99) and other post procedure complications and in-hospital outcomes for patients with post -procedure normal flow and no-reflow are presented in Table 3. Significant differences in in-hospital post procedural outcomes were observed in no-reflow group of patients. Post procedure mortality was higher in no-reflow group of patients (6.8% vs. 2.9%; $p = 0.01$). Rate of post-procedure cerebrovascular accident (CVA) was significantly higher for the patients with no-reflow (1.5% vs. 0%; $p < 0.001$). Similarly, post procedure bleeding complication (2.3% vs. 0.5%; $p = 0.009$) and cardiogenic shock (3.8% vs. 1.2%; $p = 0.011$) were more common in no-reflow group of patients. Significantly low left ventricular ejection fraction (40.67 ± 15.22% vs. 43.73 ± 11.05%; $p = 0.002$) was observed among patients who developed no-reflow.

3.4. Predictors of no-reflow

Table 4 showed multivariate analysis for the predictors of no-reflow based on univariately significant demographic, clinical, and procedural characteristics. Advanced age, diabetes mellitus, prior history of coronary artery bypass grafting (CABG), pre-procedure TIMI flow grade of 0–I, longer lesion length, and raised troponin I were found to be significant predictors of no-reflow phenomenon with odds ratio of 1.63 (95% CI 1.09–2.44; $p = 0.018$), 1.66 (95% CI 1.14–2.42; $p = 0.009$), 8.70 (95% CI 1.45–52.04; $p = 0.018$), 2.04 (95% CI 1.3–3.21; $p = 0.002$), 1.51 (95%

Table 1
Demographic and clinical characteristics.

Characteristics	Total	Post-procedure TIMI Flow		
		Normal flow	No-reflow	*p*-Value**
N	3255	3123	132	–
Clinical characteristics				
Age (mean ± SD) years	55.2 ± 11.16	55.07 ± 11.1	58.4 ± 12.02	<0.001
Advanced age (>65 years)	584 (17.9%)	547 (17.5%)	37 (28%)	0.002
BMI (mean ± SD) kg/m^2	25.95 ± 4.58	25.93 ± 4.52	26.32 ± 5.69	0.342
Female gender	563 (17.3%)	530 (17%)	33 (25%)	0.017
Diabetes	861 (26.5%)	808 (25.9%)	53 (40.2%)	<0.001
Hypertension	1421 (43.7%)	1348 (43.2%)	73 (55.3%)	0.006
Smoking	817 (25.1%)	794 (25.4%)	23 (17.4%)	0.038
Positive family history	118 (3.6%)	110 (3.5%)	8 (6.1%)	0.126
Currently on dialysis	9 (0.3%)	9 (0.3%)	0 (0%)	0.537
Chronic lung disease	17 (0.5%)	16 (0.5%)	1 (0.8%)	0.702
Prior peripheral artery disease (PAD)	9 (0.3%)	9 (0.3%)	0 (0%)	0.537
Past medical history				
Myocardial infarction (MI)	216 (6.6%)	211 (6.8%)	5 (3.8%)	0.180
Percutaneous coronary intervention (PCI)	95 (2.9%)	90 (2.9%)	5 (3.8%)	0.545
Heart failure (HF)	14 (0.4%)	14 (0.4%)	0 (0%)	0.441
Coronary artery bypass grafting (CABG)	7 (0.2%)	5 (0.2%)	2 (1.5%)	<0.001
Cardiogenic shock	4 (0.1%)	4 (0.1%)	0 (0%)	0.681
Cardiac arrest	7 (0.2%)	6 (0.2%)	1 (0.8%)	0.170

** *p*-Values are based on *t*-test or Mann–Whitney *U* test for continuous variables and chi-square test for categorical variables.

CI 1.06–2.16; $p = 0.023$), and 1.55 (95% CI 1.08–2.23; $p = 0.018$) respectively.

4. Discussion

Current study was performed to assess the incidence of no-reflow phenomenon after primary PCI, its clinical and procedural predictors, and associated in-hospital outcomes. Main findings of the were, no-reflow phenomenon was observed in 4.1% of the patients after primary PCI, it was found to be associated with adverse post-procedure hospital course including mortality, cerebrovascular accident, bleeding, and cardiogenic shock, and advanced age, diabetes mellitus, prior history of CABG, low baseline TIMI flow grade, 10 fold increased troponin I, and longer lesion length were found to be in independent predictors of no-reflow phenomenon after primary PCI.

This is the first largest study on no-reflow phenomenon among Pakistani patients with STEMI undergoing primary PCI. In our study the incidence of no-reflow was 4.1% (132) whereas, globally incidence of no-reflow phenomenon reported ranging from 2 to 44% among patients undergoing primary PCI for STEMI [11,12]. The Ndreppa et al. reported no-reflow in 29% of 1406 STEMI patients undergoing primary PCI [10], whereas, in HORIZONS-AMI trial, 10.2% of the patients developed no-reflow phenomenon [13]. The genesis of no-reflow after primary PCI is multifactorial pathophysiological process. The possible mechanisms of no-reflow include microcirculation, reperfusion injury and microthrombosis [14].

No-reflow phenomenon is a common and severe complication strongly linked with poor prognosis after PCI [6,7], as registered in the current study. We observed a significant association of no reflow with post procedure in-hospital mortality, cerebrovascular accident (CVA), bleeding complication, cardiogenic shock, and low left ventricular ejection fraction. The poor prognosis with no-reflow is due to adverse left ventricular remodeling, larger infarct sizes, and reduced left ventricular systolic function. In various studies it has been the keen observation that the development of the post-procedure adverse clinical outcomes in patients with STEMI are strongly and adversely influenced by the

Table 2
Pre and periprocedural characteristics.

Characteristics	Total	Post-procedure TIMI Flow		
		Normal Flow	No-reflow	*p*-Value**
N	3255	3123	132	–
Pre-procedure TIMI (0 to I)	2224 (68.3%)	2116 (67.8%)	108 (81.8%)	<0.001
Creatinine (mean ± SD) mg/dl	0.89 ± 0.9	0.88 ± 0.91	0.98 ± 0.57	0.253
Increased creatinine (≥1.3 mg/dl)	461 (14.2%)	432 (13.8%)	29 (22%)	0.009
Troponin I (mean ± SD) ng/ml	23.73 ± 22.32	23.53 ± 22.32	27.81 ± 21.96	0.031
Raised troponin I (10 fold)	981 (30.1%)	928 (29.7%)	53 (40.2%)	0.01
Lesion length (mean ± SD) mm	19.24 ± 8.53	19.18 ± 8.46	20.81 ± 9.86	0.031
Longer lesion length (>20 mm)	1094 (33.6%)	1035 (33.1%)	59 (44.7%)	0.006
Left ventricular ejection fraction (LVEF %)	43.2 ± 10.05	43.3 ± 9.96	40.29 ± 12.14	<0.001
Decreased LVEF (<40%)	496 (15.2%)	467 (15%)	29 (22%)	0.028
Fluro time (mean ± SD) min	14.01 ± 8.39	13.98 ± 8.37	14.73 ± 8.83	0.314
Contrast volume (mean ± SD) ml	138.87 ± 46.81	138.68 ± 46.84	143.4 ± 45.99	0.256
Intra-aortic balloon pump (IABP) used	13 (0.4%)	11 (0.4%)	2 (1.5%)	0.038
Mechanical ventilator support	2 (0.1%)	2 (0.1%)	0 (0%)	0.771
Multivessels diseased	1868 (57.4%)	1785 (57.2%)	83 (62.9%)	0.193
Cardiogenic shock	99 (3%)	91 (2.9%)	8 (6.1%)	0.039
High-C lesion	1462 (44.9%)	1395 (44.7%)	67 (50.8%)	0.168
Presence of thrombus	2580 (79.3%)	2474 (79.2%)	106 (80.3%)	0.763
Bifurcation lesion	835 (25.7%)	796 (25.5%)	39 (29.5%)	0.296

** *p*-Values are based on *t*-test or Mann–Whitney *U* test for continuous variables and chi-square test for categorical variables.

Table 3
Post procedure in-hospital outcomes by patients with no-reflow and normal flow.

Complications	Total	Post-procedure TIMI Flow		
		Normal flow	No-reflow	*p*-Value**
N	3255	3123	132	–
Mortality	99 (3.0%)	90 (2.9%)	9 (6.8%)	0.01
Myocardial infarction	17 (0.5%)	15 (0.5%)	2 (1.5%)	0.106
Cardiogenic shock	43 (1.3%)	38 (1.2%)	5 (3.8%)	0.011
Heart failure	29 (0.9%)	26 (0.8%)	3 (2.3%)	0.085
Cerebrovascular accident	3 (0.1%)	1 (0%)	2 (1.5%)	<0.001
Dialysis	5 (0.2%)	4 (0.1%)	1 (0.8%)	0.070
Other vascular complications	1 (0%)	1 (0%)	0 (0%)	0.837
Transfusion	3 (0.1%)	3 (0.1%)	0 (0%)	0.722
Bleeding	19 (0.6%)	16 (0.5%)	3 (2.3%)	0.009
Post procedure LVEF (%)	43.56 ± 11.3	43.73 ± 11.05	40.67 ± 15.22	0.002

** *p*-Values are based on *t*-test or Mann–Whitney *U* test for continuous variables and chi-square test for categorical variables.

procedural complications during primary PCI such as no-reflow. Ndrepepa et al. [10] associated no reflow with poor outcomes after six months of reperfusion, such as, worse left ventricular ejection fraction (LVEF), larger infarct size, and reduced myocardial salvage and increased risk of mortality after one year of reperfusion.

In the current study, univariate analysis showed that age, female gender, diabetes, hypertension, prior history of CABG, slow pre-procedure TIMI (0 to I), increased creatinine (≥1.3 mg/dl), raised troponin I (10 fold), longer lesion length (>20 mm), decreased LVEF (<40%), pre and perioperative used of intra-aortic balloon pump (IABP), and PCI in cardiogenic shock were positively associated with no reflow. However, there was a negative association of smoking with no reflow, which can be partly explained by the fact that the smokers were significantly younger than non-smokers in our study. The multivariate analysis showed advanced age (>65 years), diabetes, prior history of coronary artery bypass grafting (CABG), low pre procedure TIMI flow grade (0–I), longer length of target lesion (>20 mm), and raised troponin I (10 fold) at baseline were the independent predictors of no reflow.

Elderly, and STEMI patients carries a greater thrombus and atherosclerotic plaque burden [15]. Advance age is well documented as independent predictor of no reflow in past studies [6,12,14]. In present study, patients who developed no reflow were significantly older, 58.4 ± 12.02 vs. 55.07 ± 11.1 years ($p < 0.001$), and proportion of advanced age (>65 years) were higher among no reflow subgroup as compared to patients with normal flow (28% vs. 17.5%, $p = 0.002$) and on multivariate analysis, risk of no reflow was 1.63 times higher in patients above 65 years of age.

Table 4
Predictors of no-reflow (multivariate logistic regression analysis).

Predictors	OR	95% CI for OR	*p*-Value
Advanced age (>65 years)	1.63	1.09–2.44	0.018
Female gender	1.33	0.87–2.05	0.189
Diabetes	1.66	1.14–2.42	0.009
Hypertension	1.28	0.88–1.86	0.198
Smoking	0.79	0.49–1.27	0.332
Prior coronary artery bypass grafting	8.70	1.45–52.04	0.018
Pre procedure TIMI flow grade (0–I)	2.04	1.3–3.21	0.002
Increased creatinine (≥1.3 mg/dl)	1.36	0.87–2.12	0.172
Longer lesion length (>20 mm)	1.51	1.06–2.16	0.023
10 fold increased troponin I	1.55	1.08–2.23	0.018
Decreased LVEF (<40%)	1.46	0.94–2.25	0.089
Intra-aortic balloon pump (IABP) used	3.30	0.63–17.3	0.158
Cardiogenic shock at start of PCI	1.95	0.89–4.27	0.095
Constant	0.01	–	<0.001

Dependent variable No Reflow [yes].
Hosmer and Lemeshow Test (chi-square = 11.439, df = 8, $p = 0.178$).

Diabetes mellitus is a well-established risk factor of atherosclerotic cardiovascular disease (ASCVD), a meta-analysis by Fajar et al. [12] reported diabetes and elevated baseline blood glucose level as significant predictors of no-reflow phenomenon. Similarly, Iwakura et al. [16] reported blood glucose level as an independent prognostic factor for no reflow phenomenon in patients with acute myocardial infarction (AMI). In current study, diabetes was found to be the independent predictors of no-reflow phenomenon and no-reflow was 1.66 folds higher in the patients with diabetes at baseline.

Total occlusion may be a sign of a fully developed thrombus and prolonged ischemia, and causes lower pre-procedure TIMI flow. The pre-procedure TIMI flow grade is one of the significant factors associated with the no-reflow phenomenon [6,10,12,14,17]. Indeed, in the present study, patients who developed no-reflow after primary PCI had lower degrees of pre-procedural TIMI blood flow grade (0–I) and on multivariate analysis, patients with pre-procedure TIMI flow grade of 0 or I had >2-fold higher risk of no-reflow phenomenon. Kirma et al. [18] and De Luca et al. [19] showed better procedure success and lesser degrees of catheterization complications after primary PCI among patients with higher TIMI flow at baseline. In general, high baseline TIMI flow grade suggests smaller thrombus burden and spontaneous lysis of the thrombus indicating good patency of the culprit artery [14,17].

Pathophysiologically, lesion characteristics, such as length of lesion, type of occlusion, and thrombus burden, can have a profound role in the development of no-reflow phenomenon [20]. Longer lesions indicate high coronary plaque burden in the atherosclerotic artery and various studies have reported it as an important and independent predictor of the no-reflow phenomenon [6,12,14,17]. The findings of the present study revealed that type of occlusion (baseline TIMI flow) and longer length of culprit lesion were the independent predictors of no-reflow phenomenon and no-reflow was 1.5 folds higher in the patients with lesion lengths above 20 mm.

The findings of the present study revealed that prior history of coronary artery bypass grafting and baseline troponin I rise of 10 fold upper limit of the normal range independently predict no-reflow phenomenon with multivariate logistic regression odd ratios of 8.70 and 1.55 respectively. However, in our study, female gender, hypertension, smoking, increased creatinine at baseline, decreased LVEF at baseline, pre and periprocedural use of intra-aortic balloon pump (IABP), cardiogenic shock at start of procedure failed to attain the required statistical significance on multivariate analysis for the prediction of no-reflow phenomenon.

Other independent predictors of no-reflow reported in past studies are delayed reperfusion [6,12,14,17], thrombus grade [7,12,14,17], intra-aortic balloon pump (IABP) use [14], white blood cell (WBC) count [7], C-reactive protein [10], previous myocardial infarction [10], and low systolic blood pressure on admission [14]. A meta-analysis by Fajar et al. [12] further reported that male gender, hypertension, family history of coronary artery diseases (CAD), killip class above I, elevated creatinine, increased heart rate, elevated peak CK, collateral flow, multi-vessel disease, decreased LVEF, and reference luminal diameter. In contrary to the Fajar et al. [12] we observed association of female gender with no-reflow phenomenon, while, the finding of our study regarding the association of no-reflow with hypertension, increased creatinine, decreased LVEF, and intra-aortic balloon pump (IABP) were align with Fajar et al. [12].

This study is the first largest study on our local population and in South Asian belt. Nevertheless, study had certain limitations. Due to observational nature it was possible for us to evaluate the incidence rate of no-reflow phenomenon, however, causal link of no-reflow phenomenon and long-term post procedural outcomes could not be established, and only immediate in-hospital outcomes were evaluated.

5. Conclusion

The findings of the present study indicated that the no-reflow phenomenon after primary percutaneous coronary intervention (PCI)

would be predictable and is not that uncommon. It is associated with an increased risk of adverse post-procedure hospital course including mortality, cardiogenic shock, cerebrovascular accident, and bleeding. Pathophysiology of the no-reflow phenomenon is complex and opaque, however, independent clinical and procedural predictors include advanced age, diabetes mellitus, prior history of coronary artery bypass grafting (CABG), low baseline TIMI flow grade, 10 fold increased troponin I, and longer lesion length. There is an increasing need for continuation of research to better understand, and prevent this unique phenomenon in patients with STEMI undergoing PCI.

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Declaration of Competing Interest

None to declare.

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