European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 08, Issue 03, 2021

Prediction and Short-Term Effects of Slow Flow and No Reflow Post Primary Percutaneous Coronary Intervention

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Abstract

Background:In a large number of primary percutaneous coronary intervention patients, angiographic non-reflow and slow flow phenomena are observed in recent developments in interventional equipment and techniques (PCI). In ST-Segment Elevation Myocardial Infarction (STEMI), we have investigated clinical, electrocardiographic preoperative findings that could predict a slow flow / no-reflow in patients treated with PCI, as well as predictor and effect of slow flux / no-reflow during hospital stays and short-term results.

Aim:Identifying the pre-procedure, clinical, electrocardiographic findings that may predict a slow flow/no-reflow in PCI-treated STEMI patients and identifying adverse clinical event predictors while in the hospital and the short-term in a slow flow/no-reflow population.

Patients and Methods:The study included 72 patients divided into 2 groups, slow-flow / non-reflow group I and normal-flow group II. All were monitored for three months after PCI.

Results:RWPT also indicates an important indicator for non-PCI reflow after multivariate analysis QRST has been applied to PCI, before PCI. Our analysis has shown a statistically significant difference in the short-term outcomes after three months between the two groups tested.QRST parameter before and after PCI and RWPT before PCI were statistically significant predictors for short-term impact but there was no statistically significant difference among both studied groups regarding angiographic findings of the lesion.

Conclusion:Our study showed that initial coronary patenting is correlated with the immediate post-PCI TIMI flow when angiographic is diagnosed. TIMI baseline 0 grade before primary PCI. Furthermore, no-reflow phenomena pathogenesis is complicated and complex. Given our recent research, patients with no reflux following primary PCI can be classified with simple clinical and angiographical characteristics.R-wave peak time (RWPT) is closely related to the production of NR and a major indicator of adverse clinical conditions in the hospital and follow-up.

Keywords:ST-Segment Elevation Myocardial Infarction, Percutaneous Coronal Interference (PCI), R Wave peaked time (SWPT) (STEMI).

Introduction

Significant morbidity and mortality related. Following the latest guidelines, diagnostic angiography and a percutaneous coronary intervention are the standard treatments for STEMI (PCI) in ST-elevation myocardial infarction (STEMI)(**1**).

Primary PCI refers to an emergent coronary angiography strategy accompanied by an angioplasty coronary artery stent, without prior thrombolytic therapy administered **(2).**

Patenting of the infarct-related arteries does not necessarily mean restoring regular cardiac blood flow, as epicardial coronary artery reperfusion is accomplished in some patient groups but with no myocardial reperfusion following primary PCI **(3).**

A serious problem after the percutaneous coronary intervention is the sluggish flow/no-reflow phenomenon (PCI). In the presence of a patent epicardial coronary artery, myocardial hypofusion is established **(4).**

No-reflow is a multifactorial phenomenon and five mechanisms have been recognized to be responsible for no-reflow; (1) pre-existing microvascular obstruction, (2) distal micro-thrombo-embolization, (3) ischemic injury, (4) reperfusion injury, and (5) individual susceptibility **(5).**

The slow flux/no-reflow affects clinical outcomes in patients suffering from AMI. Despite its relatively low level of occurrence, it is primarily associated with increased mortality or ventricular restructure **(6).**

No reflux phenomena can be seen in the absence of a dissection, thrombus, spasm, or high-grade residual stenosis at the initial lesion site by an acute decrease in coronary flow (thrombolysis in myocardial infarction [TIMI] flow grade=0-1). The "slow flux" is usually called a lower flow degradation (TIMI score=2). 2 grades of TIMI flow of 2 or less **(7).**

Slow reflow is detected as myocardial blush 1 or 2, whereas no-reflow is myocardial blush equals zero **(8).**

Patients and Methods

This study was designed as a cohort study and permitted at the cardiology department, Zagazig University Hospital (ZUH), and National Heart Institute (NHI) from March 2020 to June 2020.

The study included STEMI patients who underwent PCI and they were divided into two groups,

Group I included STIMI patients with slow flow/no-reflow as evident by TIMI flow grade and MBG.The no-reflow phenomenon is manifested by an acute reduction in the coronary flow (thrombolysis in myocardial infarction [TIMI] flow grade=0–1) in the absence of dissection, thrombus, spasm, or high-grade residual stenosis at the original lesion site. Lesser degrees of flow impairment (TIMI score=2) are generally referred to as “slow flow". Slow reflow is detected as myocardial blush 1 or 2, whereas no-reflow is myocardial blush 0, **Group II** included STIMI patients with the normal coronary flow as evident by TIMI flow grade 3 and MBG grade 3.

Patients who were included were patients with STEMI who underwent PCI. STEMI is defined as an increase in troponin I above 1 ng/mL, a new ST-segment elevation as measured at the J point, should be found in two contiguous leads and be ≥0.25 mV in men below the age of 40 years, ≥0.2 mV in men over the age of 40 years, or ≥0.15 mV in women in leads V2–V3 and/or

≥0.1 mV in other leads (in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB) pattern.

Exclusion criteria were as follows, Patients with previous revascularization, unsuccessful primary PCI, presence of mechanical complications such as dissection, and severe renal or hepatic diseases.
All patients were subjected to a detailed history, including CAD risk factors, physical examination including Killip class, Electrocardiography (ECG) within 10 minutes of first medical contact to detect ST-segment and T wave abnormalities.

Clinical examination of all patients was done including vital signs, admission systolic blood pressure, heart rate (HR), and duration of chest pain were recorded.

Cardiac examination was done to assess the Killip classification for each patient, Patients were ranked by Killip class in the following way, Killip class I (included individuals with no clinical signs of heart failure), Killip class II (included individuals with rales or crackles in the lungs, an S3, and elevated jugular venous pressure), Killip class III (described individuals with frank acute pulmonary edema) and Killip class IV (described individuals in cardiogenic shock or hypotension and evidence of peripheral vasoconstriction (oliguria, cyanosis or sweating).

Electrocardiography:Twelve-lead ECG (recorded at a speed of 25 mm/s and a voltage of 10 mm/mV) was obtained from all patients at admission and 60 min after PCI and all measurements were obtained from these ECG papers. Pre-procedural and post-procedural (at 60 min) were performed by two independent cardiologists blinded to other patients' data. QRSD and RWPT were measured from the beginning of the QRS complex to the J point and from the beginning of the QRS complex to the R-peak, respectively; the average of three consecutive beats from V5 to V6 leads in anterior STEMI, leads II and AVF in inferior STEMI, and leads I and AVL in high lateral STEMI that had the longest duration was recorded. The durations were given as milliseconds.

Coronary angiography: Coronary angiography was done to detect TIMI flow grade before and after primary PCI.Thrombolysis in myocardial infarction (TIMI) flow before and after the procedure **(9)**: **Grade 0** (no perfusion): There is no antegrade flow beyond the point of occlusion. **Grade 1** (penetration without perfusion): The contrast material passes beyond the area of obstruction but "hangs up" and fails to opacify the entire coronary bed distal to the obstruction for the duration of the angiographic filming sequence. **Grade 2** (partial perfusion): The contrast material passes across the obstruction and opacifies the coronary bed distal to the obstruction. However, the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance from the distal bed (or both) is perceptibly slower than its entry into or clearance from comparable areas not perfused by the previously occluded vessel (e.g. the opposite coronary artery or the coronary bed proximal to the obstruction).

The TIMI thrombus scale(10)was performed, **Grade 0**: no angiographic evidence of thrombus, **Grade 1**: angiographic features suggestive of thrombus:decreased contrast density, haziness of contrast, irregular lesion contour, a smooth convex meniscus at the site of a total occlusion Suggestive, but not firmly diagnostic of thrombus, **Grade 2:** definite thrombus present in multiple angiographic projections with marked irregular lesion contour with a significant filling defect-the thrombus' greatest dimension is <1/2 vessel diameter.

Grade 3: definite thrombus appears in multiple angiographic views: Greatest dimension from >1/2 to <2 vessel diameters, **Grade 4**: definite large size thrombus present: Greatest dimension >2 vessel diameters, and **Grade 5**: definite complete thrombotic occlusion of a vessel: A convex margin that stains with contrast, persisting for several cardiac cycles.

Myocardial blush grade scoring (MBG): where angiographic myocardial blush was graded densitometrically based on visual assessment of relative contrast opacification of the myocardial territory subtended by the infarct vessel with epicardial density **(11)**

MBG score 0: Absence of contrast opacification in the myocardial zone, **MBG score 1:** Minimal contrast opacification or persistent stain without washout. The dye staining is present on the next injection (approximately 30 seconds between injections), meaning failure to exit the microvasculature; **MBG scores 2:** Reduced but evident blush in the infarct zone compared to the contralateral noninvolved epicardial vessel(s); **MBG score 3:** normal entry and exit of dye from the microvasculature. The opacification of the myocardium clears normally at the end of the washout phase, similar to that in the non-involved territory.

All procedural details were recorded as regards the use of balloon predilatation, use of a stent.

Follow-Up: was doneduring the hospital stay and for 3 months post PCI by calling patients or there relevant to detect the clinical events.

Statistical Analysis:

According to the type of data qualitative representation was done as number and percentage, the quantitative group wasrepresented by mean ± SD, the following tests were used to test differences for significance; difference and association of qualitative variable by Chi-square test (X2). Differences between quantitative multiple by ANOVA. P-value was set at <0.05 for significant results &<0.001 for high significant result. All the data collectedthroughout the study were analyzed usingStatistical Package software for analysis (SPSS version 20.0).For all mentioned statistical tests are done, the threshold of significance was fixed at a 5% level (*P*-value). *P*-value of > 0.05 indicates non-significant results. *P*-value of < 0.05 indicates significant results, the smaller the *P*-value obtained the more significant are the results.

Results

The demographic and clinical characteristics of the studied groups are shown in **(Table 1).** In group I the mean age was 56.4 ± 12.4, most of them were male (88.9%), 27.8% of patients were diabetic, 41.7 were hypertensive, 36.1 were dyslipidemic, 13.9 has a positive family history. The mean BMI 27.4 ± 2.21, the mean of HR, SBP, and DBP were 78.8 ± 6.77, 132.2 ± 18.3, and 83.8 ± 8.9 respectively. In group II the mean age was 56.2 ± 11.8, most of them were male (91.7%), 33.3% of patients were diabetic, 41.7 were hypertensive with a mean BMI 27.8 ± 1.97, 36.1% of patients were dyslipidemic, 13.9% of patients had a positive family history of

CAD. The mean of HR, SBP, and DBP were 78.1 ± 6.97, 129.8 ± 16.7, and 83.1 ± 8.4 respectively.There was no statistically significant difference between both studied groups regarding demographic data and clinical characteristics (p>0.05)**(Table 1).**

The mean of CK was 1391.6 ± 840.6 and 1390.8 ± 786.8 in patients with slow flow /no-reflow and patients with normal flow respectively. The mean of CK-MB was 191.9 ± 76.8 and 286.1 ± 65.7 in patients with slow flow /no-reflow and patients with normal flow respectively. The mean of Creatinine was 1.1 ± 0.27 and 1.12 ± 0.26 in patients with slow flow /no-reflow and patients with normal flow respectively. The mean of Troponin was 1 ± 0.0 in both groups there was no statistically significant difference between studied groups regarding laboratory findings (p >0.05)**(Table 2).**

There was no statistically significant difference between both studied groups regarding echo-cardio graphic parameters (p >0.05).

(Table 3).

There was no statistically significant difference between both studied groups regarding door to balloon and characters of the lesion (p >0.05)**(Table 4).**

We found that the RWPT parameter had a higher sensitivity in detecting cases with expected no-reflow after PCI (100% versus 86.1% of ST-elevation parameter), with higher test accuracy than the ST elevation parameter (90.3% versus 84.7% respectively)**(Table 5).**

Table 1: Demographic data and clinical characteristics of the studied groups.

		Slow reflow/ no reflow N=36	**Normal flow N=36**	**test**	**P**
Age/years (X ±SD)		**56.4 ± 12.4**	**56.2 ± 11.8**	**0.08**	**> 0.05 NS**
Gender	Male N (%)	**32 (88.9%)**	**33 (91.7%)**	Fisher	**> 0.05 NS**
	Female N (%)	**4 (11.1%)**	**3 (8.3%)**		
Smoking N (%)		**28 (77.8)**	**28 (77.8)**	0.00	**> 0.05 NS**
DM N (%)		**10 (27.8)**	**12 (33.3)**	**0.26**	**> 0.05 NS**
Hypertension N (%)		**15 (41.7)**	**11 (30.6)**	0.96	**> 0.05 NS**
Dyslipidemia N (%)		**13 (36.1)**	**13 (36.1)**	0.00	**> 0.05 NS**
+ve Family history of CAD N (%)		**5 (13.9)**	**5 (13.9)**	0.00	**> 0.05 NS**
Duration of chest pain (h) (X ±SD)		**4.61 ± 2.2**	**4.39 ± 2.1**	0.44	**> 0.05 NS**
BMI(X ±SD)		**27.4 ± 2.21**	**27.8 ± 1.97**	0.73	**> 0.05 NS**

HR (X ±SD)	**78.8 ± 6.77**	**78.1 ± 6.97**	0.36*	**> 0.05 NS**
SBP (X ±SD)	**132.2 ± 18.3**	**129.8 ± 16.7**	0.58*	**> 0.05 NS**
DBP(X ±SD)	**83.8 ± 8.9**	**83.1 ± 8.4**	0.34*	**> 0.05 NS**

NS: P-value>0.05 is not significant X: Mean, SD: Standard deviation, BMI: Body massindex, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic Blood Pressure, DM: Diabetes Mellitus, CAD: Coronary artery disease

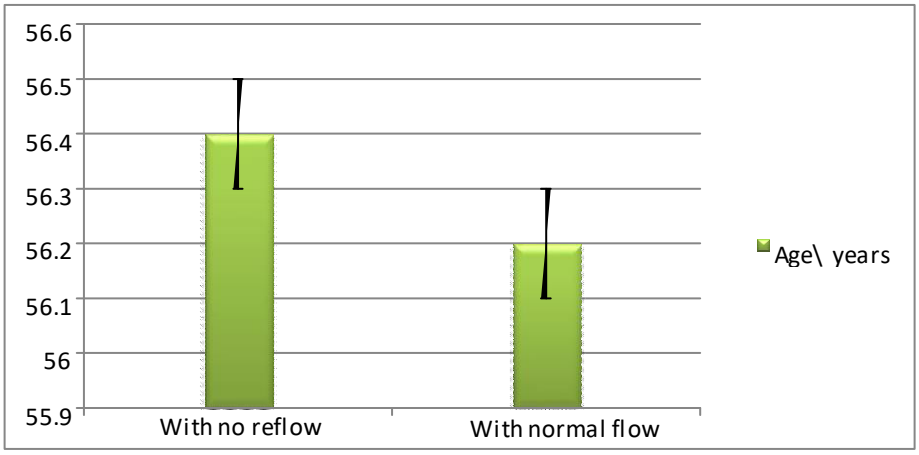

Figure (1): Age difference among both studied groups.

Table 2: Laboratory data between both studied groups

	Slow flow/no reflow N=36	**Normal flow N=36**	**t-test MW***	**P value**
CK **X ±SD** **Median (range)**	**1391.6 ± 840.6** **1235 (350-3568)**	**1390.8 ± 786.8** **1240 (515-3318)**	0.15*	**>0.05** NS
CK-MB **X ±SD** **Median (range)**	**191.9 ± 76.8** **271.5 (187 – 459)**	**286.1 ± 65.7** **271.5 (187 – 421)**	0.15*	**>0.05** NS
Serum Creatinine X ±SD **(range)**	**1.1 ± 0.27** **1 (0.74 – 1.65)**	**1.12 ± 0.26** **1 (0.75 - 1.65)**	0.37	**>0.05** NS
Troponin X ± SD **(range)**	**1 ± 0.0** **1**	**1 ± 0.0** **1**	**----**	**-----**

NS: P-value>0.05 is not significant

CK: Creatine kinase, CK-MB: Creatine kinase myocardial band

Table 3: Echo-cardio graphic parameters between both studied groups.

	Slow flow/ no reflow N=36	Normal flow N=36	t-test\	P-value
LVEDD **X ±SD** **Range**	**5.18 ± 0.6** **3.9 – 6.3**	**5.18 ± 0.61** **3.9 – 6.3**	0.00	>0.05 NS
LVESD **X ± SD** **Range**	**3.82 ± 0.62** **2.8 – 4.9**	**3.83 ± 0.64** **2.8 – 4.9**	0.07	>0.05 NS
EF% **X ± SD** **Range**	**50.3 ± 7.58** **33 - 70**	**50.6 ± 7.87** **33 – 70**	0.19	>0.05 NS
FS **X ± SD** **Range**	**25.6 ± 4.61** **16 - 39**	**25.9 ± 4.83** **16 – 39**	0.23	>0.05 NS

Table 4: Angiographic Characters of the studied groups

	Slow flow/no reflow N=36	Normal flow N=36	X2 t-test*	P value
Door to balloon (minutes) **X ± SD**	**129.2 ± 29.8**	**129.2 ± 29.8**	0.00	>0.05 (NS)
Length of lesion X ±SD **Median (Range)**	**22.3 ± 9.15** **22 (10 – 45)**	**25.6 ± 14.1** **22 (10 – 84)**	MW 0.79	>0.05 NS
Reference luminal diameter **X ±SD** **Median (Range)**	**2.97 ± 0.43** **3 (2 - 4)**	**3 ± 0.46** **3 (2 - 4)**	0.27*	>0.05 NS
Type of occlusion **Total** **Sub-total**	**20 (55.6%)** **16 (44.4%)**	**21 (58.3%)** **15 (41.7%)**	0.05	>0.05 NS
Site of lesion **LAD** **LCX** **RCA**	**17 (47.2%)** **6 (16.7%)** **10 (27.8%)**	**18 (50%)** **6 (16.7%)**	0.08	>0.05 NS

OM	**3 (8.3%)**	**9 (25%)** **3 (8.3%)**		
Location **Proximal** **Middle** **Mid to distal** **Para-osteal &osteal** **Distal**	 **20 (55.6%)** **8 (22.2%)** **2 (5.6%)** **2 (5.6%)** **4 (11.1%)**	 **20 (55.6%)** **7 (19.4%)** **2 (5.6%)** **3 (8.3%)** **4 (11.1%)**	0.27	>0.05 NS
Thrombus burden degree **Mild** **Moderate** **Heavy**	**0 (0.0%)** **15 (41.7%)** **21 (58.3%)**	**2 (5.6%)** **14 (38.9%)** **20 (55.6%)**	2.06	>0.05 NS

Table 5: Validity data for ECG parameters used as predictors of no-reflow after PCI between studied cases.

	RWPT	**ST-elevation**
Cut off	**53.5 msec**	**5.5**
AUC	**0.857**	**0.733**
p-value	**<0.001 HS**	**< 0.05 S**
Sensitivity	**100%**	**86.1%**
Specificity	**80.6%**	**83.3%**
PVP	**83.7%**	**83.8**
PVN	**100%**	**85.7%**
Accuracy	**90.3%**	**84.7%**

Discussion

Restoration of blood flow by primary percutaneous coronary intervention has proven to be the most important therapeutic strategy as it has increased survival and improved prognosis and quality of life of patients.The cause of no-reflow after primary PCI in patients with STEMI is complex. The possible mechanisms of no-reflow include spasm, endothelial dysfunction, microvascular disorders, embolization, and reperfusion injury**(12).**

Our study aimed to improve STEMI management and outcome by identify the clinical, periprocedural finding that could predict slow flow/ no-reflow in STEMI patients treated with

PCI and to detect the impact of slow flow/ no-reflow during hospital stay and short term outcome of such patients.

In agreement with **Wagdy et al., (13)** our study shows the statistically non-significant difference between the two groups ($p>0.05$) regarding age, However, in contrast to**Çağdaş et al., (14)** who revealed that patients with NR are more significantly older than those without no-reflow this may be explained by older age group of patients taken for their study (63 ± 11).The understanding regarding age-related to no-reflow is limited. This mechanism is probably through pre-existing microvascular dysfunction.

The current study shows the statistically non-significant difference between the two groups regarding BMI,SBP, DBP, and duration of chest pain it was concordant with **Zhou et al., (12)** study which includes 312 patients there were no significant differences between the reflow group and the no-reflow group in SBP,DBP and duration of chest pain ($P>0.05$ for all). In contrast,**Sabin et al., (8)** study that included 181 patients, patients who were taking anticoagulation medications for any reason, and patients who had undergone a rescue PCI were excluded, also**Fajar et al., (15)** showed that there was a statistically significant difference regarding age, SBP, DBP and duration of chest pain

Our study shows that there was no statistically significant difference between both studied groups regarding cardiac biomarkers CK, CK-MB, and troponin. This was concordant with **Kim et al(16)** and, **Sabin et al.(8)** study showed that there was no statistical difference regarding CK, CKMB, and troponin. In contrast, it was discordant with **Zhou et al. (12)** who demonstrated that peak Ck-MB level was statistically significantly elevated in no-reflow patients as this reflects large infarction size.

Our study shows that there was no statistically significant difference among both studied groups regarding echocardiographic parameters Similar to **Kim et al., (16)** study that found no difference between groups in Echo data. In contrast to our study,**Fajar et al., (15)** conduct a meta-analysis study of a total of 27 retrospective and prospective studies, which revealed that increasing risks of no reflow were associated with decreased left ventricular ejection fraction (LVEF).

There was no statistically significant difference between both groups regarding door to balloon as patients with good tissue reperfusion following PCI have minimal time between attending to the hospital and underwent primary PCI. This was concordant with **Sharma et al., (17)and Fajar et al., (15)** as there was no statistically significant difference regarding door to balloon time. This was disconcordant with **Zhou et al., (12)**, demonstrate that Significant longer reperfusion time in patients with no-reflow as delayed presentation is a potentially preventable factor and associated with a greater ischemic injury which leads to edema of the capillary bed, swelling of myocardial cells and neutrophil plugging. It is well known from animal studies that a longer duration of occlusion of the coronary artery is associated with no-reflow after reopening the artery **Rezkallaet al.,(18).**

The current study showing there was no statistically significant difference among both studied groups regarding angiographic findings of the lesion (TIMI flow grade, MBG, thrombus burden degree, location, site, and type of occlusion in concordant with our results **Acet et al., (19)** and **Çağdaş et al., (20),** similar to our study found that there was no significant difference between groups regarding IRA or number of diseased vessels.

Our study shows that the RWPT parameter had a higher sensitivity in detecting cases with expected slow flow /no-reflow after PCI (100% versus 86.1% of ST-elevation parameter), with higher test accuracy than ST elevation parameter (90.3% versus 84.7% respectively). **Çağdaş et al., (20)** showed that RWPT before and post PCI were significantly associated with the slow flow /no-reflow phenomenon and RWPT before PCI was found to be an independent predictor for no-reflow,pre-procedural RWPT value of >28.2 ms is the best cut-off value to predict NR with a sensitivity and specificity of 61.6% and 56% respectively (AUC 0.679, P > 0.001). Also, the association between angiographic slow flow /no-reflow and RWPT post PCI was statistically equal to no-reflow and STR% in the ROC curve comparison.

Conclusion

Our study established that initial coronary patency upon diagnostic angiography is significantly associated with immediate post PCI TIMI flow. Baseline TIMI flow 0 grade before primary PCI. Besides, the pathogenesis of the no-reflow phenomenon is complex and multifactorial. In light of our recent study, patients who are likely to develop no-reflow after primary PCI can be identified by simple clinical and angiographic features. RWPT is strongly associated with and significantly predicts the development of NR and significant predictor for the occurrence of adverse clinical events in hospitals and on follow-up.

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