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Slow flow and No Reflow Post PrimaryPercutaneous Coronary Intervention: Prediction and Short term Impact

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Abstract

Background:Despite recent advances in interventional equipment and techniques, the angiographic no-reflow and slow flow phenomenons occurs in a considerable number of patients undergoing primary percutaneous coronary intervention (PCI). We investigated the clinical, electrocardiographic, pre procedural finding that could predict slow flow/ no reflow in ST-segment Elevation Myocardial Infarction (STEMI) patients treated with PCI, also to detect predictors and impact of slow flow/ no reflow during hospital stay and short term outcome of such patients

Aim: 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.

Patients and Methods: Thestudy included 72 patients who were divided into 2 groups, group I with slow flow/no reflow and group II with normal flow. All of them were subjected to follow up for 3months after PCI.

Results:Regarding ECG there was a statistically significant increase in all ECG parameters between patients with no reflow before PCI except STR there was significant decrease between patients with no reflow before PCI.After applying multivariate analysis QRST before PCI, RWPT before PCI still significant predictors for occurrence of no reflow after PCI.Our study shows a statistically significant difference in short term outcome occurred on follow up after 3 months between both studied groups.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 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. In addition, 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.R wave peaked time (RWPT) is strongly associated with and significantly predicts the development of NR and significant predictor for occurrence of adverse clinical events in hospital and on follow up.

Keywords:percutaneous coronary intervention (PCI), R wave peaked time (RWPT), ST-segment Elevation Myocardial Infarction (STEMI).

Introduction

ST-elevation myocardial infarction (STEMI) is associated with a significant morbidity and mortality. According to the current guidelines, the standard of care treatment for STEMI is a diagnostic angiography followed by percutaneous coronary intervention (PCI) **(1)**.

Primary PCI refers to a strategy of emergent coronary angiography followed by coronary angioplasty with stenting of the infarct-related artery without prior administration of thrombolytic therapy **(2).**

Patency of the infarct-related artery does not always mean restoration of normal coronary blood flow as in certain group of patients epicardial coronary artery reperfusion is achieved but without myocardial reperfusion after primary PCI, this condition is known as no-reflow **(3).**

The slow flow/no reflow phenomenon is a serious complication following percutaneous coronary intervention (PCI). It is defined as a state of myocardial hypo perfusion in the presence of a patent epicardial coronary artery **(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 flow/no reflow negatively affects the clinical outcome in patients with acute myocardial infarction (AMI) and it is associated mainly with increased mortality or left ventricular remodeling, despite its relatively low incidence **(6).**

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”.2 TIMI flow grades of 2 or lower**(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 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 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 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 were 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. Preprocedural and post-procedural (at 60 min) 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 prior to and after procedure **(9)**: **Grade 0** (no perfusion): There is no ante grade 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 cineangiographic 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 in relation to 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 score 2:** Reduced but clearly 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 use of balloon predilatation, use of stent.
Follow-Up: was doneduring 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, quantitative group were represented 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 done, the threshold of significance was fixed at 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

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 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 mean BMI 27.8 ± 1.97, 36.1% of patients were dyslipidemic , 13.9% of patients had 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).**

There was a statistically significant increase in all ECG parameters between patients with no reflow before PCI except STR that was significantly decrease in patients with no reflow PCI**(Table 2).**

After applying multivariate analysis QRST before PCI, RWPT before PCI were significant predictors for occurrence of no reflow after PCI (p< 0.05) **(Table 3).**

There was a statistically significant increase in short term (after 3 months) adverse clinical events in group I (p<0.05) **(Table 4).**

QRST parameter before and after PCI and RWPT before PCI were statistically significant predictors for adverse clinical events occurrence on follow up. There was no statistically significant difference among both studied groups regarding angiographic findings of the lesion **(Table 5).**

After applying multivariate analysis RWPT before PCI still significant predictor for occurrence of complication in hospital stay and on follow up**(Table 6).**

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 mass index, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood Pressure, DM: Diabetes Mellitus, CAD: Coronary artery disease

Table 2: ECG parameters of the studied groups

		Slow flow/noreflow N=36	Normal flow N=36	t-test\ MW*/X2**	P value
Site of MI	Anterior N (%)	**17(88.9)**	**19(52.8)**	**0.22****	**> 0.05 NS**
	Inferior N (%)	**19(52.8)**	**17(47.2)**		
ST elevation before PCI X ±SD Range		**8.8 ± 2.42 5 – 12**	**6.9 ± 1.33 5 – 10**	**4.11**	**<0.001 HS**
QRST before PCI X ± SD Range		**107.4 ± 6.92 95 – 122**	**84.8 ± 10.78 75 – 105**	**10.61**	**<0.001 HS**
RWPT before PCI X ± SD Range		**62.1 ± 3.29 58 – 70**	**45.2 ± 10.5 36 – 65**	**9.25**	**<0.001 HS**
Sum. of ST elevation Mean ± SD (Range)		**17.1 ± 4.69 10 – 23**	**13.8 ± 3.46 10 – 22**	**3.41**	**<0.05 S**
ST elevation after PCI X ± SD Median (Range)		**4.46 ± 1.83 5 (2 – 6.5)**	**1.6 ± 0.56 1.75 (1 – 2.5)**	**6.33***	**<0.001 HS**
QRST after PCI X ± SD Range		**97.6 ± 5.5 89 – 115**	**75.9 ± 10.4 66 - 95**	**11.3**	**<0.001 HS**
RWPT before PCI X ± SD Range		**49.3 ± 17.8 6 – 66**	**40.3 ± 9.88 31 – 59**	**2.67**	**<0.05 S**
Sum. of ST elevation X ± SD Median (Range)		**15.4 ± 17.6 10.5 (3.7 – 58)**	**4.98 ± 5.7 3 (2 – 19)**	**5.1***	**<0.001 HS**
STR X ± SD Range		**58.9 ± 9.73 20 – 71**	**77.4 ± 5.58 71 - 87**	**9.98**	**<0.001 HS**

HS: P-value<0.001 is high significant S: P-value<0.05 significant MI: Myocardial Infraction, STR: ST-segment resolution.

Table 3: Multivariate analysis of significant predictors for slow/ no reflow.

Variables	Multivariate analysis		
	B	95% CI	P value
ST elevation before PCI	0.528	0.398 – 1.05	>0.05
QRST before PCI	0.435	0.753-1.34	< 0.05 S
RWPT before PCI	1.824	1.273- 2.982	< 0.05 S
Sum of ST elevation before PCI	-0.051	0.324-0.534	>0.05

CI: confidence Interval, S: statistically significant (p< 0.05)

Table 4: Short term outcome after 3 months of the studied groups

Complications	Slow flow/ no reflow N=36 N (%)	Normal flow N=36 N (%)	X2	P
No complications With complications	27 (75%) 9 (25%)	34 (94.4%) 2 (5.6%)	Fisher	< 0.05 S
MI Acute heart failure Cardiac death Stroke	2 (5.6%) 3 (8.3%) 2 (5.6%) 2 (5.6)	1 (2.8%) 1 (2.8%) 0 (0.0%) 0 (0.0%)	Fisher Fisher Fisher Fisher	>0.05 NS >0.05 NS >0.05 NS >0.05 NS

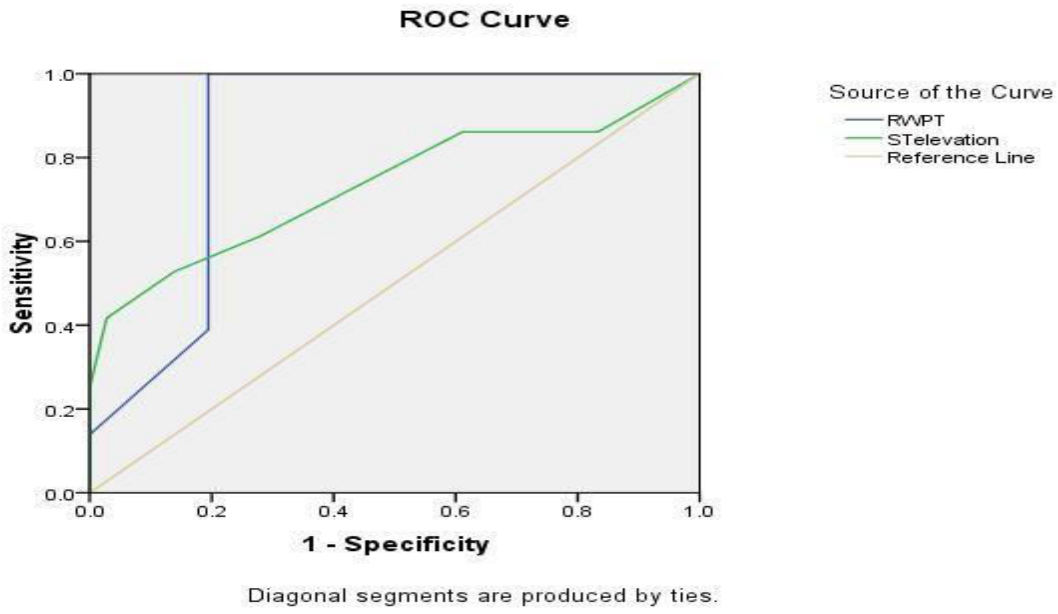

Fig 1: Receiver operating characteristics (ROC) curve for ECG parameters used as predictors of no reflow after PCI between studied cases.

Table 5: Univariate analysis of short term outcome

	With adverse clinical events N=11	Without adverse clinical events N=61	t-test\ MW*	P value
	Mean SD			
Age	51 13.4	57.1 11.7	1.3	>0.05 NS
CK	1538.1± 947.2	1370.2 739.2	0.51*	>0.05 NS
CK-MB	299.2 68	287.6 71.8	0.48	>0.05 NS
Serum creatinine	1.1 0.13	1.1 0.27	0.72	>0.05 NS
ST elevation before PCI	8.4 ± 2.5	7.7 ± 2.1	0.75	>0.05 NS
QRST before PCI	105 ± 10.2	94.8 ± 14.78	**2.01**	< 0.05 S
RWPT before PCI	60 ± 7.1	52.7 ± 11.5	**2.52**	< 0.05 S
Sum. of ST elevation	16.6 ± 4.8	15.3 ± 4.6	0.77	>0.05 NS
ST elevation after PCI	4 ± 2.1	2.9 ± 1.9	1.58*	>0.05 NS
QRST after PCI	95.3 ± 8.8	85.5 ± 13.9	**2.83**	< 0.05 S
RWPT before PCI	48.7 ± 17.2	44.3 ± 14.8	1.17*	>0.05 NS
Sum. of ST elevation	13.4 ± 17.2	9.8 ± 13.6	1.05*	>0.05 NS
STR	62 ± 10.7	68.95 ± 12.3	1.79	>0.05 NS
LVEDD	4.8 ± 0.61	5.2 ± 0.59	1.94	>0.05 NS
LVESD	3.7 ± 0.57	3.9 ± 0.63	1.43	>0.05 NS
FS	24.2 ± 4.4	25.96 ± 4.7	1.11	>0.05 NS
EF	47.9 ± 8.4	50.8 ± 7.6	0.99	>0.05 NS
Door to ballon	124.4 ± 23.5	129.8 ± 30.4	0.51	>0.05 NS
Lesion length	28.7 ± 10.5	23.3 ± 12	1.62*	>0.05 NS
Luminal diameter	3.03 ± 0.38	2.98 ± 0.45	0.34	>0.05 NS
	N (%)	N (%)	X2	
Sex: Male Female	10 (90.9%) 1 (9.1%)	55 (90.2%) 6 (9.8%0	Fisher	>0.05 NS
DM	4 (36.4%)	18 (29.5%)	Fisher	>0.05 NS
Hypertension	6 (54.5%)	20 (32.8%)	Fisher	>0.05 NS
Dyslipidemia	4 (36.4%0	22 (36.1)	Fisher	>0.05 NS
CAD	1 (9.1%)	9 (14.8)	Fisher	>0.05 NS
Smoking	7 (63.6%)	49 (80.3%)	1.51	>0.05 NS

TIMI after PCI **No reflow** **Slow reflow** **Normal flow**	**1** **(9.1%)** **7 (63.6%)** **3 (27.3%)**	**11** **(18%)** **16 (26.2%)** **34 (55.7%)**	5.99	>0.05 NS
MBG				
No reflow **Slow reflow** **Normal flow**	**5 (45.5%)** **4 (36.4%)** **2 (18.2%)**	**17 (26.2%)** **9 (14.8%)** **36 (59%)**	**6.54**	**<0.05 S**
Thrombus burden degree **Mild** **Moderate** **Severe**	**0** **(0.0%)** **5 (45.6%)** **6 (54.4%)**	**2** **(3.3%)** **24 (39.3%)** **37 (57.4%)**	0.46	>0.05 NS
Location **Proximal** **Middle** **Mid to distal** **Para-osteal** **Distal**	**6 (54.5%)** **2 (18.2%) 0** **(0.0%)** **2 (18.2%)** **1 (9.1%)**	**34 (55.7%)** **13 (21.3%)** **4 (6.6%)** **3 (4.9%)** **7 (11.5%)**	3.18	>0.05 NS
Occlusion Total **Sub-total**	**7 (63.6%)** **4 (36.4%)**	**34 (55.7%)** **27 (44.3%)**	Fisher	>0.05 NS
Site of lesion LAD **LCX** **RCA** **OM**	**4 (36.4%)** **2 (18.2%)** **4 (36.3%)** **1 (9.1%)**	**31 (50.8%)** **10 (16.4%)** **15 (24.6%)** **5 (8.2%)**	0.91	>0.05 NS

Table 6: Multivariate analysis of short term outcome.

Variables	**Multivariate analysis**		
	B	**95% CI**	**P value**
In- hospital outcome finding			
RWPT before PCI	1.517	1.241 - 2.28	< 0.05 S
History of CAD	0.003	0.873- 1.12	>0.05 NS
STR	-0.044	0.52 -1.23	>0.05 NS
On follow up adverse clinical events			
QRST before PCI	0.435	0.53-1.22	>0.05 NS
RWPT before PCI	1.24	1.23- 4.12	< 0.05 S

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- ref low include spasm, endothelial dysfunction, microvascular disorders, embolization, and reperfusion injury**(12).**

Our study aimed to improve STEMI management and outcome by identify the clinical, periprocedul 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 statistically non significance difference between 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 statistically non significance difference between two groups regarding BMI ,SBP, DBP and duration of chest pain it was concordant with **Zhou et al., (12)** study which include 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 include 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 statistically significant difference regarding to age, SBP, DBP and duration of chest pain

Our study shows a statistically significant increase in all ECG parameters (Preprocedural QRSD, Postprocedural QRSD, Preprocedural RWPT, Postprocedural RWPT, Preprocedural Maximum ST elevation and STR) among patients with no reflow before PCI except STR which was lower among patients with no reflow before PCI. The nuances between QRSD and RWPT in prediction for NR could be explained on a pathophysiological basis if we consider that STEMI causes a localized segmental myocardial ischemia and hence a localized conduction delay in various ECG infarct-related artery (IRA) leads, therefore RWPT (which reflects early intra-ventricular conduction) could be more sensitive in this regard than QRSD (which reflects the conduction status of the Purkinje system as a whole). It was reported that successful reperfusion improves QRS duration and decreases mortality **(16).**

Our study results was concordant with **Çağdaş et al., (14),Bendary et al., (17)** in which there was statistical significant difference between both group regarding to Preprocedural QRSD, Postprocedural QRSD, Preprocedural RWPT, Postprocedural RWPT, Preprocedural Maximum ST elevation and STR, these parameters increase among patients who develop no reflow except STR that significantly decreases in no reflow group.

Regarding Short term outcome after 3 monthsin 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.

In agreement with **Zhou et al., (12**) showed that no reflow is associated with high incidence of adverse clinical events. **Van Kranenburg et al., (18)** concluded that microvascular obstruction is responsible for no reflow, which is an independent predictor of adverse clinical events and cardiac death at 2 years.

Our study shows that RWPT before PCI was statistically significant predictors for hospital and short term outcome in concordant with our results **Alameddin et al., (19)**, which Correlate between risk factors and angiographic data with the incidence of adverse clinical events after PCI stated that there was no correlation between risk factors or demographic data and adverse clinical events.

Our study shows that after applying multivariate analysis RWPT before PCI still significant predictor for occurrence of adverse clinical events in hospital and on follow up and this was concordant with results of **Bendary et al., (17)** Pre-procedural RWPT is a significant predictor of no reflow after PCI. In **Çağdaş et al., (14)** study that include 233 consecutive STEMI patients treated with PCI after exclusion of patients with a history ofcoronary artery disease by applying multivariate analysis, STR (%) and RWPT before PCI are significant independent predictor of no reflow post PCI, while in contrast to our study QRSD before PCI is not a significant predictor of slow flow /no reflow.

Our study shows that RWPT parameter had a higher sensitivity in detecting cases with expected slow flow /noreflow 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., (14)** showed that RWPT before and post PCI were significantly associated with the slow flow /noreflow 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 /noreflow 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. In addition, 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 occurrence of adverse clinical events in hospital and on follow up.

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