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Can Simple Electrocardiographic Parameters Predict Reperfusion in Acute ST Elevation Myocardial Infarction?

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ABSTRACT

Background: The optimum treatment for ST-elevation myocardial infarction (STEMI) is rapid reperfusion with primary percutaneous coronary intervention (PCI). Myocardial tissue hypoperfusion persists in many patients after initial PCI despite the epicardial coronary circulation achieving restored patency. Therefore, our study aimed to assess the relationship between simple electrocardiographic (ECG) parameters and reperfusion in STEMI. **Patients and methods:** A cross sectional study was carried out one hundred STEMI patients subjected to 12 leads ECG before and sixty minutes after (PPCI) to assess QRS duration, T wave peak to end terminal(TPE) interval. **Results:** One hundred cases were classified into two groups according to MBG (myocardial blush grade), group I (30) cases (Impaired flow MBG<3) and group II (70) cases (successful flow MBG=3). Significant longer duration of pre PCI QRS, post PCI QRS, pre corrected PCI TPE, post PCI TPE, post PCI corrected TPE were in group I. **Conclusion:** Prolonged pre QRS duration 86ms and maximal ST elevation in mm were significant predictors of no reflow in STEMI treated with primary PCI.

Keywords: Myocardial blush grade; QRS duration; T wave peak to end terminal.

INTRODUCTION

The ultimate goal of reperfusion therapy is to maintain a healthy microvascular flow and the patency of the infarct-related artery (IRA). However, the tissue level reperfusion does not always return after the epicardial flow is restored **[1]**.

The optimum treatment for ST elevation myocardial infarction (STEMI) is rapid reperfusion via percutaneous intervention (PCI). Even if the epicardial coronary circulation has been fully restored in a significant majority of patients following primary PCI, myocardial tissue hypoperfusion still exists in these patients. This syndrome, known as no-reflow, is mostly brought on by significant loss of integrity and microvascular dysfunction **[2,3]**.

In many cases, poor microvascular reperfusion in clinical practice is likely to go undetected. QRS duration and T wave peak end terminal have been thoroughly investigated as a predictor of poor outcome; very few studies have addressed their role in reperfusion state after primary PCI.

So, this study aimed to determine how basic electrocardiographic variables such as T wave peak to end terminal (TPE), QRS duration, and microvascular reperfusion graded by myocardial blush grade (MBG) in acute ST-Elevation MI related to one another.

PATIENTS AND METHODS

A cross-sectional study involved one hundred acute STEMI patients treated by primary PCI, during the period of march 2021 to July 2021.

Ethical Approval:

The study was approved by the Zagazig University Faculty of Medicine's research ethical committee, and each participant's parents were completed informed permission forms. The conduct of the study adhered to the Declaration of Helsinki, the World Medical Association's Code of Ethics for Human Research.

Inclusion criteria:

Patients admitted with acute STEMI within 12 hours of experiencing typical chest pain lasting 20 minutes or less with ST-segment elevation of more than 1mm in at least two contiguous precordial leads or one mm in at least two limb leads; it was later confirmed by rise in (CK-MB or troponin)**[4]**.

Exclusion criteria

Intraventricular conduction abnormalities, such as bundle branch block (BBB) block. Atrioventricular block of the second or third degree with a QRS of greater than 120 ms. Patients with low -systolic function EF less than 50% , more than mild valvular lesion. Patients managed with thrombolytic therapy. Patients with old MI or prior PCI.

Methods

Full medical history and risk factors recording. Cardiac enzymes, liver and kidney function lipid profile were all withdrawn.

Twelve leads surface ECG , the speed of paper was fifty mm/s and amplification of ten mm / mV before and sixty minutes after PCI was done to assess QRS duration which was measured from the onset of QRS onset to the J point from the Infarct related artery (IRA) leads **[1]**.

The average measurement value for the three consecutive beats, as determined by the two investigators, was (3% and 2%, respectively), with inter-observer and intra-observer variability.

The TPE interval was measured from the peak of T wave till the end of T wave, the junction of the isoelectric line and the tangent to the downslope of T wave was defined as the end of T wave **[5]**.

TPE was measured from a lead with the least ST-segment deviation, TPE was corrected by heart rate according to same formula based for QT, Bazette formula; TPE\√R-R **[5].**

Maximum ST elevation was assessed through the lead showing the maximum ST elevation, and percentage of ST segment resolution (STR) was calculated by. The percentage of ST segment resolution (STR) was determined as the initial sum of ST elevation minus the sum of ST elevation after sixty minutes from the PCI divided by the initial sum of ST elevation, value more than seventy percent was considered successful reperfusion **[6]**.

Two dimensional transthoracic echocardiography by VIVIED e-95,Norway) was used \to assess left ventricular (LV) systolic function.

Coronary angiography and primary (PCI): the traditional Judkine technique was used for coronary angiography. A six or seven-french catheter was used for primary PCI.

IRA was only accessed unless patient was in cardiogenic shock, ballon dilatation, direct stenting ,aspiration catheter and glycoprotein IIb,3Ainhibitors usage were all based up on the operator decision, thrombolysis in myocardial infarction (TIMI) flow grades**[7],** TIMI frame counts **[8]** and MBG **[9]** were assessed by two experienced operators. Number of diseased vessels was also recorded **[10]**.

Our study groups were classified according to MBG as no-reflow group I (MBG ≤3 n=30) and patients with successful reflow (MBG =3 n=70).

Statistical analysis

The Statistical Package for the Social Sciences (SPSS) version 20.0 was used to analyze the data. Mean and standard deviation were used to express quantitative data, and the t test was used to compare quantitative and qualitative data. The relationship between variables was determined using Pearson correlation, the predictors of no-reflow were determined using logistic regression, and the cut-off for significant variables was determined using the receiver operating curve (ROC).

RESULTS

We enrolled 100 cases of acute STEMI and patients were classified into 2 groups according to MBG ,group I no-reflow30cases and group II successful-flow 70 cases, group I were significantly older age, with more prevalence of hypertension ,diabetes, and smoking **(Table 1).**

Peak CKMB was significantly higher in group II, pre PCI QRS ,post PCI QRS ,pre PCI TPE, post PCI TPE, pre PCI cTPE and post PCI cTPE duration were all longer in group I with significant difference, maximum ST elevation was higher in group I but complete STR >70% were higher in group II **(Table 2)**.

Group II had more prevalent single vessel disease (SVD) when compared to group I with significant difference ,IRA (LAD) was more prevalent in group I with significant difference ,TIMI frame count mean value was higher in group I in comparison to group II, TIMI III post PCI was higher in group II **(Table 3)**.

Univariate predictors of no reflow were demonstrated in **Table (4**).

Pre-QRS duration of 86 or more and increased maximum ST segment elevation were the predictive variables for no reflow in multivariate regression **(Table 5)**.

Pre PCI cTPE had a cut-off value of 104.5, which had a sensitivity of 71.6%, a specificity of 65.5%, and an area under the curve (AUC) of 0.618. Post PCI cTPE intervals had a cut-off value of 108.5, which had a sensitivity of 77.3%, a specificity of 69.8, and an AUC of 0.734. (**Figure 1)**.

Pre QRS had a cut-off value of 86, sensitivity 78.6%, specificity 75.2%, and area under the curve (AUC) of 0.709, whereas Post QRS had a cut-off value of 81, sensitivity 73.3%, specificity 73.2, and area under the curve (AUC) of 0.672 **(Figure 2).**

Post cTPE and TIMI frame showed a positive connection (r=0.31, p=0.001) (**Figure 3).**

Table (1): Demographic data of the studied groups:

	Post PCI MBG				**N**	**χ^2**	**p-value**
	Group I No-re flow 30 (30%)		**Group II Successful flow 70 (70%)**				
	No.	**%**	**No.**	**%**			
Age							
≤60 years	10	33.3	40	57.1	50	4.8	0.029
>60years	20	66.7	30	42.9	50		
Sex							
Males	18	60	46	65.7	64	0.29	0.58
Females	12	40	24	34.3	36		
HTN							
Yes	23	76.7	38	54.3	61	4.4	0.035
DM							
Yes	28	93.3	50	71.4	78	5.9	0.015
Family history of premature CAD							
Yes	22	73.3	45	64.3	67	0.78	0.38
Dyslipidemia							
Yes	25	83.3	56	80	81	0.15	0.69
Smoking habit							
Smoker	16	53.3	18	25.7	34	7.1	0.008

HTN:Hypertension , DM:diabetes mellitus, CAD:coronary artery disease , PCI:percutaneous coronary intervention , MBG:myocardial blush grade

Table (2): Laboratory, ECG, and Echocardiographic data of the studied groups

Variables		Post PCI MBG		T	P
		Group I No-re flow 30 (30%)	Group II Successful flow 70 (70%)		
CK MB Peak 6 hours	Mean ±SD	206.9±97.5	236.82±97.1	3.58	0.001
Troponin	Mean ±SD Median (range)	1517.4±1316 1077 (567-5210)	1700.2±1313.7 1700 (860-5335)	0.637	0.525
AST	Mean ±SD	68.6±8.5	72.3±11.2	1.766	0.082
ALT	Mean ±SD	36.9±6.4	37.6±5.7	0.524	0.602
Pre QRS Duration	Mean ±SD	87.45±3.7	83.6±5.2	4.218	0.0001
Post QRS Duration	Mean ±SD	81.5±3.4	78.6±5.1	2.882	0.005
Pre PCI TPE	Mean ±SD	104±5.9	105 ±5.1	0.848	0.398
Post PCI TPE	Mean ±SD	104.9±4.1	100.6±5.9	3.66	0.0001
Pre PCI cTPE	Mean ±SD	114.4±13.8	108.9±10.8	2.14	0.035
Post PCI cTPE	Mean ±SD	112.3±5.7	105.6±8.5	3.9	0.0001
Variables		**Group I (n.30)**	**Group II(n.70)**	**T**	**P**
Maximum ST elevation		4.8±0.55	3.7±0.74	7.2	0.001
STR complete >70%		2(6.7)	52(74.3)	$\chi 2$ 3 8.2	0.001
incomplete <70%		28(93.3)	18(25.7)		
EF%		46.4±6.24	54±4.9	6.4	0.001
ESVml		37.8±2.9	36.8±3.3	1.5	0.1
EDVml		51.4±4.7	52.9±3.4	1.6	0.1

CKMB:creatin kinase M band; AST: aspartate aminotransferase; ALT: alanine transaminase; TPE: T wave peak to end terminal ,cTPE: corrected T wave peak to end terminal, EDV: end diastolic volume, ESV: end systolic volume, EF: ejection fraction, STE: ST segment resolusion ; PCI: percutaneous coronary intervention ; MBG:myocardial blush grade; T:t- test to compare between quantitative data.

Table (3): Angiographic data in the studied groups

	Group I no reflow (MBG<3) 30 (30%)		Group II Successful flow (MBG=3) 70 (70%)		n.	χ^2	p-value
	No.	%	No.	%			
Number of vessels affected							
SVD	21	70	65	92.9	86	9.5	0.009
DVD	5	16.6	2	2.8	7		
TVD	4	13.3	3	4.3	7		
(IRA)							
LAD	21	70	33	47.1	54	4.4	*0.036*
RCA	1	3.3	24	34.3	40	0.2	0.66
LCX	5	16.7	10	14.3	17	0.003	0.95
LM	2	6.7	1	1.4	3	F	0.21
OM	1	3.3	2	2.9	6	F	0.064
TIMI frame count Mean ±SD	27.5±1.5	-	22.7±1.2	-	-	T 16.5	*0.0001*
Baseline TIMI0-1	25	83.3	62	88.7	87	5.7	*0.06*
Post-TIMI 3	5	16.7	63	90	95	4.9	*0.001*

SVD: single vessel disease; DVD: double vessel disease; TVD: trip le vessel disease; OM: obtuse marginal; IRA: infarct related artery; LAD: left anterior descending; RCA: right coronary artery; LCX: left circumflex; TIMI: thrombolysis in myocardial infarction.

Table (4): Univariate logistic regression for no reflow predictive variables.

	Univariate			
	Sig	Exp(β)	Lower	Upper
Age >60years	0.032	2.667	1.090	6.524
Hypertension	0.039	2.767	1.051	7.284
Diabetic patients	0.027	5.6	1.218	25.743
Smokers	0.009	3.302	1.348	8.083
CK MB	0.0001	1.01	1.004	1.016
maximum ST elevation in mm	0.0001	8.546	3.593	20.325
Incomplete STR	0.0001	40.444	8.745	187.04
Pre PCI cTEP≥104.5	0.619	1.256	0.511	3.085
Post PCI cTEP≥I108.5	0.001	4.654	1.812	11.953
Pre PCI QRS.duration.≥86	0.000	5.778	2.281	14.633
Post PCI QRS duration≥ 81	0.009	3.302	1.348	8.083
TIMI frame count	0.0001	7.346	2.621	20.591

CKMB: creatin kinase M band;TIMI: thrombolysis in myocardial infarction; STR:STsegment resolusion; cTPE: corrected T wave peal to end terminal.

Table (5): Multivariate logistic regression for no reflow predictive variables

Variables	Sig.	Exp (β)	95% C.I for EXP (B)	
			Lower	Upper
Age >60years	0.076	4.72	1.025	21.7
Diabetic patients	0.085	11.86	1.054	133.48
Smokers	0.01	8.509	1.684	43
Pre PCI QRS.duration.≥86	0.008	8.07	1.720	37.85
Maximum ST elevation in mm	0.001	10.98	3.654	32.99

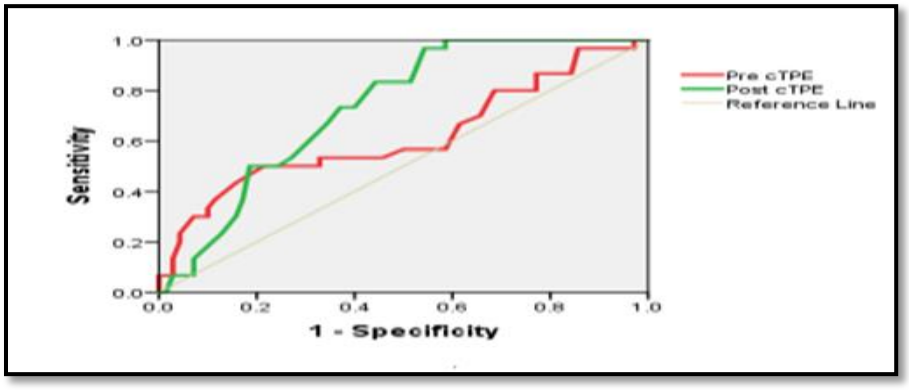

Figure (1): ROC of pre PCI cTPE and post PCI corrected T wave peak to end terminal (cTPE).

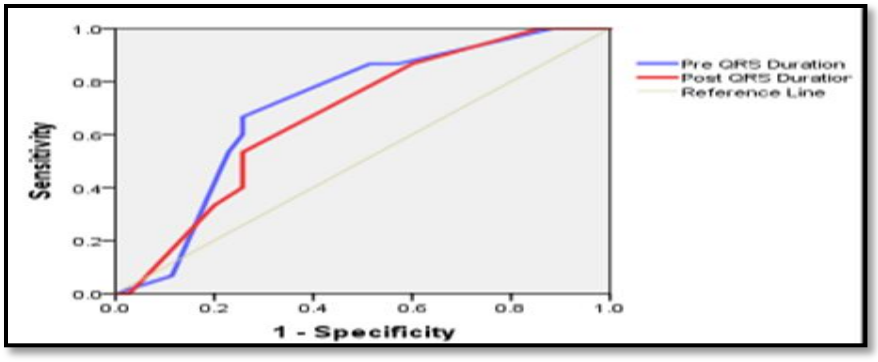

Figure (2): ROC of pre PCI QRS and post PCI QRS

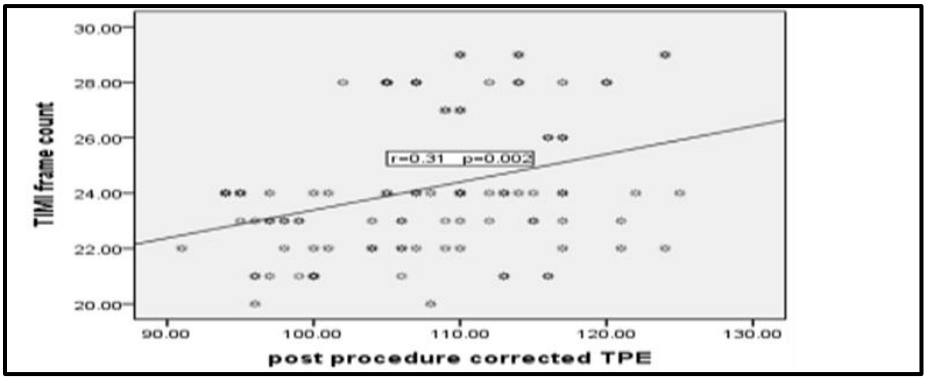

Figure (3): Correlation between the TIMI frame count and post-procedure cTPE.

DISCUSSION

Acute STEMI contributes considerably to both mortality and morbidity. The primary objectives of reperfusion therapy include adequate microvascular flow in addition to restoring IRA patency **[11,12]**.

Even with the patency of IRA post-PCI, there is evidence that over fifty percent of microvascular poor perfusion on cardiac magnetic resonance following STEMI **[13]**. This is referred to as acute microvascular blockage, which may be reversible, and may be caused by microvascular thrombi and endothelial disruption **[14]**.

The ECG should not be utilised only for the classification of an acute coronary syndrome, even when imaging modalities like cardiac magnetic resonance, echocardiography, and radionuclide methods offer far more accurate, although expensive, information. In order to identify patients who need more aggressive treatment plans and close monitoring, it is still a crucial tool for risk.

This study aimed to assess the relationship between QRS duration, TPE interval, cTPE interval, and microvascular reperfusion in patients with STEMI who underwent primary PCI.

TPE interval is associated with transmural ventricular repolarization **[15]**, and shown to be related to ventricular arrhythmias and also linked to high risk for sudden cardiac death (SCD) **[16, 17],** but assessing its value in reperfusion success need to be elucidated.

Acute myocardial infarctions actually cause alterations in electrochemical and metabolism of the cardia muscles, which affect the pH, tissue oxygen level, ion channel conditions, and electrochemical gradient. The duration of action potential is extremely affected by these alterations in the infarcted areas where TPE display compatible changes **[18]**.

Pre PCI cTPE had a statistically higher value in the no-reflow group compared to the success flow group. Additionally, a univariate regression test revealed that the odds ratio (OR) for post-cTPE individuals was 4.654.

Similarly, **Duyuler et al. [19]** reported that the MBG 3 group's post-PCI c-TPE interval was shorter than those of the MBG 0-1 and MBG 2 groups.

Çoner et al. [5] concluded that the average TPE interval was much shorter at admission time, and it was found that a TPE value of 89 ms on the admission ECG was associated with successful reperfusion.

In the current study, we found that the success reflow group's pre- and post PCI QRS durations were much shorter than in no-reflow group. These results were concordant with **Tawfik et al. [12]** who reported that at admission and post angioplasty QRS duration was longer in impaired perfusion group in comparison to normal perfusion group.

Yusuf et al. [1] found non-significant difference between normal and impaired reperfusion groups on term of admission time QRS duration. However, when compared to post angioplasty QRS duration was longer in group of impaired reperfusion when compared to normal reperfusion. **Karahan et al. [20]** and **Ilkay et al. [21]** reported similar observation.

All of these researches support the concept that in patients with STEMI, ischemia results in QRS prolongation and dynamic PCI QRS change. Our study, demonstrated that the cut-off value of pre QRS duration 86 msec is a reliable indication of acute STEMI patients' no reflow.

Similar to a prospective observational trial with 201 STEMI patients, this study found that the QRS duration had an association with microvascular obstruction assessed by cardiac magnetic resonance (CMR) (OR1.362, p = 0.024) **[21].**

Yusuf et al. [1] concluded that the best differentiating value for the reperfusion success was a QRS duration cut-off of 89.5 ms following angioplasty (sensitivity 81.7%, specificity 74.3%).

This was concordant with **Al Daydamny et al. [22]** who observed that microvascular hypoperfusion was predicted by QRS duration prior to PCI with a cut-off point of 89 ms.

This supports the concept that persistent ischemia to the Purkinje-ventricular conduction pathway causes QRS enlargement, which is linked to the increased risk of heart failure, arrhythmia, and ischemia, all of which increase long-term mortality **[20]**.

Our results showed that maximum ST elevation was significantly higher in the no reflow group. A finding was powered by multivariate regression analysis to be one of the predictive variables of no

reflow. According to a prospective study involving 85 STEMI patients, ST segment elevation greater than three millimetres 90 minutes after PCI is a reliable indicator of poor reperfusion **[23]**.

CONCLUSION

Prolonged pre PCI QRS duration ≥86 was related to low MBG, a sign of impaired microvascular reperfusion, and also maximum ST elevation were predictive variables for no reflow in STEMI.

LIMITATIONS

Single centre study, small sample size , subjective assessment of the ECG, being assessed manually, we didn't assess the microvascular function by cardiac magnetic resonance (CMR) instead of that we depend on subjective assessment of MBG , we did not keep track of our patients to determine how the prolonged QRS and the reduced MBG affected cardiac events.

RECOMMENDATIONS

We recommend using QRS duration as a simple; available , sensitive parameter predicting the no-reflow, future multi-centres with larger sample size to conform our results , future large trials to compare between the cardiac magnetic resonance (CMR) and this electrocardiographic parameter.

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Author contribution: Authors contributed equally in the study.

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