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ORIGINAL ARTICLE

Value of cTPE\ c QT in the Prediction of No-reflow in Acute ST-Elevation Myocardial Infarction

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ABSTRACT

Background: The occurrence of no-reflow in patients with STEMI means inadequate myocardial reperfusion despite patent coronary arteries and it accounts for as much as twenty percent of patients undergoing PPCI. Electrocardiogram (ECG) is a widely available non-invasive diagnostic tool in daily practice that is easy to use. So our study aimed to evaluate the corrected value of T wave peak interval to end terminal **(**cTPE) and corrected QT and assess the ratio of cTPE\ c QT in predicting no-reflow. **Methods**: A total of 120 patients who fulfilled inclusion and exclusion criteria were subjected to 12 leads surface ECG, TPE interval was measured from the peak of the T wave to the end terminal of the T wave tangent to the baseline in the leads the least ST changes (defined as less than <0.055 mV from the isoelectric line. QT interval was assessed from the onset of the QRS complex to the T wave end, other clinical and angiographic data were compared in successful and no-reflow groups. **Results**: The population study was divided into no-reflow group I accounting for 33.3% and successful reperfusion group II accounting for 66.7%. Admission time cTPE\ cQT was significantly more prolonged in group I 0.281±1.94 compared to 0.192±1.07 in group II. Multivariate analysis showed that admission time from symptoms onset to hospital admission (0.017), followed by Hs-troponin peak value pg\ml (0.007), then Admission time cTPE\ cQT (0.001) were significant predictors of impaired flow TIMI <3 post PCI. **Conclusions**: Admission time cTPE\ cQT at a cut-off value ≥ 0.243 ms could accurately predict impaired flow TIMI <3 post PCI, with a sensitivity of 78.8% and, a specificity of 66.5 %.**keywords** : no-reflow , ST elevation myocardial infarction (STEMI) , cTPE\ Cqt

INTRODUCTION

The optimal treatment for patients with ST-segment elevation myocardial infarction (STEMI) is primary percutaneous coronary intervention (PPCI), however, in those who experience the no-reflow phenomenon, the ability to restore myocardial reperfusion may be compromised [1]. The occurrence of no-reflow in patients with STEMI means inadequate myocardial reperfusion despite patent coronary arteries and it accounts for as much as twenty percent of patients undergoing PPCI [2]. Electrocardiogram (ECG) is a common diagnostic tool that is easy to apply in clinical settings. The usefulness of certain ECG abnormalities, including ST-segment elevation, T-wave inversion, and prolonged QT interval, in predicting the no-reflow phenomenon has been studied in cohort studies [3]. A previous study [4,5] analyzed the impact of TPE\ cQT in no-reflow prediction in STEMI, but this study neglected that TPE varies with HR variation as equal to QT variation with HR. So the aim of the current study

was to evaluate the corrected value of T wave peak interval to end terminal **(**cTPE) and corrected QT and assess the ratio of cTPE\ c QT in predicting no-reflow.

METHODS

Study population : a total 160 STEMI patients underwent PPCI between September 2023 and April 2024 in AL-Ahrar Teaching Hospital and Zagazig University Hospitals. STEMI was defined as ST-segment elevation in at least two contiguous leads and persistent chest pain or angina equivalent . The following patients were excluded from the study: bundle branch block, high grade AV block, QRS duration more than 120 ms (10 cases), structural heart disease and prior MI (4 cases and 6 cases, respectively) and subjects with U waves and negative T waves on their ECGs (20 cases).Thus 140 patients made up the actual study's population.

ECG analysis: A digital 12-lead ECG was performed to all patients at admission, recording at a speed of 25 mm/s and a voltage of 10 mm/mV. The ECG data was analyzed by two independent cardiologists who were blind to the clinical information of the patients. Successful reperfusion was defined as ST segment resolution (STR) ≥ 50% after 60 minutes from PPCI [6]. The TPE interval was measured from the T wave peak to the end terminal of the T wave tangent to the baseline in the lead that had the least ST changes (defined as less than <0.055 mV from the isoelectric line [7]. QT interval was assessed from the onset of the QRS complex to the T wave end, All of the measurements (Tpe and QTc intervals) analyzed were the average of 3 consecutive beats. Since the QT and TPE intervals vary with heart rate, Bazett's formula (corrected index interval=index interval/√R–R) was used to evaluate the corrected values of the QT (cQT) and TPE (cTPE) intervals, respectively [8].

Laboratory data: Cardiac enzymes and Troponin (the highest level of cardiac enzymes were collected), renal function tests, and lipid profile were collected and analyzed.

Coronary Angiography: The infarct-related artery was identified via coronary angiography (IRA). Options for revascularization and coronary angiography adhered to the guidelines [5]. Following the femoral sheath insertion, a 100 IU/kg heparin bolus was given. If, upon a visual assessment, the residual stenosis in the IRA was less than twenty percent, the PPCI treatment was deemed successful. The Siemens Axiom Artis Zee® instrument (Siemens Healthcare Erlangen, Germany) was used to record coronary angiography images. The myocardial perfusion in IRA was assessed using the Thrombolysis in Myocardial Infarction (TIMI) flow grading system both before and following PCI [9]. Using the myocardial blush grade (MBG), post-procedural myocardial perfusion was assessed following the methodology outlined by van't Hof et al [10]. TIMI flow less than grade 3 (TIMI 0\2) was considered no-reflow in our study according to it our population study was classified into two groups no-reflow group I and successful flow (TIMI 3) group II ,

Ethical standards

Verbal and written consent was taken from every patient accepted to participate in the study. Study was approved by General Organization for Teaching Hospital and Institutes(GOTHI) with an IRB number coded as HAH00031,date :27\9\2023

STATISTICAL ANALYSIS

The SPSS (21) was utilized to analyze the data. Quantitative data was illustrated as mean standard deviation. The means of the two groups were compared using the Student t-test. To compare between the qualitative data, the Chi-square test was utilized. Univariate and multivariate regression test was applied to assess the predictors of impaired flow. Through the use of a Receiver Operating Characteristic curve (ROC) analysis, a predictive factor cut-off was determined.

RESULTS

The population study was divided into no-reflow group I accounting for 33.3% and successful reperfusion group II accounting for 66.7%. No statistically significant difference between the studied groups regarding age, sex, and risk factors, troponin was statistically significantly higher in successful flow group II 27.878±66.1 compared to 10.657±55.8 in no-reflow group I, successful flow group II presented earlier than no-reflow group I .Table 1

Admission time TPE was significantly prolonged in no reflow group I 97.8±6.1 compared to 81.5 ±9.1

in successful flow group II. cTPE was also more prolonged in the same group in comparison to group II . Admission time TPE\ QT interval was statistically more prolonged in no group I 0.255±0.49 compared to 0.208±0.28 in group II , admission time cTPE\ cQT was more prolonged in group I 0.281±1.94 compared to 0.192±1.07 in group II. Table 2

DTB time \hour was statistically prolonged in no-re flow group I compared to successful flow II MBG 3 was more prevalent in successful flow group II 68.7% compared to 37.5% in No-re flow group I with significant difference . Table 3

After univariate analysis table (4), multivariate analysis showed that Admission time from symptoms onset to hospital admission (0.017), followed by Hs-Troponin peak value pg\ml (0.007), then Admission time cTPE\ cQT (0.001) were significant predictors of impaired flow TIMI <3 post PCI. Table 5

Cut -off value of Admission time cTPE\ cQT in predicting impaired flow (TIMI<3) was ≥ 0.243 ms, sensitivity 78.8%, specificity 66.5 %. Table 6, figure 1

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

Variables	Post PCI TIMI		P
	Group I No-re flow TIMI<3 40 (33.3%)	Group II Successful flow TIMI 3 80 (66.7%)	
Age	55.6±5.5	51.3±1.5	0.645
Sex, male (n) %	20(50%)	45(56.2%)	0.451
Smokers	19(47.5%)	47 (58.7)	0.435
BMI	29.65	27.6	0.348
DM	21 (52.5%)	44 (55%)	0.557
HTN	25(62.5%)	40 (50%)	0.235
Family history of CAD	12(30%)	25(31.25%)	0.767
Admission time from symptoms onset to hospital admission(in hours)	4 (3-7)	2(1-3)	<0.001
Hs-Troponin peak value pg\ml	10.657±55.8	27.878±66.1	<0.001
Triglycerides mg\dl	130.7±17.6	112.9±15.7	0.98
LDL mg\dl	180.9±19.8	186±18.9	0.45

BMI: body mass index, DM: diabetes mellitus, HTN: hypertension , CAD: coronary artery disease LDL: low density lipoprotein , TIMI: thrombolysis in myocardial infarction

Table (2): ECG data of the studied groups

Variables	Post PCI TIMI		P
	Group I No-re flow TIMI<3 40 (33.3%)	Group II Successful flow TIMI 3 80 (66.7%)	
ST segment resolution > 50%	10 (25%)	45(54.8%)	<0.001
Admission time TPE ms	97.8±6.1	81.5 ±9.1	0.038
Admission time cTPE ms	117.8±11.1	101.8±13.1	0.001
Admission time QT ms	383.4±12.4	390.3± 31.5	0.635
Admission time cQT ms	419.3±5.7	422.6±8.5	0.879
Admission time TPE\ QT	0.255±0.49	0.208±0.28	0.002
Admission time cTPE\ cQT	0.281±1.94	0.192±1.07	<0.001

cTPE :corrected T wave peak to end terminal, QT :Q wave to T wave, c QT:corrected Q wave to T wave, TIMI: thrombolysis in myocardial infarction

Table 3: Angiographic data of the studied groups

Variables	Post PCI TIMI		P
	Group I No-re flow TIMI<3 40 (33.3%)	**Group II Successful flow TIMI 3 80 (66.7%)**	
LAD as a culprit	22(55%)	33 (41.2%)	0.456
LCX as a culprit	10(25%)	20(25%)	
RCA as a culprit	8(20%)	27(33.7%)	
MBG 3	15(37.5%)	55(68.7%)	0.001
DTB time \hour	4 (5-7)	1 (0.5-3)	0.002
TIMI flow (baseline)			
0\1	20(50%)	45(56.2%)	0.676
II	20(50%)	35(43.5%)	
Direct stent	25(62.5%)	60(75%)	0.78
Thrombus Aspiration Cath	27(62.5%)	66(82.5%)	0.45
SVD	5(12.5%)	12(15%)	
DVD	10(25%)	25(31.2%)	0.676
TVD	25(62.5%)	43(53.7%)	

LAD: anterior descending artery, LCX: left circumflex, RCA: right coronary artery , DTB:door to ballon time , MBG: myocardial blush grade , TIMI: thrombolysis in myocardial infarction , SVD: single vessel disease, DVD: double vessel disease. TVD: triple vessel disease

Table (4): Univariate logistic regression analysis for prediction impaired flow TIMI < 3

	Coefficient	SE	P	Odds ratio	95% CI
Age (years)	0.076	0.081	0.827	1.047	1.005 to 1.080
Sex	1.650	0.723	0.883	4.711	1.989 to 12.135
Admission time from symptoms onset to hospital admission	0.032	0.008	**0.004***	1.7421	1.008 to 1.048
Hs-Troponin peak value pg\ml	0.495	0.093	**0.03***	1.641	1.367 to 1.970
Admission time cTPE ms	-0.073	0.010	**0.02***	0.820	0.783 to 0.968
Admission time cQT ms	-0.008	0.003	0.128	1.009	1.002 to 1.011
Admission time cTPE\ cQT	-0.0005	0.0001	**0.003***	1.0005	1.0001 to 1.0007
LAD as a culprit	0.096	0.091	0.617	1.033	1.006 to 1.070
TVD	0.078	0.087	**0.04***	1.068	1.007 to 1.098
DTB time \hour	0.345	0.057	**0.006***	1.061	1.786 to 1.980

cTPE :corrected T wave peak to end terminal, QT :Q wave to T wave, c QT:corrected Q wave to T wave, TIMI: thrombolysis in myocardial infarction , LAD: left anterior descending artery , TVD: triple vessel disease, DTB:door to ballon time

Table (5): Multivariate logistic regression analysis for prediction impaired flow TIMI < 3

	Coefficient	SE	t	Odd ratio	P
Age	0.006	0.003	1.675	1.009	0.096
Sex	0.158	0.097	1.621	2.78	0.107
Admission time from symptoms onset to hospital admission	0.003	0.001	2.416	**1.5699**	**0.017***
Hs-Troponin peak value pg\ml	0.063	0.014	4.659	**6.898**	**0.007**
Admission time cTPE ms	-0.012	0.003	-3.97	0.546	0.123
Admission time cQT ms	-0.000	0.000	0.693	1.008	0.489
Admission time cTPE\ cQT	-0.013	0.004	0.467	**2.779**	**0.001***
LAD as a culprit	0.009	0.004	1.899	1.098	0.0989
TVD	0.244	0.089	1.568	1.0456	0.211
DTB time \hour	0.002	0.001	1.675	1.0098	0.134

cTPE :corrected T wave peak to end terminal, QT :Q wave to T wave, c QT:corrected Q wave to T wave, TIMI: thrombolysis in myocardial infarction , LAD: left anterior descending artery , TVD: triple vessel disease, DTB:door to ballon time

Table (6) : cutoff value of Admission time cTPE\ cQT in predicting impaired flow (TIMI<3)

Cutoff	Sensitivity	Specificity	PPV	NPV	P	Accuracy	AUC
≥0.243ms	78.8%	66.57%	51.75	77.8%	0.001	0.80	0.832

PPV: positive predictive value, NPV: negative predictive value, AUC: area under curve

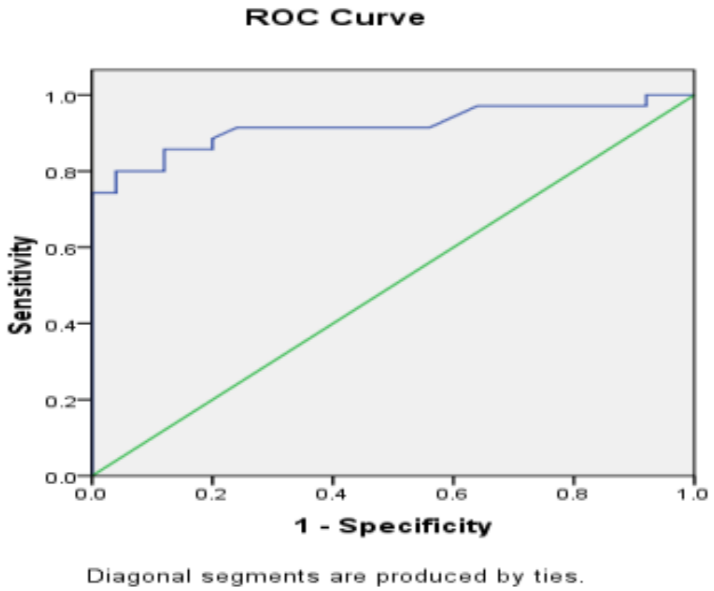

Figure (1): Sensitivity and specificity of Admission time cTPE\ cQT in predicting impaired flow (TIMI<3)

DISCUSSION

The ventricular myocardium is composed of three distinct cell types and is an electrically heterogeneous structure. (the endocardial layer, M cells, and the epicardial layer) that each has unique electrophysiological characteristics [11]. An indicator of the whole dispersion of repolarization (transmural, endocardial , and global) is the time interval between the T wave's peak, which corresponds with the end of repolarization of epicardial cells, and the T wave's end, which coincides with the end of repolarization of endocardial cells [12]. Ventricular arrhythmia development has been observed to be associated with TPE interval [13]. The Tpe interval, the ratio of Tpe, and the Tpe/QTc ratio have been identified as indicators of myocardial perfusion in a previously published clinical research [4]. The current study demonstrated that TPE interval and c TPE\c QT were more prolonged in no-reflow patients compared to successful flow this was concordant with Çoner et al [6] with the difference that his study groups based on STR percentage and patients were managed by thrombolytic therapy. This could be illustrated by the fact that acute MI causes metabolic and electrochemical changes in the myocytes, which in turn influence the pH, ion channel conditions tissue oxygen level, and electrochemical gradient. The a forementioned alterations have a complex effect on the action potential duration in the ischemic tissues; and thus reflected on ECG as TPE and QT intervals changes [14].

We supposed that these metabolic changes reflected on ECG might induce ischemia-reperfusion injury and myocardial reperfusion may not completely recover and so might be associated with no-reflow phenomenon. Çağdaş et al [15] reported that; for patients with STEMI receiving primary PCI, the development of a no-reflow pattern is closely associated with the admission Tpe interval. On the contrary; Duyuler et al [4] reported a nonsignificant difference among his study groups regarding admission TPE or TPE\c QT; this discrepancy might be related to that his study groups were divided upon MBG not TIMI which is more sensitive parameter than TIMI but he concluded that post-PCI TPE and TPE\c QT were more shortened in successful flow MBG 3 group .

Çoner et al [6] revealed acorrelation value for AUC: 0.678; p value<0.001, ROC analysis of the Tpe/QTc ratio also showed an association with the efficacy of reperfusion success. Our study revealed Admission time cTPE\ cQT was one of the significant predictors of impaired flow TIMI <3 post PCI with AUC 0.832 , P<0.001. QT and cQT intervals were in-significantly different in our study this was concordant with; Duyuler et al [4], this could be explained by the fact that QT duration varies with the dynamic changes with ECG and it was one of the study limitations that we assessed ECG changes at a single point at admission time. Bonnemeier et al [16] concluded that the QTc interval slightly lengthens in the first hour and shortens later, and the Tpe interval shortens in the first few hours of acute infarct and stabilizes after three to four hours of revascularization.

Additionally, a thorough assessment of the interval between the chest pain onset and time of admission to medical services is necessary as our study showed that delayed hospital admission was one of the predictors of impaired reperfusion TIMI<3 in regression analysis .

Coner et al [6] reported that the increase in failed reperfusion was found to be related delayed hospital admission (r=0.516; p<0.001). Lastly, troponin was one of the predictors of impaired flow this was in accordance with Refaat et al [17]. This agrees with the fact of the strong association between TIMI flow grade and admission cTnT levels with significant cardiac events. This association is most likely caused by a substantial amount of necrotic tissue, reduced myocardial salvage, and most likely microvascular dysfunction [17].

CONCLUSIONS

TPE and c TPE\ c QT ratio were more prolonged in no reflow patients. Admission time from symptoms onset to hospital admission (0.017), followed by Hs-Troponin peak value pg\ml (0.007),then Admission time cTPE\ cQT (0.001) were significant predictors of impaired flow TIMI <3 post PCI. Admission time cTPE\ cQT at a cut - off value ≥ 0.243 ms could accurately predict impaired flow TIMI <3 post PCI, with sensitivity 78.8% , specificity 66.5 %.

LIMITATIONS

Our study had some limitations such as, the relatively small number of the sample size, being two center study, ECG variables of interest in our study were assessed at a single point at admission time ignoring the dynamic changes of these variables after PCI procedure and lastly clinical adverse cardiac events weren't monitored in our study as ventricular arrhythmias or mortality needing long term of follow up. QT duration prolongation although was non- significant in our study might be because of the presence of non-ischemic causes, such as autonomic alterations, even after successful restoration of tissue perfusion.

RECOMMENDATIONS

The cTPE\c QT variable was a simple sensitive, easy applicable non-invasive parameter that could accurately predict impaired flow TIMI <3 in STEMI patients managed by primary PCI. It is recommended to do an intermediate- to long-term follow-up study to estimate how cTPE\c QT may affect clinical unfavourable cardiac events.

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