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T Peak-to-T End/QT is an Independent Predictor of Early Ventricular Arrhythmias and Arrhythmic Death in Patients with ST Segment Elevation Myocardial Infarction

Mohammed Kassem[1*], Abou Bakr E. Tammam[2], Wael M. Attia[2] and Mohammed Sarhan[2]

[1]*Department of Cardiology, Al Mataria Teaching Hospital, Cairo, Egypt*

[2]*Department of Cardiology, Faculty of Medicine, Al-Azhar University, Egypt*

[*]**Corresponding author:** Mohammed Sarhan, Department of Cardiology , Al Mataria T eaching Hospital, Cairo, Egypt, Phone: 201 1 14807970; E-mail: dr.moh.kassem@gmail.com

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Abstract

Background: The interval from the peak to the end of the T wave (Tp-Te) on 12-lead electrocardiogram (ECG) is a measure of transmural dispersion of repolarization; prolonged Tp-Te interval and Tp-Te/QT ratio have been shown to be markers of arrhythmogenesis in various cardiac disorders.

Aim of the work: To evaluate the usefulness of Tp-Te interval and Tp-Te/QT ratio at admission in patients with acute ST-Segment Elevation Myocardial Infarction (STEMI) undergoing a Primary Percutaneous Coronary Intervention (pPCI) in predicting malignant ventricular arrhythmias and death.

Material and methods: we prospectively studied 100 patients with acute STEMI were treated by pPCI. All patients were subjected to 12-leads resting ECG, echocardiography and laboratory investigations. During hospitalization, patients were monitored for sustained malignant ventricular arrhythmias.

Results: The incidence of ventricular arrhythmia among studied population was 14% (5% had Ventricular Tachycardia (VT) and 9% had Ventricular Fibrillation (VF)), There is statistically significant difference between patients with arrhythmic events and patients with no arrhythmic events regarding Tp-Te tangent (117.9 ± 16.7 ms *vs.* 77.0 ± 10.9 ms, P value<0.001) and Tp-Te/QT (0.332 ± 0.045 *vs.* 0.217 ± 0.033, P value<0.001). On the multivariate analysis only Tp-Te/QT values remained significantly associated with in-hospital life-threatening arrhythmias (OR=2.012, 95%, CI=1.254-4.394, P value=0.002). The incidence of in-hospital death among studied population was 6%. There is statistically significant difference between deaths and survivors regarding Tp-Te tangent (126.7 ± 20.7 ms *vs.* 80.0 ± 14.5 ms, P value<0.001) and Tp-Te/QT (0.359 ± 0.052 *vs.* 0.225 ± 0.042, P value<0.001). On the multivariate analysis only Tp-Te/QT values remained significantly associated with in-hospital death (OR=3.053, 95% CI=1.838-8.467, P value=0.016).

Conclusion: Tp-Te interval and Tp-Te/QT ratio are prolonged in patients with STEMI with malignant ventricular arrhythmias and arrhythmic death. Tp-Te/QT was an independent predictor of early ventricular arrhythmias and death in patients with STEMI. Especially, Tp-Te/QT>0.27 and Tp-Te/QT>0.30 respectively.

Keywords: Tpeak-to-Tend; Arrhythmias; ST elevation myocardial infarction; Dispersion of ventricular repolarization

Introduction

STEMI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent ECG ST elevation and subsequent release of biomarkers of myocardial necrosis. The vast majority of these patients show a typical rise of biomarkers of myocardial necrosis and progress to Q-wave myocardial infarction [1].

Among patients admitted with acute STEMI, 2-10% suffers from malignant ventricular arrhythmias during first hour of Myocardial Infarction (MI). Identification of patients at increased risk of malignant ventricular arrhythmias is critical to the development of effective strategies to prevent sudden cardiac arrest and also to identify patients requiring close monitoring and early and effective therapy to restore sinus rhythm [2].

A prolonged QT interval has been shown to be closely associated with increased risk for Sudden Cardiac Death (SCD) in organic diseases, including myocardial infarction [3].

The Tp-Te interval on 12-lead ECG seems to be a measure of Transmural Dispersion of Repolarization (TDR) in the left ventricle and a prolongation of this interval might represent a period of potential vulnerability for re-entrant ventricular arrhythmias [4]. The Tp-Te interval has been proposed to predict the risk of malignant arrhythmia and SCD in some ion channel diseases [3].

Higher Tp-Te values have been associated with SCD in the general population. Prolonged Tp-Te seems to be also related to increased risk of death and main cardiovascular events in patients undergoing pPCI for STEMI, both in hospital and after discharge. However, little is

known about the dispersion of ventricular repolarization and its relationship with life-threatening arrhythmias in patients with STEMI [5]. So the aim of the study was to evaluate the usefulness of Tp-Te interval and Tp-Te/QT ratio at admission in patients with acute STEMI undergoing pPCI in predicting malignant ventricular arrhythmias and death.

Patients and Methods

This was a prospective observational study that was conducted from June, 2017 to December, 2017 and included 100 patients with acute STEMI presented to the emergency department of Al-Hussein university hospital and were treated using pPCI.

All patients presented, within 12 h from symptoms onset mainly with typical chest pain lasting for at least 30 mins not responsive to nitrates, with ECG showed ST-segment elevation at the J point in at least 2 contiguous leads of 0.2 mV in men or 0.15 mV in women in leads V_2-V_3 and/or of 0.1 mV in other contiguous chest leads or the limb leads according to ESC universal definition of MI [6].

Exclusion criteria were: Previous acute myocardial infarction, Arrhythmias (atrial fibrillation, bundle branch block, pacemaker rhythm), Electrolytes disturbances (low potassium, magnesium, or calcium levels), Significant valvular or congenital heart disease, Patients with Left Ventricular Ejection Fraction (LVEF)<40%, Drug intake causing prolongation of the QT interval (e.g., Amiodarone, Dronedarone, Ibutilide, Quinidine, Procainamide, Disopyramide, Dofetilide, and Sotalol), Family history of sudden cardiac death, Poor ECG quality and Patients with ST-segment elevation>0.1 mV at the J-point in leads V_4, V_5 and V_6.

A standard 12-lead resting ECG was performed on admission to all patients at the setting of 25 mm/mV. QT interval was measured from the onset of the QRS complex to the end of the T-wave (defined as a return to the T-P baseline). If U-waves were present, the QT interval was measured to the nadir of the curve between the T and U waves. Three consecutive cycles were analyzed for each lead. Leads with T-wave amplitude<1.5 mm were excluded from the analysis [7]. The QT intervals were adjusted for heart rate using Bazett [8] and Fridericia's [9] corrections (QTc) and were evaluated in ECGs in which at least six leads were measurable. QT dispersion (QTd) was measured as the difference between the maximum and the minimum QT intervals on the standard 12-lead ECG [10]. Tp-Te interval was assessed in leads unaffected by the infarction where ST-segment deviation was below 0.1 mv at the J-point to avoid problems in assessing QT and Tp-Te measurements. Therefore, Tp-Te was assessed in V_4 or, if this lead was excluded for ST elevation or small T-wave amplitude, in V_5 or V_6 in descending order. Both the tangent and the tail methods were used for each ECG [11] (Figure 1). In the tail method, Tp-Te is the time from the peak of the T wave to the end of the T wave (the point where the descending limb of the wave returns to the isoelectric line) [12]. In the tangent method, the Tp-Te is the time from the peak of the T wave to the intersection between the tangent line on the descending limb of the T wave (through the steepest downslope of the wave) and the isoelectric line [13]. The peak of the T wave was defined as a point of the highest amplitude of the T-wave deflection. If a U wave followed the T wave, the nadir between the T wave and the U wave was considered T-wave offset. If T wave is negative or biphasic, the Tp-Te interval was measured from the lowest point of the first component to the final end of the wave. The Tp-Te/QT ratio was calculated as the ratio of Tp-Te in that lead measured using tangent method to the corresponding QT interval [14]. Standard ECG parameters were also calculated: heart rate, QRS duration and maximal ST elevation.

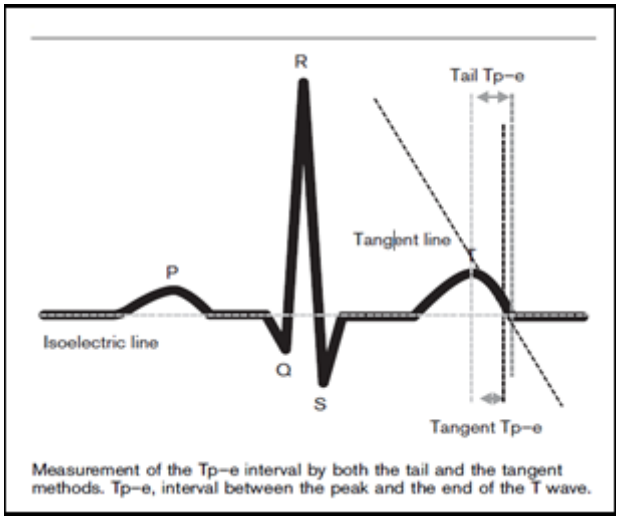


Figure 1: Measurement of the Tp-Te interval [11].

Conventional resting echocardiography was performed within 24 h after admission LVEF was evaluated using modified biplane Simpson's method as recommended by American society of echocardiography guidelines [15].

Serum creatinine and electrolytes (sodium, potassium, magnesium and calcium) were measured at the time of admission prior to the primary PCI procedure.

After angioplasty, all patients were admitted to the coronary care unit, where the conventional anti-ischemic therapies were continued. During their hospital stay, patients were monitored for development of sustained malignant ventricular arrhythmias (sustained ventricular tachycardia or ventricular fibrillation) and arrhythmic deaths were evaluated during the whole hospital stay.

Statistical Analysis

Data were analyzed using Statistical Program for Social Science (SPSS) version 23. Quantitative data were expressed as mean ± Standard Deviation (SD). Qualitative data were expressed as frequency and percentage. P-value ≤ 0.05 was considered significant, P-value ≤ 0.001 was considered as highly significant and P-value>0.05 was considered insignificant. Multivariate analysis was performed using a logistic regression model including all factors that were significantly associated with arrhythmic events and death on univariate analysis.

Result

According to the occurrence of malignant arrhythmia, the patients were classified into two groups; Patients with arrhythmic events (n=14) and Patients with no arrhythmic event (n=86). Arrhythmic events consisted of ventricular tachycardia 5% (5 patients) and 9% ventricular fibrillation (9 patients).

Table 1 summarizes the comparison between patients with arrhythmic events and patients with no arrhythmic events regarding demographic, electrographic, echocardiographic, and angiographic

and laboratory data. There is statistically significant difference between patients with arrhythmic events and patients with no arrhythmic events regarding the history of hypertension (71.4% *vs.* 31.4%, P value=0.004), left ventricular ejection fraction (47.8 ± 7.7% *vs* 53.2 ± 8.9% P value 0.026), QRS duration (83.6 ± 10.8 ms *vs.* 74.9 ± 11.2 ms, P value=0.006) and maximum ST elevation (4.64 ± 1.95 mm *vs.* 3.15 ± 1.65 mm, P value=0.002). The Tp-Te tail (125.7 ± 16.0 ms *vs.* 87.6 ± 17.4 ms, P value<0.001), Tp-Te tangent (117.9 ± 16.7 ms *vs.* 77.0 ± 10.9 m,s P value<0.001) and Tp-Te/QT (0.332 ± 0.045 *vs.* 0.217 ± 0.033, P value<0.001) were prolonged in patients with arrhythmic events.

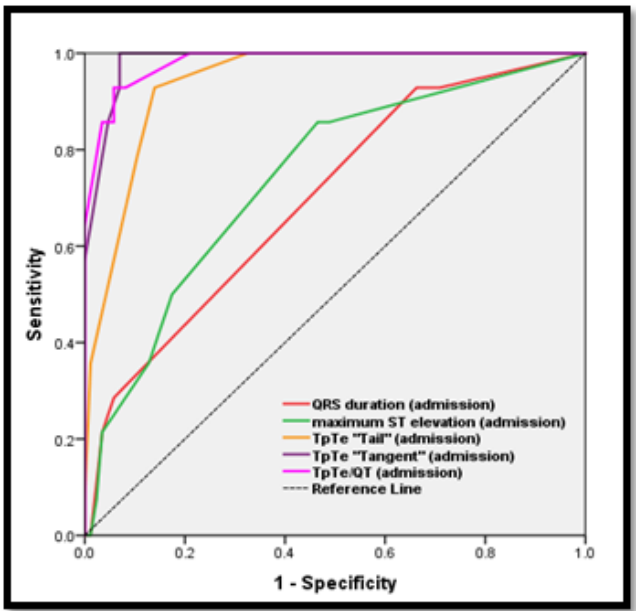
Variables		Arrhythmic events (N=14)	No arrhythmic events (N=86)	P-value
Age (years)		50.8 ± 7.8	52.1 ± 11.6	0.747
Gender	Male	12 (85.7%)	70 (81.4%)	1.00
	Female	2 (14.3%)	16 (18.6%)	
Diabetes mellitus		5 (35.7%)	24 (27.9%)	0.540
Hypertension		10 (71.4%)	27 (31.4%)	0.004
Smoking		10 (71.4%)	61 (70.9%)	1.00
Dyslipidemia		2 (14.3%)	14 (16.3%)	1.00
Family history of IHD		1 (7.1%)	8 (9.3%)	1.00
Type of MI	Anterior MI	8 (57.1%)	49 (57%)	1.00
	Inferior MI	6 (42.9%)	37 (43%)	
LVEF		47.8 ± 7.7	53.2 ± 8.9	0.026
Heart rate (beat/min)		82.4 ± 13.8	80.4 ± 16.7	0.669
QRS duration (ms)		83.6 ± 10.8	74.9 ± 11.2	0.006
Maximum ST elevation (mm)		4.64 ± 1.95	3.15 ± 1.65	0.002
QT interval (ms)		355.7 ± 21.0	357.1 ± 35.3	0.963
QTc interval (ms) "Bazett"		414.7 ± 28.0	408.7 ± 35.1	0.547
QTc interval (ms) (Fridericia)		393.6 ± 19.8	390.0 ± 29.5	0.835
QT dispersion (ms)		38.6 ± 12.3	32.9 ± 9.8	0.103
Tp-Te (ms) (Tail)		125.7 ± 16.0	87.6 ± 17.4	<0.001
Tp-Te (ms) (Tangent)		117.9 ± 16.7	77.0 ± 10.9	<0.001
Tp-Te/QT		0.332 ± 0.045	0.217 ± 0.033	<0.001
The culprit	Proximal LAD	3 (21.4%)	33 (38.4%)	0.288
	Mid LAD	5 (35.7%)	16 (18.6%)	
	RCA	6 (42.9%)	31 (36%)	
	LCX or OM	0 (0%)	6 (7%)	
Na level (mmol/L)		137.0 ± 2.0	137.5 ± 2.6	0.585
K level (mmol/L)		4.0 ± 0.4	3.9 ± 0.4	0.288
Mg level (mEq/L)		2.02 ± 0.15	2.04 ± 0.17	0.777
Ionized Ca level (mg/dL)		4.7 ± 0.2	4.8 ± 0.2	0.308
Serum creatinine (mg/dL)		1.03 ± 0.27	0.91 ± 0.29	0.069

Table 1: Comparison between subgroups of patients with and without arrhythmic events regarding study variables.

Table 2, Figures 2 and 3 represents the ROC curves and their analyses for detection of cut value of arrhythmia occurrence. The ROC curve analysis showed that Tp-Te tangent values>105 ms predicted ventricular arrhythmias with sensitivity of 85.7%, specificity of 95.3% positive predictive value of 75%, negative predictive value of 97.6% with diagnostic accuracy of 94% and Tp-Te/QT values>0.27 predicted death with sensitivity of 92.9%, specificity of 94.2%, positive predictive value of 72.2%, negative predictive value of 98.8% with diagnostic accuracy of 94%.

Cut-off value	Sensitivity %	Specifity %	PPV %	NPV %	Accuracy	AUROC	P-value
QRS duration>75 ms	0.929	0.337	0.186	0.967	0.42	0.699	0.017 (S)
Max. STE>3.5 mm	0.643	0.709	0.265	0.924	0.7	0.741	0.004 (S)
Tp-Te (Tail)>115 ms	0.786	0.895	0.55	0.963	0.88	0.939	<0.001 (HS)
Tp-Te (Tangent)>105 ms	0.857	0.953	0.75	0.976	0.94	0.984	<0.001 (HS)
Tp-Te/QT>0.2722	0.929	0.942	0.722	0.988	0.94	0.982	<0.001 (HS)
LVEF<48.5%	0.643	0.698	0.257	0.923	0.69	0.685	0.027 (S)

Table 2: ROC curve analysis of ECG variables and LVEF regarding incidence of arrhythmic events.



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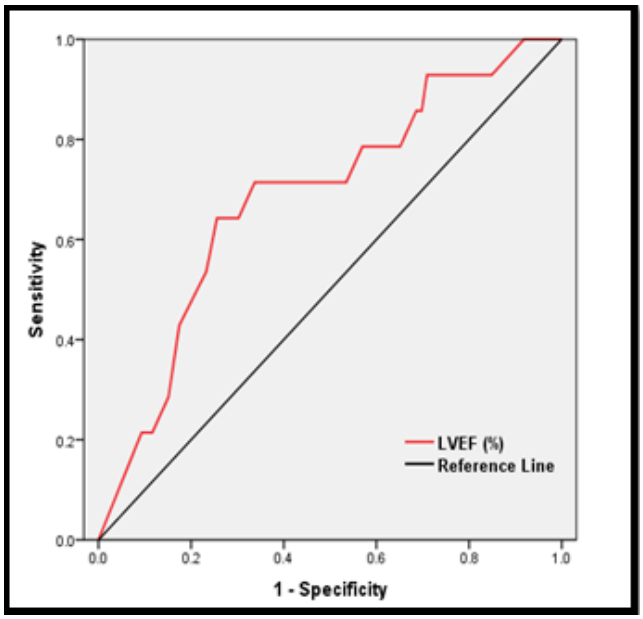

Figure 3: ROC curve analysis of LVEF (%) regarding incidence of arrhythmic events.

Tables 3 and 4 present the univariate and multivariate regression analysis for predicting the arrhythmic events. On the multivariate analysis only Tp-Te/QT values remained significantly associated with in-hospital life threatening arrhythmias (OR=2.012, 95% CI=1.254-4.394, P value=0.002) independently from QRS duration, maximal ST elevation, history of hypertension and LVEF. Tp-Te/QT was the strongest parameter associated with ventricular arrhythmias on the univariate analysis (P value<0.001).

Incidence of arrhythmia (odds ratio)	Significance	Odds ratio	95% C.I for odds ratio	
			Lower	Upper
QRS duration	0.016	1.069	1.013	1.128
Maximum ST elevation	0.007	1.493	1.116	1.996
Tp-Te (Tail)	0.000	1.125	1.061	1.193
Tp-Te (Tangent)	0.000	1.190	1.094	1.296
Tp-Te/QT	0.000	3.030	1.688	5.454
HTN	0.008	5.463	1.572	18.987
LVEF (%)	0.041	0.921	0.851	0.996

Table 3: Univariate regression analysis for predicting arrhythmic events.

Incidence of arrhythmia (odds ratio)	Significance	Odds ratio	95% C.I for odds ratio	
			Lower	Upper
QRS duration	0.935	1.004	0.905	1.114
Maximum ST elevation	0.511	0.769	0.352	1.681
Tp-Te/QT	0.002	2.012	1.254	4.394
HTN	0.215	4.668	0.408	53.455
LVEF (%)	0.593	0.966	0.849	1.098

Table 4: Multivariate regression analysis for predicting arrhythmic events.

According to the occurrence of arrhythmic death, the patients were classified into Deaths group (n=6) and survivors group (n=94).

Table 5 summarizes the comparisons between deaths and survivors groups regarding demographic, electrographic, echocardiographic, angiographic and laboratory data. There is statistically significant difference between deaths and survivors regarding history of hypertension (83.3% *vs.* 34%, P value=0.025), left ventricular ejection fraction (45.3 ± 5.2% *vs.* 52.9 ± 8.9%, P value=0.029), QRS duration (86.7 ± 10.3 ms *vs.* 75.4 ± 11.3 ms, P value=0.024), QT dispersion (43.3 ± 8.2 ms *vs.* 33.1 ± 10.2 ms, P value=0.029) and serum creatinine (1.15 ± 0.31 mg/dl *vs.* 0.91 ± 0.28 mg/dl, P value=0.049). The Tp-Te tail (126.7 ± 20.7 ms *vs.* 90.7 ± 20.0 ms, P value=0.001), Tp-Te tangent (126.7 ± 20.7 ms *vs.* 80.0 ± 14.5 ms, P value<0.001) and Tp-Te/QT 0.359 ± 0.052 *vs.* 0.225 ± 0.042, P value<0.001) were prolonged in patients who died.

Variables		Deaths (N=6)	Survivors (N=94)	P-value
Age (years)		52.2 ± 11.1	51.9 ± 11.2	0.873
Gender	Male	5 (83.3%)	77 (81.9%)	1.00
	Female	1 (16.7%)	17 (18.1%)	

Diabetes mellitus		4 (66.7%)	25 (26.6%)	0.057
Hypertension		5 (83.3%)	32 (34%)	0.025
Smoking		4 (66.7%)	67 (71.3%)	1.00
Dyslipidemia		1 (16.7%)	15 (16%)	1.00
Family history of IHD		1 (16.7%)	8 (8.5%)	0.441
Type of MI	Anterior MI	3 (50%)	54 (57.4%)	1.00
	Inferior MI	3 (50%)	40 (42.6%)	
LVEF		45.3 ± 5.2	52.9 ± 8.9	0.029
Heart rate (beat/min)		82.3 ± 20.2	80.5 ± 16.1	0.907
QRS duration (ms)		86.7 ± 10.3	75.4 ± 11.3	0.024
Maximum ST elevation (mm)		4.67 ± 1.86	3.28 ± 1.73	0.052
QT interval (ms)		353.3 ± 32.7	357.1 ± 33.8	0.709
QTc interval (ms) (Bazett)		408.8 ± 37.5	409.6 ± 34.1	0.956
QTc interval (ms) (Fridericia)		388.7 ± 26.8	390.6 ± 28.5	0.805
QT dispersion (ms)		43.3 ± 8.2	33.1 ± 10.2	0.029
Tp-Te (ms) (Tail)		126.7 ± 20.7	90.7 ± 20.0	0.001
Tp-Te (ms) (Tangent)		126.7 ± 20.7	80.0 ± 14.5	<0.001
Tp-Te/QT		0.359 ± 0.052	0.225 ± 0.042	<0.001
The culprit	Proximal LAD	1 (16.7%)	35 (37.2%)	0.615
	Mid LAD	2 (33.3%)	19 (20.2%)	
	RCA	3 (50%)	34 (36.2%)	
	LCX or OM	0 (0%)	6 (6.4%)	
Na level (mmol/L)		136.3 ± 2.3	137.5 ± 2.5	0.211
K level (mmol/L)		4.0 ± 0.5	3.9 ± 0.4	0.754
Mg level (mEq/L)		1.98 ± 0.19	2.04 ± 0.16	0.39
Ionized Ca level (mg/dL)		4.6 ± 0.1	4.8 ± 0.2	0.132
Serum creatinine (mg/dL)		1.15 ± 0.31	0.91 ± 0.28	0.049

Table 5: Comparison between deaths and survivors regarding study variables.

Table 6, Figures 4 and 5 present the ROC curves and its analyses for detecting the cut values of death occurrence. ROC curve analysis showed that Tp-Te tangent values>110 ms predicted death with sensitivity of 83.3%, specificity of 96.8%, positive predictive value of 62.5%, negative predictive value of 98.9% with diagnostic accuracy of 96% and Tp-Te/QT values>0.30 predicted death with sensitivity of 83.3%, specificity of 95.7%, positive predictive value of 55.6%, negative predictive value of 98.9% with diagnostic accuracy of 95%.

Cut-off value	Sensitivity %	Specifity %	PPV %	NPV %	Accuracy	AUROC	p-value
QRS duration>70 ms	100%	31.9%	8.6%	100%	36%	0.739	0.002 (S)
QT dispersion>20 ms	100%	36.2%	9.1%	100%	40%	0.724	<0.001 (HS)
Tp-Te (Tail)>110 ms	83.3%	84%	25%	98.7%	84%	0.896	<0.001 (HS)
Tp-Te (Tangent)>110 ms	83.3%	96.8%	62.5%	98.9%	96%	0.971	<0.001 (HS)
Tp-Te/QT>0.3056	83.3%	95.7%	55.6%	98.9%	95%	0.972	<0.001 (HS)
Serum creatinine>1.08 mg/dL	66.7%	78.7%	16.7%	97.4%	78%	0.738	0.033 (S)
LVEF ≤ 47%	83.3%	72.3%	16.1%	98.6%	73%	0.766	0.001 (S)

Table 6: ROC curve analysis of ECG variables and LVEF regarding incidence of death.

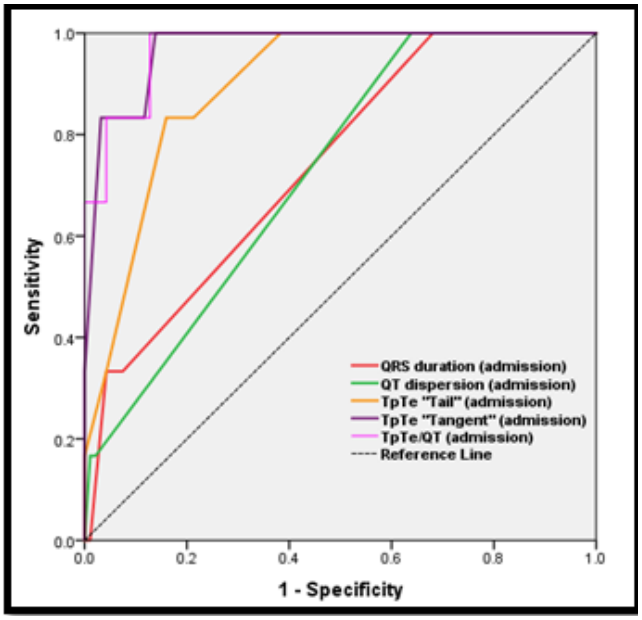

Figure 4: ROC curve of ECG variables regarding incidence of death.

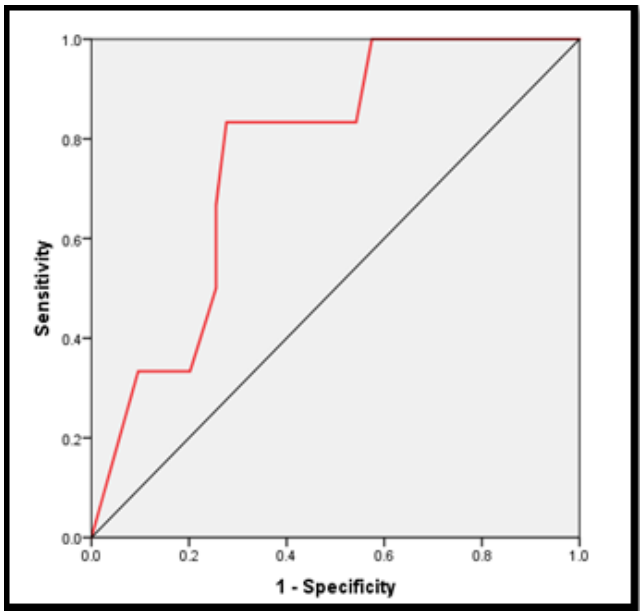

Figure 5: ROC curve of LVEF (%) regarding incidence of death.

Tables 7 and 8 present the univariate and multivariate regression analysis for predicting the arrhythmic death. On the multivariate analysis only Tp-Te/QT values remained significantly associated with in-hospital death (OR=3.053, 95% CI=1.838-8.467, P value=0.016) independently from QRS duration, QT dispersion and history of hypertension. Tp-Te/QT was the strongest parameter associated with death on the univariate analysis (P value=0.003) LVEF and serum creatinine lost significance on the univariate analysis.

Incidence of death (odds ratio)	Significance	Odds ratio	95% C.I for odds ratio	
			Lower	Upper
QRS duration	0.029	1.079	1.008	1.156
QT dispersion	0.026	1.144	1.016	1.287
Tp-Te (Tail)	0.002	1.078	1.028	1.131
Tp-Te (Tangent)	0.014	1.162	1.031	1.31
Tp-Te/QT	0.003	3.722	1.747	6.416
HTN	0.042	0.103	0.012	0.921
LVEF (%)	0.06	0.873	0.757	1.006
Serum creatinine	0.061	8.758	0.902	85.072

Table 7: Univariate regression analysis for predicting incidence of death.

Incidence of death (odds ratio)	Significance	Odds ratio	95% C.I for odds ratio	
			Lower	Upper
QRS duration	0.937	1.006	0.86	1.177
QT dispersion	0.429	1.061	0.916	1.231
Tp-Te/QT	0.016	3.053	1.838	8.467
HTN	0.353	0.164	0.004	7.460

Table 8: Multivariate regression analysis for predicting incidence of death.

Discussion

The mechanism by which Tp-Te interval and Tp-Te/QT ratio are prolonged in ischemia is not known, and various hypotheses have been proposed. Myocardial ischemia results in complex metabolic and ionic changes such as loss of intracellular potassium increase in extracellular potassium, hypoxia, and acidosis that result in transient lengthening followed by shortening of the action potential duration within the ischemic zone. This is caused by a reduction in the transmembrane potential, action potential amplitude and upstroke velocity [16].

The disparity in the action potential duration between ischemic and normal tissue along with the paradoxical lengthening of the refractory period within the ischemic myocardium creates electrophysiological inhomogeneity within the ventricular myocardium and increases the transmural dispersion of repolarization that predisposes to reentrant arrhythmias. This increase in amplitude of dispersion of repolarization is reflected in the surface ECG as prolonged Tp-Te interval and Tp-Te/QT ratio [16].

This study found that patients with malignant arrhythmic events had longer QRS duration than patients without malignant ventricular arrhythmic events (P value=0.006). This is similar to Zabel et al. [17] who studied 280 consecutive patients with acute myocardial infarction over a period of 5 yrs found that patients with arrhythmic events had a mean QRS duration of 104 ± 18 ms and patients with no arrhythmic events had a mean QRS duration of 97 ± 14 ms (P value=0.05) and

discordant with Mugnai et al. [18] found that patients with arrhythmic events had a mean QRS duration of 109 ± 25 ms and patients with no arrhythmic events had a mean QRS duration 99 ± 23 ms (P value=0.2) which may be explained by different type of patient population.

This study found that patients with malignant arrhythmic events had higher maximum ST elevation than patients without malignant ventricular arrhythmias (P value=0.002). This is concordant with Mugnai et al. [18] found that patients with arrhythmic events had a mean maximum ST elevation of 6.3 ± 4.0 mm and patients with no arrhythmic events had a mean maximum ST elevation 4.4 ± 2.3 mm (P value=0.001).

This study found that patients with malignant arrhythmic events had longer mean Tpeak-to-Tend interval measured using both tail and tangent method than patients without malignant arrhythmic events (P value<0.001). This is concordant with Shenthar et al. [14] found that patients with arrhythmic events had a longer mean Tp-Te interval measured by tangent method 200 ± 110 ms while patients with no arrhythmic events had a mean Tp-Te interval 100 ± 20 ms (P value<0.001). Also, patients with malignant arrhythmic events had higher Tp-Te/QT ratio than patients without malignant arrhythmic events (P value<0.001). This is concordant with Mugnai et al. [18] found that patients with arrhythmic events had a mean Tp-Te/QT of 0.38 ± 0.10 while patients with no arrhythmic events had a mean Tp-Te/QT of 0.31 ± 0.08 (P value 0.02) and with Shenthar et al. [14] found that patients with arrhythmic events had a mean Tp-Te/QT of 0.41 ± 0.09 while patients with no arrhythmic events had a mean Tp-Te/QT of 0.26 ± 0.05 (P value<0.001).

This study demonstrated that the best cut-off points of Tp-Te and Tp-Te/QT for prediction of occurrence of malignant ventricular arrhythmias. Tp-Te Tail (ms) was>115, with sensitivity of 78.6% specificity of 89.5%, PPV of 55%, NPV of 96.3% and diagnostic accuracy of 88%. Tp-Te Tangent (ms) was>105, with sensitivity of 85.7% specificity of 95.3%, PPV of 75%, NPV of 97.6% and diagnostic accuracy of 94%. Tp-Te/QT was>0.27, with sensitivity of 92.9% specificity of 94.2%, PPV of 72.2%, NPV of 98.8% and diagnostic accuracy of 94%.

Mugnai et al. [18] found that Tp-Te/QT>0.31 had sensitivity and specificity (69.7% and 63.7%, respectively) along with positive predictive value of 25% and negative predictive value of 92% which was lower values than our study.

Also, Shenthar et al. [14] found that both Tp-Te>100 ms and Tp-Te/QT ratio >0.3 predicted primary VF with a sensitivity of 100%. However, the Tp-Te/QT ratio had a higher specificity (82.9% for Tp-Te/QT ratio *vs* 44.7% for Tp-Te) and overall accuracy (84% for Tp-Te/QT ratio *vs* 48% for Tp-Te) as compared with Tpeak-Tend interval.

In our study we found that only Tp-Te/QT values remained significantly associated with in-hospital life-threatening arrhythmias on the multivariate analysis (OR=2.012, 95% CI=1.254-4.394, P value=0.002) independently from QRS duration, maximal ST elevation, history of hypertension and left ventricular EF. Tp-Te/QT was the strongest parameter associated with ventricular arrhythmias on the univariate analysis (P value<0.001). This is concordant with Mugnai et al. [18] found that only Tp-Te/QT values remained significantly associated with in-hospital life-threatening arrhythmias on the multivariate analysis (OR=1.04, 95% CI=1.003-1.10, P value=0.03) independently from heart rate and maximal ST elevation, which have been diffusely associated with prognosis and severity of myocardial infarction. Tp-Te/QT was the strongest parameter associated with ventricular arrhythmias and arrhythmic mortality on the univariate analysis (OR=1.07, 95% CI=1.03-1.11, P value<0.001).

Also, the study demonstrated that patients who died had longer mean QRS duration than patients who survived (P value=0.024). This was in agreement with Tatlisu et al. [11] found that patients who died had a mean QRS duration of 102.5 ± 22.4 ms and patients who survived had a mean QRS duration of 94.8 ± 17.7 ms (P value 0.02) and discordant with Zabel et al. [17] found that patients who died had a mean QRS duration of 101 ± 23 ms and patients who survived had a mean QRS duration of 98 ± 14 ms. The difference in our results may be related to smaller patient number.

This study found that patients who died had a prolonged mean Tp-Te interval using both tail and tangent methods than patients who survived. This is concordant with Tatlisu et al. [11] found that patients who died had a median Tp-Te interval using both tail and tangent methods 120 ms and 100 ms, respectively. While patients who survived had a median Tp-Te interval using both tail and tangent methods 100 ms and 80 ms, respectively (P value=0.02) and (P value=0.03), respectively and discordant with Zabel et al. [17] who found that patients who died had a mean Tp-Te interval 100 ± 20 ms while patients who survived had a mean Tp-Te interval 96 ± 20 ms. The difference in our results may be related to Zabel et al. [17]. ECG recordings were obtained at the time of hospital discharge.

This study found that patients who died had a higher mean Tp-Te/QT than patients who survived (P value<0.001). This is concordant with Mugnai et al. [18] found that patients who died from arrhythmic death exhibited very prolonged Tp-Te/QT ratio 0.44 ± 11.

This study demonstrated that the best cut-off points of Tp-Te and Tp-Te/QT for the prediction of death. Tp-Te Tail (ms) was>110, with sensitivity of 83.3%, specificity of 84%, PPV of 25%, NPV of 98.7% and diagnostic accuracy of 84%. Tp-Te Tangent (ms) was>110, with sensitivity of 83.3%, specificity of 96.8%, PPV of 62.5%, NPV of 98.9% and diagnostic accuracy of 96%. Tp-Te/QT was>0.30, with sensitivity of 83.3%, specificity of 95.7%, PPV of 55.6%, NPV of 98.9% and diagnostic accuracy of 95%.

Mugnai et al. [18] found that Tpeak-to-Tend/QT ≥ 0.31 for prediction of arrhythmic death showed the best combined sensitivity and specificity (100% and 60.1%, respectively) along with positive predictive value of 5.3% and negative predictive value of 100%.

In our study we found that only Tp-Te/QT values remained significantly associated with in-hospital death on the multivariate analysis (OR=3.053, 95% CI 1.838-8.467, P value 0.016) independently from QRS duration, QT dispersion, history of hypertension. On the univariate analysis both left ventricular EF and serum creatinine lost significance (P value 0.060) and (P value=0.061), respectively. This is concordant with Mugnai et al. [18] found that only Tp-Te/QT values remained significantly associated with arrhythmic death on the multivariate analysis (OR=1.09, 95% CI=1.01-1.19, P value=0.04) independently from heart rate and maximal ST elevation. Tp-Te/QT was the strongest parameter associated with arrhythmic mortality on the univariate analysis (OR=1.10, 95% CI=1.03-1.19, P value=0.008).

Conclusion

- Both Tpeak-to-Tend interval and Tp-Te/QT ratio are more prolonged in patients with arrhythmic events than patients with no arrhythmic events in STEMI patients treated with primary PCI.

- In patients with STEMI, Tp-Te interval measured using tangent method more than 105 ms or Tp-Te/QT ratio more than 0.27, serves as a predictor of malignant ventricular arrhythmias within post STEMI hospital stay.

- Both Tpeak-to-Tend interval and Tp-Te/QT ratio are more prolonged in patients who died than in patients who survived in patients with STEMI treated with primary PCI.

- In patients with STEMI, Tp-Te interval measured using tangent method more than 110 ms or Tp-Te/QT ratio more than 0.30, serves as a predictor of arrhythmic death within post STEMI hospital stay.

Limitations

- Difficulty in recognizing the end of the T wave when the T wave is flat or multiphasic can sometimes affect the measurement of Tp-Te interval and Tp-Te/QT ratio.

- Relatively small sample size of this study and inclusion of patients with STEMI presented only within 12 h of onset of chest pain.

- The utility of the measurements in patients presenting later cannot be commented upon and requires further research.

- The results were obtained from only one center (Al-Hussein university hospital).

- Different operators with variable skills.

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