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QT Interval and P Wave Dispersion in Slow Coronary Flow Phenomenon in Patients with Acute Coronary Syndrome

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ABSTRACT

Background: When there is minimal epicardial coronary stenosis, the coronary slow flow phenomenon (CSFP) is characterized by delayed distal artery opacification. The sluggish velocity of dye in coronary arteries is known as the slow coronary flow (SCF) phenomenon. Dispersion in QT interval and P wave are 2 electrocardiographic findings which can predict predisposing of individuals for developing fatal arrhythmia. The aim of this study was to find association between QT interval and P wave dispersion in acute coronary syndrome patients with CSFP.

Patients and methods: This cross-sectional study was carried out on 200 cases undergoing diagnostic coronary angiography; *Group 1* included 100 patients with acute coronary syndrome and CSFP, and *Group 2* included 100 patients with acute coronary syndrome without CSFP.

Results: We found no statistically significant differences between *Group 1* and *Group 2* according to demographic data, diabetes mellitus, hypertension, dyslipidemia and smoking. QTD, PWD, CTFC LAD, CTFC LCX, CTFC RCA and CTFC were significantly higher among *Group 1***.** QTD showed AUC of 0.996. At best cutoff value of 46.5, sensitivity was 99.75% and specificity was 99.8%. PWD showed AUC of 0.99. At best cutoff value of 23.5, sensitivity was 96.55% and specificity was 98.3%. QT interval showed significant positive correlations with PWD and CTFC. Otherwise, QT interval showed non-significant correlations with other parameters in all studied cases**. Conclusion:** There is an association between QT interval and PWD in acute coronary syndrome patients with slow coronary flow phenomenon**.**

Keywords: Acute Coronary Syndrome, Slow Coronary Flow Phenomenon, QT interval, P wave dispersion.

INTRODUCTION

Most fatal myocardial infarctions were brought on by plaque rupture, which is occasionally referred to as a fissure. These discoveries gave rise to the idea of the high-risk or susceptible plaque, which is distinguished by a thin fibrous cap, a big central lipid core, a surplus of inflammatory cells, and a dearth of smooth muscle cells (SMCs). These findings gave rise to the now-accepted theory that the fissuring of a thin-capped fibroatheroma occurred from an inflammatory-induced weakening of its collagen structure, which led coronary atheromata to become unstable. A measure of inflammation called C-reactive protein (CRP) was present in about half of acute coronary syndromes (ACS) cases [(1)].

In the absence of substantial epicardial coronary stenosis, the coronary slow flow phenomenon (CSFP) is an angiographic clinical entity defined by delayed distal artery opacification. Although interventional cardiologists have been aware of it for around 40 years, the pathogenic mechanisms are still not fully understood. Since CSFP has been connected to clinical signs of myocardial ischemia, life-threatening arrhythmias, sudden cardiac death, and recurring acute coronary syndromes, it has direct clinical consequences rather than being only an angiographic curiosity [(2)].

QT interval extends from the beginning of QRS complex to the end of T wave. Thus, it includes the duration of ventricular depolarization (QRS) and repolarization (J point to end of T wave). It corresponds to the duration of cellular action potential. “long-” and “short”-QT intervals are considered as risk markers for cardiac arrhythmias and sudden death. In the last decade, there have been significant advances in our understanding about measurement and significance of QT interval [(3)].

P-wave dispersion, which is the difference between the highest P-wave duration and the shortest P-wave duration measured from numerous separate Electrocardiographic (ECG) leads, is a contribution to the study of noninvasive electrocardiology. The method for recording and analyzing P-wave inscriptions has been improved, which may lead to the widespread use of this ECG marker in clinical settings, particularly in the determination of atrial fibrillation (AF) risk [(4)].

Eshraghi *et al.* [(5)] evaluated the relation between SCF and presence of P-wave and QT-interval dispersion in electrocardiography. They showed that TIMI Frame Count (TFC) TFC among patients with SCF will result in P wave and QT interval dispersion and therefore this finding can be considered as an indicative marker for cardiac events. Therefore, this study aimed to find the association between QT interval and P wave dispersion in ACS patients with slow coronary flow phenomenon.

PATIENTS AND METHODS

Our cross-sectional study included patients with unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI) candidate for coronary angiography at Cardiology Department, Zagazig University Hospitals.

The patients were divided into:

Group 1 included patients with acute coronary syndrome and slow coronary flow, and *Group 2* included patients with acute coronary syndrome without slow coronary flow.

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Exclusion criteria

Patients with cardiomyopathies, valvuler heart disease, renal failure, conduction abnormalities, atrial arrhythmias, electrolyte imbalance, and patients on drugs affecting QT interval.

Methods:

1. Complete history taking: Detailed present history included age, gender, presenting complaint, important associated symptoms (dyspnea), and drugs taken. Past history included diabetes mellitus, hyperlipidemia, systemic hypertension, cigarette smoking. Finally, patients were asked about family history of diabetes, hypertension and coronary artery diseases.

2. General and local examination was done included blood pressure (systolic and diastolic) (mmHg) and heart rate (beats/min) and rhythm.

3. Cardiac examination: For murmurs and additional sounds.

4. Chest examination: For detection of fine basal crepitations.

5. Electrocardiography: Standard 12 lead ECG with speed 50mm/sec, QT dispersion (based on the difference between maximum and minimum QT) and P dispersion (based on the difference between maximum and minimum P wave duration) were calculated.

6. Echocardiography: A standard transthoracic echocardiogram (TTE) was performed using commercially available systems. Images were obtained using a 2.5 MHz transducer.

7. Coronary angiographic examination:

All the angiographies were performed by two expert interventional cardiologists who were blinded to the clinical details of the study. SCF was identified in normal coronary vessels by use of TIMI frame count (TFC) method in at least one of the main coronary vessels. Study data including TFC of the three main coronary arteries, maximum and minimum of QT and P wave duration in both groups were analyzed. TFC value greater than 27 was considered as SCF. While normal frames for left anterior descending artery (LAD) were 1.7 times more than mean value of right coronary artery (RCA) and left circumflex artery (LCX), the mean corrected TFC (CTFC) values were calculated as follow: CTFC mean = 1/3 (LAD/1.7 + RCA + LCX).

Ethical consent:

An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

Data entered and analyzed using Microsoft Excel software. Data were then imported into Statistical Package for the Social Sciences (SPSS version 20.0) software for statistical analysis. According to the type of data; qualitative represent as number and percentage and quantitative continues group represent by mean and standard deviation (SD). The following tests were used to test differences for significance; difference and association of qualitative variable by Chi square test (X^2).

Differences between quantitative independent groups by Student's t test. P value was set at $\leq$0.05 for significant results and $\leq$0.001 for high significant result.

RESULTS

Table 1 summarizes the distribution of the basic demographic data and medical history of the 2 studied groups. No significant difference founded between groups regard age and any other parameters.

Table (1): Basic demographic data and medical history distribution between studied groups.

Variable			Group 1 (N=100)	Group 2 (N=100)	t/ X^2	P-value
Age			47.78±12.0	51.50±8.73	1.771	0.080
Sex	Female	N	36	48	1.47	0.22
		%	36.0%	48.0%		
	Male	N	64	52		
		%	64.0%	52.0%		
DM	-VE	N	68	68	0.0	1.0
		%	68.0%	68.0%		
	+VE	N	32	32		
		%	32.0%	32.0%		
HTN	-VE	N	42	50	0.64	0.42
		%	42.0%	50.0%		
	+VE	N	58	50		
		%	58.0%	50.0%		
CAD	-VE	N	76	66	1.21	0.27
		%	76.0%	66.0%		
	+VE	N	24	34		
		%	24.0%	34.0%		
Dyslipidemia	-VE	N	84	80	0.27	0.60
		%	84.0%	80.0%		
	+VE	N	16	20		
		%	16.0%	20.0%		
Family history	-VE	N	66	74	0.76	0.38
		%	66.0%	74.0%		
	+VE	N	34	26		
		%	34.0%	26.0%		
Smoker	Non	N	54	64	1.03	0.30
		%	54.0%	64.0%		
	Smoker	N	46	36		
		%	46.0%	36.0%		
Total		N	100	100		
		%	100.0%	100.0%		

Majority of patients had no Ischemic changes and NSTEMI with any significant difference between groups **(Table 2)**.

Table (2): Ischemic changes and infarction distribution between studied groups.

Variable			Group		X^2	P-value
			Group 1	Group 2		
Ischemic changes	No	N	54	68	2.06	0.15
		%	54.0%	68.0%		
	Yes	N	46	32		
		%	46.0%	32.0%		
STEMI	No	N	96	96	0.0	1.0
		%	96.0%	96.0%		
	Yes	N	4	4		
		%	4.0%	4.0%		
NSTEMI	No	N	76	86	1.62	0.202
		%	76.0%	86.0%		
	Yes	N	24	14		
		%	24.0%	14.0%		
Total		N	100	100		
		%	100.0%	100.0%		

There was no significant difference found between groups as regard Troponin and CKMB **(Table 3)**.

Table (3): Troponin and CKMB distribution between studied groups.

Variable	Group 1	Group 2	Mann-Whitney	P-value
Troponin	0.1 ± 0.01	0.1 ± 0.01	0.358	0.845
CKMB	14.0 ± 3.12	12.0 ± 2.64	1.321	0.298

QTD, PWD, CTFC LAD, CTFC LCX, CTFC RCA and CTFC means were significantly higher among group 1 **(Table 4)**.

Table (4): ECG and ECHO parameters distribution between studied groups.

Variable	Group 1	Group 2	t	P-value
QTD	85.48 ± 12.8	36.56 ± 4.92	25.211	0.000
PWD	40.98 ± 9.1	18.06 ± 2.92	16.946	0.000
EF	59.94 ± 6.56	58.18 ± 4.99	1.508	0.135
CTFC LAD	23.62 ± 5.27	14.52 ± 2.29	8.437	0.000
CTFC LCX	20.26 ± 4.80	11.70 ± 1.41	12.071	0.000
CTFC RCA	20.08 ± 4.82	11.46 ± 1.44	10.153	0.000
CTFC mean	20.8 ± 2.71	11.06 ± 1.05	23.671	0.000

Regarding UA and RWMA distribution, there was no significant difference found between groups **(Table 5, Figure 1)**.

Table (5): UA and RWMA distribution between studied groups.

Variable			Group		X^2	P-value
			Group 1	Group 1		
UA	**-VE**	**N**	30	18	1.97	0.16
		%	30.0%	18.0%		
	+VE	**N**	70	82		
		%	70.0%	82.0%		
RWMA	**-VE**	**N**	72	66	0.42	0.51
		%	72.0%	66.0%		
	+VE	**N**	28	34		
		%	28.0%	34.0%		
Total		**N**	100	50		
		%	100.0%	100.0%		

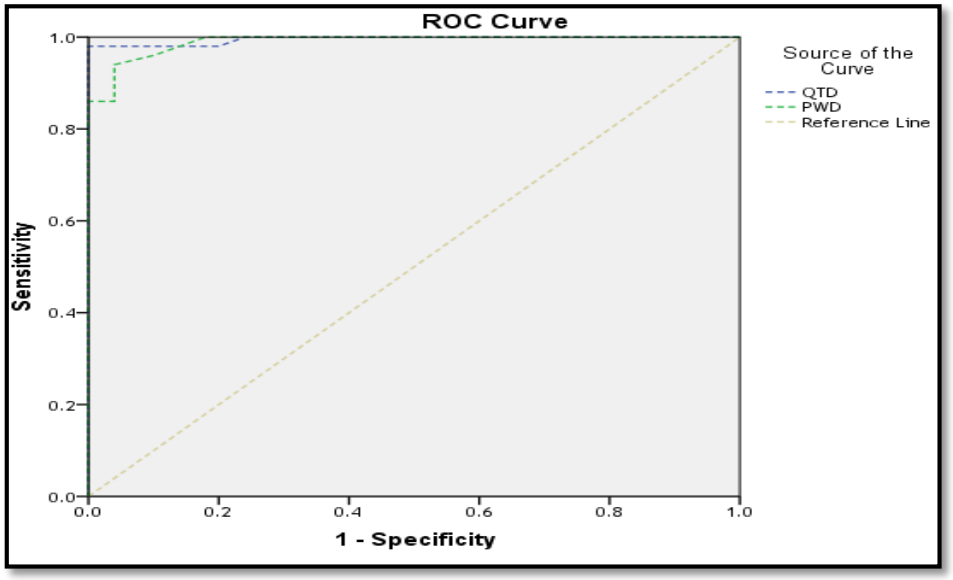

Figure (1): ROC curve for suggested slow flow cutoff regard QTD and PWD

A significant area under curve with valid cutoff >46.5 and >23.5 respectively with sensitivity 99.75% and 96.55% and specificity 96.55% and 98.3% respectively **(Table 6, Figure 2)**.

Table (6): Validity of QTD and PWD among the studied patients.

Test Result Variable(s)	Area	Cutoff	P	95% Confidence Interval		Sensitivity	Specificity
				Lower Bound	Upper Bound		
QTD	0.996	>46.5	0.00**	0.986	1.000	99.75%	99.8%
PWD	0.990	>23.5	0.00**	0.977	1.000	96.55%	98.3%

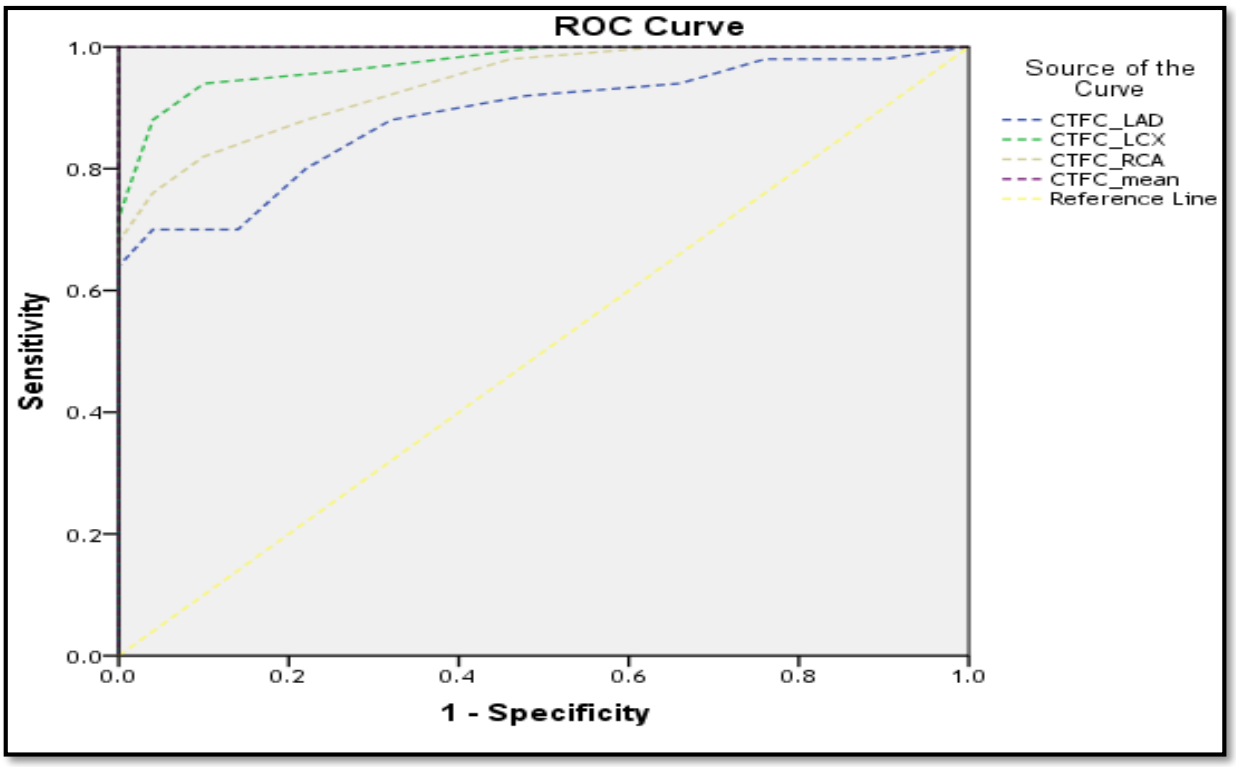

Figure (2): ROC curve for suggested slow flow cutoff regard CTFC

A significant area under curve with valid cutoff >16.5, >14.0, >12.5 and >14.5 respectively with sensitivity 80.0%, 94.5%, 83.3% and 100.0% and specificity 78.0%, 98.3%, 92.8%, 85.0% and 100.0% respectively **(Table 7)**.

Table (7): Validity of CTFC among the studied patients.

Test Result Variable(s)	Area	Cutoff	P-value	95% Confidence Interval		Sensitivity	Specificity
				Lower Bound	Upper Bound		
CTFC_LAD	0.884	>16.5	0.00**	0.817	0.951	80.0%	78.0%
CTFC_LCX	0.974	>14.0	0.00**	0.948	1.000	94.5%	92.8%
CTFC_RCA	0.940	>12.5	0.00**	0.897	0.982	83.3%	85.0%
CTFC mean	1.000	>14.5	0.00**	1.000	1.000	100.0%	100.0%

QTD was significantly positive correlated with PWD and also with all CTFC **(Table 8)**.

Table (8): Correlation with QTD among the studied patients.

Variable		QTD
PWD	r	0.862
	P	0.000
SBP	r	0.031
	P	0.759
DBP	r	0.024
	P	0.811
HR	r	-0.052-
	P	0.610
Troponin	r	0.163
	P	0.105
CKMB	r	0.158
	P	0.116
EF	r	0.104
	P	0.305
CTFC_LAD	r	0.580
	P	0.000
CTFC_LCX	r	0.716
	P	0.000
CTFC_RCA	r	0.700
	P	.000
CTFC mean	r	0.876

A case of 45-years-old man with history of hypertension, smoker presented with typical recurrent chest pain, Serial ECG and troponin were done. PWD was 49 ms, QTD was 80, Tnt at admission time was 4.2 ng\L. Other labs were done, all within normal. Ischemic heart disease with presserved systolic function, RWMA: hypokinesia in anterior and antero lateral wall segment. CTFC (LAD) was 39 f/s , CTFC(LCX) : 27f/s , CTFC (RCA): 14 f/s, CTFC mean was 21 f/s. Patient was managed according to latest guidelines **(Figure 3)**.

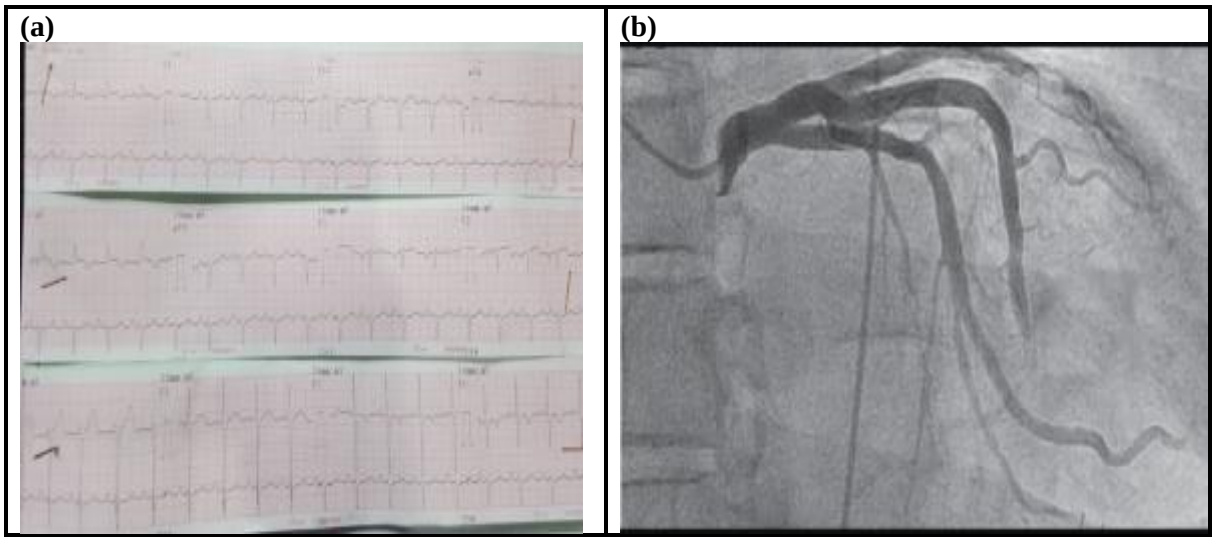

Figure (3): A case of 45-years-old man, troponin were done. PWD was 49 ms , QTD was 80, Tnt at admission time was 4.2 ng\L. (a): ECG at admission time with ischemic changes inverted T wave In Lead I , AVL , V4-V6; (b) RAO caudal view showing significant LAD slow flow.

A case of 65-years-old woman with history of hypertension, diabetes presented with typical recurrent chest pain,ECG and troponin were done. PWD was 45ms, QTD was 73. **Tnt at admission time:** 0.01ng\L and **after 2 hours was** 0.01 ng/L. Good LV systolic function , no RWMA, mild diastolic function. CTFC (LAD) was b15f/s, CTFC (LCX) was 16f/s, CTFC(RCA) was 27 f/s, CTFC mean was 20 f/s. Patient was managed according to latest guidelines **(Figure 4)**.

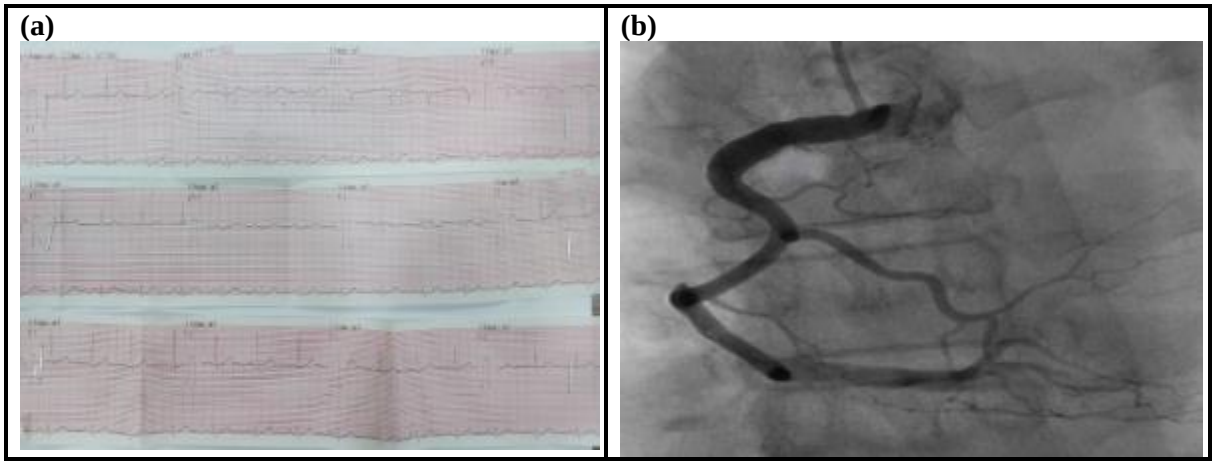

Figure (4): A case of 65-years-old woman presented with typical recurrent chest pain, ECG and troponin were done. PWD was 45ms, QTD was 73; (a) ECG at admission time was within normal; (b) Cranial view showing ectasia and significant RCA slow flow.

DISCUSSION

Some cardiac consequences, including ST-elevation myocardial infarction (MI) and arrhythmias, are characterized by the slow coronary flow (SCF) phenomenon. Patients with patent arteries are considerably more at risk for having arrhythmias than those with fragile myocardium, such as those with ischemic heart disease [(6)].

Two electrocardiographic findings, QT interval dispersion and P wave, can identify who is more likely to develop a fatal arrhythmia. Increased ventricular arrhythmias, cardiac death, and overall mortality are associated with QT interval dispersion [(7)].

P-wave dispersion (PWD), a novel ECG marker in addition to QT interval dispersion, has been connected to irregular and uneven sinus impulse propagation. A number of cardiovascular diseases, including diabetes mellitus, obesity, hypertension, peripheral vascular disease, and myocardial infarction, are more common among people with greater PD levels. Additionally, it has been discovered that PD is linked to a rise in carotid intima-media thickness and inflammatory indicators such C-reactive protein [(8)].

The P wave and QT interval dispersion is an interesting area of research and there is not enough evidence available for evaluation of these electrocardiographic findings among patients undergoing SCF phenomenon. According to prevalence of arrhythmias in SCF and predicting role of electrocardiographic findings such as P wave and QT interval dispersion for arrhythmias, we tried in this cross-sectional study to find the possible association between QT interval and P wave dispersion in acute coronary syndrome patients with slow coronary flow phenomenon.

This cross-sectional study was carried out on 200 cases undergoing diagnostic coronary angiography; Group 1 included 100 patients with acute coronary syndrome and slow coronary flow. Group 2 included 100 patients with acute coronary syndrome without slow coronary flow. Age was distributed as 47.78 (SD 12) and 51.5 (SD 8.73), respectively. Regarding sex distribution, males represent 58% and females represent 42%**.** We found no statistically significant difference between group 1 and group 2 according to demographic data.

Akin *et al.* [(9)] showed that there was no difference in comparison of groups with regard to age. **Eshraghi *et al.*** [(5)] evaluated the relation between SCF and presence of P-wave and QT-interval dispersion in electrocardiography. A total of 87 patients (47 patients in case group and 40 patients in control group) were participated in this study. Mean and SD of age in normal and SCF groups were 53.78 (SD 9.72) and 51.62 (SD 7.35), respectively. The age distribution in both groups was normal and groups were homogenous for gender.

In our study, DM patients represent 32% from studied participants, hypertension 54% and dyslipidemia 18%. Statistically, there were no significant differences between both groups according to diabetes mellitus, hypertension, dyslipidemia and smoking. Also, **Akin *et al.*** [(9)] showed that there was no

difference in comparison of groups with regard to hypertension, diabetes and smoking and **Eshraghi *et al.*** [5] stated that smoking, having DM, hypertension, or hyperlipidemia were not significantly different between both groups.

Ramakrishnan *et al.* [10] reported that dyslipidemia, smoking, and hypertension were significantly associated with SCF and recommended endothelial dysfunction as a significant contributor in SCF phenomenon. Similarly, in a study by **Sanati *et al.*** [11], it was concluded that low level of high hyperalphalipoproteinemia (HDL-c) and hypertension were independent predictors of SCF phenomenon.

Some authors have evaluated the relation of more parameters and SCF phenomenon. **Naing and Qiu** [12] reported uric acid level as an independent predictor of SCF phenomenon. **Hawkins *et al.*** [13] reported obesity as an independent predictor of this phenomenon.

In our study, QTD, PWD, CTFC LAD, CTFC LCX, CTFC RCA and CTFC were significantly higher among *Group 1*. **Özcan *et al.*** [14] have reported that prolonged Pd is associated with stable angina pectoris and acute coronary syndrome. Considering that CSFP is a variant of ischemic heart disease, Pd is likely to be increased in patients with CSFP.

Yılmaz *et al.* [15] investigated the presence of a relationship between Pd and QTcd and found that Pmax, Pd, QTcmax, QTcmin and QTcd were higher in the Coronary slow flow phenomenon (CSFP) group than in the normal coronary artery (NCA) group. **Eshraghi *et al.*** [5] showed that QT interval and PWD, mean CTFC, and TFC in 3 coronary vessels were significantly different in both groups. These variables were not normally distributed in groups.

P wave and QT interval dispersion can be seen with SCF. **Mahmoud** [16] evaluated P-duration, PD and QT dispersion in patients with CSF and its relationship with Thrombolysis in Myocardial Infarction frame count in comparison to normal subjects and had found that SCF phenomenon was associated with dispersion of P wave and QT interval. Also, **Mahfouz *et al.*** [17] evaluated the role of non-invasive measures in predicting primary coronary slow flow (PCSF) patients and have approved that QT interval and PWD were associated with SCF. These studies concluded that PCSF is associated with diabetes, greater PWD and QTc dispersion, higher HCT and HsCRP levels. However, there are some clinical conditions with significant impact on electrocardiographic findings, especially QT interval dispersion.

As an example, there are some environmental causes for QT dispersion such as smoking. **Akbarzadeh *et al.*** [18] reported that even smoking a single cigarette among nonsmokers will increase QT dispersion. Moreover, **Kelmanson** [19] had shown that anxiety in clinically healthy patients would affect QT interval dispersion and predispose patients to develop arrhythmias.

Receiver operating characteristic (ROC) curve of QTD and PWD was conducted for discrimination between group I and Group II. QTD showed AUC of 0.996. At best cutoff value of 46.5, sensitivity was 99.75% and specificity was 99.8%. PWD showed AUC of 0.99. At best cutoff value of 23.5, sensitivity was 96.55% and specificity was 98.3%.

In our study, QT interval showed significant positive correlations with PWD and CTFC. Otherwise, QT interval showed non-significant correlations with other parameters in all studied cases. Also, **Eshraghi *et al.*** [5] showed that CTFC showed significant correlations with QT disturbance and PWD, which were absent in normal flow group, indicating that QT interval and PWD significantly increased with increasing the CTFC among patients with SCF. Therefore, this finding can be considered as an indicative marker for cardiac events.

Heterogeneous nature of this phenomenon might be the explanation of its association with different comorbidities [13]. **Sanati *et al.*** [11] suggested that a possible reason for different predictors in different studies could be an unknown confounder.

There is no consensus on the treatment protocols for SCF patients. Generally, as in coronary artery disease patients, those cases are treated by agents against angina attacks such as organic nitrates, beta–blockers and calcium channel blockers along with acetylsalicylic acid and statin therapy against a possible atherosclerotic pathogenesis.

However, these therapies often fail to control the angina attacks. In our study, we did not focus on the efficacy of a certain agent, however, because of patients with SCF have atherosclerosis, inflammation, endothelial dysfunction, and elevated thrombogenicity, they may need an aggressive antiplatelet therapy with modification of risk factors.

Although the cases in this study were matched and the study protocol considered many of the confounding factors; however, due to various effective factors on electrocardiogram (ECG), considering an exact conclusion about the effect of SCF on P wave and QT interval is difficult. However, from the results of the present study, it be concluded that there is an association between QT interval and PWD in acute coronary syndrome patients with slow coronary flow phenomenon.

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