Journal of Cardiovascular Disease Research
ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 07, 2021

Relation between QT and P wave Dispersions in Coronary Slow Flow patients

Salwa Mohamed Ghoneim (1), **Ghada Ibrahim Mohamed** (1), **Mohamed Maher Ahmed Allam** (2) **and Ahmed Ali Othman** (3)

(1) Professor of Cardiology, **Faculty of Medicine- Zagazig University**

(2) Lecturer of Cardiology, **Faculty of Medicine- Zagazig University**

(3) M.B.B.Ch, Faculty of Medicine - Garyounis University, Libya

Corresponding author

Ahmed Ali Othman

M.B.B.Ch, Cardiology Department, Faculty of Medicine - Garyounis University, Libya

E-mail : ahmadelmansory46@gmail.com

tel: 201026754002

Abstract

Background: Slow coronary flow (SCF) is defined as a slow progression of the contrast to distal vessels in the absence of any stenosis during angiography. The aim of this work was to investigate the relation between QT interval, p wave dispersions in patient with coronary slow flow. **Methods:** This case control study was conducted on 60 patients whom undergoing coronary angiography (CAG) in Cardiology Department at Zagazig University Hospitals during the period from October 2019 to March 2020. The study population was divided into two groups: **CSF Group:** included 30 patients with normal coronary arteries (obstructive lesion <40%) and slow flow phenomenon. **Control Group:** included 30 patients with normal coronary arteries and normal coronary flow. All patients were subjected to full history taking, Laboratory investigations, Electrocardiograms, Echocardiology and Coronary angiography**. Results:** thrombolysis in myocardial infarction (TIMI) Frame count [left anterior descending (LAD), left circumflex (LCX) and right coronary artery (RCA)] showed significant difference being higher in CSF group compared with control group. There was a significant positive correlation between P-wave dispersion (PD) and LAD, LCX and RCA TIMI frame count**.** There was a significant positive correlation between QT dispersion (QTD) and LAD, LCX and RCA TIMI frame. **Conclusion:** The study showed that there was a significant positive correlation between PD, QTD with TIMI frame of CSF (LAD, LCX and RCA) patients. So, with increasing QTD and PD among patients with CSF can be considered as indications markers risk for arrhythmia and related adverse cardiac events.

Keywords: Coronary Angiography, Electrocardiography, Cardiac Arrhythmias, Coronary Slow Flow

INTRODUCTION

Coronary artery disease (CAD) is a progressive disease characterized by various symptoms and findings ranging from angina pectoris to sudden cardiac death as a result of decreased blood flow during rest or exercise due to the characteristics of coronary artery lesions.1) Diagnosis of CAD is made by non-invasive (electrocardiography [ECG], exercise stress test, myocardial perfusion scintigraphy, coronary computed tomography) and invasive (coronary angiography) methods [1].

CSFP is a coronary microvascular disorder.2 It is clinically distinct from other coronary microvascular disorders3 and is important to diagnose as effective therapies have been described [2].

The difference between the longest and shortest QT intervals in a 12-lead ECG is called QTd, whereas the QT interval corrected for heart rate is called corrected QTd (QTcd). Increased QTd has been reported in individuals with myocardial ischaemia. It has been reported that increased QTd is an indicator of increased risk for the development of ventricular tachycardia in patients with cardiac disease and that the measurement of QTd may help to predict which patients are most likely to develop life-threatening arrhythmias [3].

The increased QTd on electrocardiogram (ECG) indicates non-uniform ventricular repolarization and may result in increased vulnerability to malignant ventricular arrhythmia. The greater the QTd, the lower is the homogeneity of ventricular repolarisation and the higher is the ventricular instability. It is believed that the homogeneity of the total duration of ventricular depolarisation and repolarization prevents arrhythmias. P wave dispersion (PWD) is considered as an electrocardiographic marker for prediction of idiopathic paroxysmal atrial fibrillation and even its recurrence[4].

In addition to PWD and QT interval dispersion are also related to increased ventricular arrhythmias, cardiac death, and total mortality. [2].

The QT interval dispersion and PWD is an interesting area of research and there is not enough evidence available for evaluation of these electrocardiographic findings among patients with SCF phenomenon. According to prevalence of arrhythmias in SCF and predicting role of electrocardiographic findings such as P wave and QT interval dispersion possible relation between SCF and P wave and QT interval dispersion will be evaluated in this case control study.

AIM OF THE WORK

The aim of this work was to investigate the relation between QT interval and p wave dispersions in patient with coronary slow flow.

PATIENTS AND METHODS

This case control study was conducted on 60 patients whom undergoing coronary angiography (CAG) at Cardiology Department at Zagazig University Hospitals during the period from October 2019 to March 2020. Written informed consent was obtained from all participants and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) [5] for studies involving humans.

Inclusion criteria: Patients referred for coronary angiography because of suspected coronary artery disease.

Exclusion criteria: Patients with coronary artery disease (CAD) who had atherosclerotic plaques in coronary arteries angiographically, Patients with acute coronary syndrome, Patients with decompensated heart failure, Patients with congenital heart disease, significant valvular heart disease. Patients who had evidence of obstructive coronary artery disease. Patients with small P amplitude or patients with undetected P-wave end point. Atrial fibrillation. Bundle branch block. Pre-exitation syndrome. Chronic COPD. Any history of systemic diseases.

The study population was divided into two groups:

CSF Group: included 30 patients with normal coronary arteries (obstructive lesion <40%) and slow flow phenomenon.

Control Group: included 30 patients with normal coronary (obstructive lesion <40%) arteries and normal coronary flow

The SCF phenomenon will be documented angiographically as normal or near normal coronary arteries with less than 40% stenosis and Thrombolysis in Myocardial Infarction (TIMI-2) flow's in at least one major, coronary arteries [6].

Patient's evaluation

All patients underwent a detailed evaluation including, medical history, physical examinations, History of age, gender, hypertension (HTN), diabetes mellitus (DM), Smoking, Laboratory investigations (HB, WBC and platelets count, Fasting blood sugar, Blood urea, creatinine, S. cholesterol, S. TG, S. LDL (c) and S. HDL (c).

Electrocardiograms: All subjects underwent a routine standard 12-lead surface ECG recorded at a paper speed of 25 mm/s and gain of 10mm/mV (Cardiofax V, Nihon Kohden Corp., Tokyo, Japan) in the supine positionand were breathing freely but not allowed to speak during the ECG recording. ECG's were transferred to a personal computer via a scanner and then magnified 400 times by Adobe Photoshop software [7]. P-wave &QT-interval duration measurement was then marked with computer cursor manually by visual inspection and the distance was marked by cursor automatically calculated by using computer software (Image J, NIH, Bethesda, MD, USA) **[8].**

The starting point of P-wave was referred as the start of the positive deflection crossing the isoelectric line and the end point was referred as the end of the deflection crossing the isoelectric line. P-wave duration was measured from the onset to the offset of the P-wave. We accepted maximum (Max.) P-wave duration as longest P-wave duration and minimum (Min.) P-wave duration as the shortest P-wave duration. P-wave dispersion that is defined as the difference between the Max. P-wave duration and the Min. P-wave duration was also calculated. The subjects were excluded if these points were not clear. QT interval which is the duration between the beginnings of QRS complex

to the end of T-wave at the level of the TP isoelectric baseline. When U-waves was present, the QT intervals was measured from beginning of the QRS complex to nadir of the curve between the T- and U waves **[9]**. The longest QT interval (Max. QT) and the shortest QT interval (Min. QT) were measured. QT interval dispersion that is defined as the difference between the Max. QT duration and the Mini QT. All the measurements were repeated three times and average values were accepted for each of ECG values. All the measurements were performed by two experienced investigators unaware of the subject's clinical status [7].

Echocardiology

All patients were examined in left lateral and supine postion by precordial M-mode, two-dimensional (2D), left ventricular (LV) diameter and wall thickness were measured targeted M-mode. Ejection fraction (EF) was measured using modified Simpson's bipolar method, each representive value was obtained from the average of three following measurements

1- left ventricular diastolic diameter
2- left ventricular systolic diameter
3- Ejection fraction

Coronary angiography

All patients underwent selective coronary angiography. Coronary flow rates of all subjects were documented by Thrombolysis in Myocardial Infarction (TIMI) frame count. Thrombolysis in Myocardial Infarction frame count method is a simple, reproducible, objective, and quantitative index of coronary flow velocity. It has been suggested that a higher TIMI frame count may reflect disordered resistance vessel function**.** Thrombolysis in Myocardial Infarction frame count was determined for each major coronary artery in each patient and control subject according to the method first described by **Gibson et al. [10].** Briefly, the number of cineangiographic frames, recorded at 30 frames per second, required for the leading edge of the column of radiographic contrast to reach a predetermined landmark is determined. The first frame is defined as the frame in which concentrated dye occupies the full width of the proximal coronary artery lumen, touching both borders of the lumen and forward motion down the artery.

The final frame is designated when the leading edge of the contrast column initially arrives at the distal landmark.

In the left anterior descending (LAD) coronary artery, the landmark used is the most distal branch nearest the apex of the left ventricle, commonly referred as the whale's tail. Left anterior descending coronary artery is usually longer than the other major coronary arteries [11]; the TIMI frame count for this vessel is often higher. To obtain corrected TIMI frame count for LAD coronary artery, TIMI frame count was divided by 1.7 **[10].** The right coronary artery (RCA) distal landmark is the first branch of the posterolateral

RCA after the origin of the posterior descending artery, regardless of the size of this branch. The branch of the left circumflex (LCx) artery that encompassed the greatest total distance traveled by contrast was used to define the distal landmark of the LCx artery. Thrombolysis in Myocardial Infarction frame count in the LAD and LCx arteries was assessed in a right anterior oblique projection with caudal angulation and RCA in left anterior oblique projection with cranial angulation.

Statistical Analysis

Data collected, entered and analyzed using (SPSS version 20.0) **(**Statistical Package for the Social Sciences) software for analysis. According to the type of data qualitative represent as number and percentage , quantitative continues group represent by mean ± 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 t test, independent predictors by logistic regression. P value was set at <0.05 for significant results & <0.001 for high significant result.

RESULTS

Table (1): demographic data distribution between studied groups

			CSF Group	Control Group	t/X^2	P
Age			54.9±6.2	55.33±6.36	-0.267	0.790
Sex	**Female**	**N**	13	18		
		%	43.3%	60.0%		
	Male	**N**	17	12	1.66	0.19
		%	56.7%	40.0%		
Total		**N**	30	30		
		%	100.0%	100.0%		

Table (1) showed that Age distribution was 54.9±6.2 and 55.33±6.36 respectively between CSF Group and Control Group with no statistical significant difference regarding and sex distribution between both groups.

Table (2) : Risk factors distribution between studied groups

			Group		Total	X^2	P
			CSF Group	Control Group			
Smoking	**-VE**	**N**	15	25	40		
		%	50.0%	83.3%	66.7%		
	+VE	**N**	15	5	20	7.5	0.006*
		%	50.0%	16.7%	33.3%		
HTN	**-VE**	**N**	9	10	19		
		%	30.0%	33.3%	31.7%		
	+VE	**N**	21	20	41	0.07	0.78
		%	70.0%	66.7%	68.3%		
DM	**-VE**	**N**	18	20	38		
		%	60.0%	66.7%	63.3%		

	+VE	N	12	10	22	0.28	0.59
		%	40.0%	33.3%	36.7%		
Blood group	A-	N	0	2	2		
		%	0.0%	6.7%	3.3%		
	A+	N	8	9	17		
		%	26.7%	30.0%	28.3%		
	AB+	N	5	2	7	5.16	0.39
		%	16.7%	6.7%	11.7%		
	B-	N	0	1	1		
		%	0.0%	3.3%	1.7%		
	B+	N	8	10	18		
		%	26.7%	33.3%	30.0%		
	O+	N	9	6	15		
		%	30.0%	20.0%	25.0%		
Total		N	30	30	60		
		%	100.0%	100.0%	100.0 %		

Table (2) showed that there was a significant differences between both groups as it was high 50% in CSF Group compared to control group 16.7%, but as regard HTN, DM and blood group there was no significant difference between both groups.

Table (3): HB, WBC PLT, FBS, Urea and creatinine test distribution between studied groups

	CSF Group	Control Group	t	P
HB	13.12±1.4	12.68±1.54	1.154	0.253
WBC	8.63±1.52	8.75±2.74	-0.190	0.850
PLT	261.2±76.13	278.4±88.05	-0.809	0.422
FBS (mg/dL)	117.66±35.9	122.7±40.05	-0.485	0.630
Urea (mg/dL)	27.13±9.0	27.42±8.9	-0.114	0.910
Creatinine (mg/dL)	1.0±0.28	0.91±0.27	1.347	0.183

Table (3) showed that there was no significant difference between both groups regarding HB, WBC, PLT FBS, Urea and creatinine test distribution between both groups.

Table (4) : lipid profile distribution between groups

	CSF Group	Control Group	t	P
Cholesterol (mg/dL)	190.74±41.08	180.33±43.31	0.955	0.343
TG (mg/dL)	162.76±47.65	137.93±42.47	1.785	0.080
LDL (mg/dL)	100.87±33.15	100.63±33.73	0.027	0.979
HDL (mg/dL)	47.68±11.29	51.63±17.34	-1.046	0.300

Table (4) showed that there was no significant difference between both groups regarding Cholesterol, TG, LDL and HDL distribution between both groups.

Table (5): ECG distribution between both groups

	CSF Group	Control Group	t	P
P Max (m.s)	121.44±17.26	90.9±28.92	4.966	0.00**
P Min (m.s)	47.08±7.38	53.82±16.85	-1.693	0.096
P_D (m.s)	74.35±15.29	40.19±13.21	7.399	0.00**
Q_T_D (m.s)	154.6±47.25	63.69±20.58	8.755	0.00**

Table (5) showed that P Max, P_D and Q_T_D were significantly higher among CSF group compared to control group.

Table (6): ECHO data distribution between groups

	CSF Group	Control Group	t	P
LVDD (mm)	49.5±4.19	48.96±10.93	0.250	0.804
LVSD (mm)	32.03±3.95	34.5±8.67	-1.417	0.162
EF (%)	60.06±8.89	60.63±11.44	-0.214	0.831

Table (6) showed that There was no significant difference between both groups regarding ECHO parameters.

Table (7): TIMI Frame count data distribution between groups

	CSF Group	Control Group	t	P
LAD (30 folds/sec)	48.73±8.14	21.46±1.83	17.897	0.00**

LCX (30 folds/sec)	**44.5±7.52**	**17.96±1.6**	**18.890**	**0.00****
RCA (30folds/sec)	**43.1±8.79**	**18.5±1.85**	**14.986**	**0.00****

Table (7) showed that TIMI Frame count (LAD , LCX and RCA) showed significant difference being higher in CSF group compared with control group.

Table (8) : correlation between PD and angiographic finding

		P_D
LAD	**r**	**.595****
	P	**.000**
LCX	**r**	**.632****
	P	**.000**
RCA	**r**	**.544****
	P	**.000**

Table (8) showed that There was a significant positive correlation between PD and LAD TIMI frame count (r= 0.595, P = 0.00), LCX TIMI frame count (r= 0.632, P = 0.00) and RCA TIMI frame count (r= 0.544, P = 0.00).

Table (9): correlation between QTD and angiographic finding

		Q_T_D
LAD	**r**	**.676****
	P	**.000**
LCX	**r**	**.692****
	P	**.000**
RCA	**r**	**.605****
	P	**.000**

Table (9) showed that there was a significant positive correlation between QTD and LAD **TIMI frame count (r= 0.676, P = 0.00)**, LCX **TIMI frame count (r= 0.692, P = 0.00)** and RCA **TIMI frame count (r= 0.605, P = 0.00)**

DISCUSSION

P-wave defined as the difference between the Maximum P-wave duration and the Minimum P-wave duration. P-wave dispersion is related to non-homogenous and independent disruption of sinus impulse, intra and inter atrially and has been shown to distinguish patients at risk of developing paroxysmal AF [12].

QT-interval dispersion is defined as the difference between the Max. QT duration and the Mini QT duration, increased QT dispersion has been reported in patients with myocardial ischemia. It is indicated non uniform ventricular repolarization and may result

in increased vulnerability to malignant ventricular arrhythmia. This situation leads to the need of investigation for new risk stratification parameters of CSF [13].

In this study, we aimed to investigate the relation between QT interval dispersions and P wave dispersions in patient with coronary slow flow

This case control study included 60 patients undergoing coronary angiography (CAG) at our hospital. The study populations was divided into 2 groups: Control Group included 30 patients with normal coronary flow and CSF Group included 30 patients with coronary slow flow.

The current study showed that the age distribution was 54.9±6.2 and 55.33±6.36 respectively between CSF Group and Control Group with no statistical significant difference, also regarding sex distribution there in no statistical significant difference between both groups, which in agreement with the study of **Eshraghi et al. [2]** who found that the mean ± SD of age in normal and CSF groups were 53.78 ± 9.72 and 51.62 ± 7.35, respectively (P = 0.252) with no significant difference between studied groups regarding age and sex. Also, **Kuyumcu et al., [13]** concluded that there was no difference between groups in terms of age (P = 0.566), gender (P = 0.853) between studied groups. **Dogan et al., [14],** found also that there were no statistically significant differences between the 2 groups with respect to age; sex. Also, Also, **Yılmaz et al., [3]** showed that there was no significant differences in terms of age; sex; between CSF groups and control groups, also **Tenekecioglu et al. [15],** concluded a similar results.

The current study showed that the smoking showed statistical significant differences between both groups as high 50% in CSF Group compared to control group 16.7% (P = 0.006), which in agreement with the study of **Kuyumcu et al., [13]** who reported that the number of smokers were higher in the CSF group than the normal coronary flow group (P = .044) with statistical significant difference between both groups. The same results was obtained in the study of **Li et al., [16]** who concluded that there was a significant difference between both groups (P= 0.033), and our results were not in agreement with the study of **Mahmoud, [17]** who found no significant difference between CSF and control groups (P > 0.05) this might be because the large number of male patients in the current study compared to their study.

The current study showed that there was no significant difference between both CSF and control group (P = 0.78, P = 0.59 respectively) regarding HTN and DM, which in agreement with the study of **Eshraghi et al., [2]** who found that having DM or hypertension were not significantly different between both groups (P = 0.640, P = 0.777 respectively). Also, **Kuyumcu et al., [13]** concluded that there was no difference between groups in terms of diabetes mellitus (P = 0.803), hypertension (P = 0.845), **Dogan et al., [14],** found also that there were no statistically significant differences between the 2 groups with respect to diabetes mellitus and hypertension. Also, **Yılmaz et al., [3]** showed

that there was no significant differences in terms of hypertension; diabetes mellitus; between CSF groups and control groups. Also, **Tenekecioglu et al., [15],** concluded a similar results.

The current study showed that there was no significant difference between both groups regard blood group distribution. In contrast to our study **Doğanay et al. [18]** found blood group A was more common in CSF group than subjects with control group (53.5% vs 41.2%, P = 0.039), this might be due to large number of patients included in their study (250 patients).

The current study showed that there was no significant difference between both CSF and control groups regarding HB (P = 0.253), WBC (P = 0.850), and PLT distribution (P = 0.400), this is in agreement with the study of **Kuyumcu et al., [13]** reported that there was no significant difference between both groups regarding HB (P = 0.255), WBC (P = 0.269) and PLT distribution (P = 0.671).

The current study showed that in CSF vs control group the level of fasting glucose was (104 ± 22.7 and 103 ± 24, P = 0.83), urea was (36.7 ± 13.3 and 33.6 ± 9.5, P = 0.24) and creatinine was (1 ± 0.21 0.71 ± 0.15, P = 0.10) respectively with no significant difference between both groups. Which is in agreement the study of **Yılmaz et al., [3]** who showed that in CSF vs control group the level of fasting glucose was (104 ± 22.7 and 103 ± 24, P = 0.83), urea was (36.7 ± 13.3 and 33.6 ± 9.5, P = 0.24) and creatinine (1 ± 0.21 0.71 ± 0.15, P = 0.10) respectively with no significant difference between CSF and control groups. Also, **Tenekecioglu et al., [15],** concluded that the level of fasting glucose in CSF vs control group was (102 ± 18.6 and 103 ± 22.01, P> 0.05) and creatinine (0.8 ± 0.19 and 0.7 ± 0.14, P> 0.05) with no significant difference between CSF and control groups.

The current study showed that there was no significant difference between both CSF vs control group regarding Cholesterol (190.74±41.08 and 180.33±43.31 P = 0.343), **TG** (162.76±47.65 and 137.93±42.47 P = 0.080), LDL (c) (100.87±33.15 and 100.63±33.73, P = 0.979) and HDL (c) (47.68±11.29 and 51.63±17.34, P = 0.300) respectively distribution between both groups, our results are in agreement with the study of **Tenekecioglu et al., [15],** who concluded that the level Cholesterol (189 ±48.3 and 179±32.2 P > 0.05), Triglyceride (190±122.3 and 187±94.5, P > 0.05) and LDL (c) (108±31.8 and 91±24.1, P > 0.05) with no significant difference between CSF and control groups. But in contrast to our results they reported that there was a high statistical difference regarding HDL (c) (47.68±11.29 and 51.63±17.34, P = 0.001) being higher in CSF group than control Group, this may be due to the large number of patients (90 patients) in their study compared to current study (60 patients).

Also **Dogan et al.,[14],** found a that there was no significant difference between both CSF versus control group regarding Cholesterol, TG, LDL and HDL which coincide with our results.

The current study showed that in CSF vs control group P Max was (121.44 ± 17.26 and 90.9 ± 28.92, p= 0.00, respectively), PD was (74.35 ± 15.291 and 40.19 ± 13.21, p= 0.00, respectively) and QTD was (154.6 ± 47.25 and 63.69 ± 20.58, p= 0.00, respectively) with a high statistical significant difference between CSF and control group which in agreement with the study of **Yılmaz et al., [3]** who showed that Pmax was (106.2 ± 10.11 and 97.7 ± 8.17, p< 0.0001, respectively), Pd (53.2 ± 5.35 and 46.07 ± 4.12, p< 0.0001, respectively), were higher in the CSFP group than in the control group with a high statistical significant difference. **Dogan et al., [14],** found also that Pmax and PD were both significantly higher in CSF group patients than those of control group with highly significant difference between both groups (P <0.001). also, study of **Mahfouz et al., [19]** coincided with our results regarding Pd (59.44 ± 13.95 and 45.68 ± 17.34, P **<0.0001**) with a high statistical significant difference between CSF and control group, but level of P max **(**124.04 ± 16.15 and 108.6± 27.18, P = **0.01)** with a statistical significant difference between both groups while in our study it was a high statistical significant difference between both groups

Our results showed in CSF vs control group that LVDD was (49.5±4.19 and 48.96±10.93 , P = 804), LVSD was (32.03±3.95 and 34.5±8.67 0.162), EF (60.06±8.89 and 60.63±11.44, P = 0.831) respectively with no significant difference between both groups regarding ECHO parameters, our study is in agreement with the study of **Tenekecioglu et al., [15],** who concluded that in CSF vs control group the value of LVDD was (46±2.4 and 46± 1.9 P > 0.05) and LVSD was (30±2.7 and 29± 4.8 P > 0.05) and EF was (60±3.5 and 60±2.4 P > 0.05) respectively, with no significant difference between both groups regarding ECHO parameters. Also, **Mahfouz et al., [19]** reported that regarding the echo data there was no statistical significant difference concerning LVEDD, LVESD, EF between CSF group compared to control group.

The current study showed that the TIMI Frame count (LAD , LCX and RCA) showed a high statistical significant difference being higher in CSF group compared with control group, which in agreement with the study of **Eshraghi et al., (2018)** who reported that there was a high statistical significant difference regarding the mean of corrected TIMI frame count (CTFC) values in coronary vessels (LAD, LCX and RCA), also the study of **Dogan et al., [14],** who found a that there was a high statistical significant difference being higher in CSF group compared with control group TIMI Frame count (LAD , LCX and RCA) and Mean TIMI frame count. Also, **Tenekecioglu et al., [15],** and **Mahmoud [17]** concluded similar results to our.

The current study showed that there was a significant positive correlation between PD and LAD (r =0.595; P: 0.00), LCX (r =0.632; P: 0.00),and RCA (r =0.544; P: 0.00), which in agreement with the study of **Dogan et al., [14]** who found a significant positive correlation between both P_{max} and PD with mean TIMI frame count in CSF group and control group (r = 0.836 and r = 0.806, respectively; P < .0001), While **Mahmoud [17],** reported that in correlation analysis, Pd was positively correlated with TIMI frame count

LAD (r =0.42; P: 0.01); TIMI frame count LCx (r = 0.40; P: 0.01); but for TIMI frame count RCA (r =0.22; P:0.18) there was no statistical significant difference, which might be because of different of patients co-morbidity and risk factors of ischemic heart diseases between our study and **Mahmoud [17]** study.

The current study showed that there was a significant positive correlation between QTD and LAD TIMI frame count (r= 0.676, P = 0.00), LCX TIMI frame count (r= 0.692, P = 0.00) and RCA TIMI frame count (r= 0.605, P = 0.00) which is in agreement with the study of **Atak et al., [20]** who concluded that QTd was shown to have a significant correlation with TIMI frame count **(**LAD, LCX and RCA), also **Eshraghi et al., [2]** who reported significant positive correlation between CTFC and P wave, QT dispersion.

Conclusion: The study showed that there was a significant positive correlation between PD, QTD with TIMI frame of CSF (LAD, LCX and RCA) patients. So, with increasing QTD and PD among patients with CSF can be considered as indications markers risk for arrhythmia and related adverse cardiac events.

Limitations: Small number of patients included in our study. We didn't assessed presence of arrhythmia in association wit PD, QTD during the study or follow up prospectively for our patients.

Recommendation: Further long term studies with a larger number of sample size are recommended to emphasize our conclusion and shed more light on the correlation between QT and P wave Dispersions and TIMI frame count to give an aid in predicting complications in CSF patients and to help in follow up and treatment monitoring.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request

Conflicting Interest: No Conflict of Interest

No financial disclosure

-Acknowledgements

Not applicable

-Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

REFERENCES

1- Wang X, Nie SP. The coronary slow flow phenomenon: characteristics, mechanisms and implications. Cardiovasc Diagn Ther 2011; 1:37-43.

2- Eshraghi A, Hoseinjani E, Jalalyazdi M, Vojdanparast M, Jafarzadeh-Esfehani R. QT interval and P wave dispersion in slow coronary flow phenomenon. ARYA atheroscler 2018; 14(5): 212-217.

3- Yılmaz M, Korkmaz H, Uku Ö, Kurtoğlu E, Bilen M, Akbulut M. P-Wave and QT Dispersions on Electrocardiography in Coronary Artery Slow Flow Phenomenon. Koşuyolu Heart J 2017 ; 20(1): 19-23.

4- Monitillo F, Leone M, Rizzo C, Passantino A, Iacoviello M. Ventricular repolarization measures for arrhythmic risk stratification. World J Cardiol 2016; 8 (1): 57.

5- World Medical Association. Ethical principles for medical research involving human subjects. Eur J Emerg Med. 2001; 8(3):221-223.

.6- **Beltrame JF, Limaye SB, Horowitz JD.** The coronary slow flow henomenon: A new coronary microvascular disorder. Cardiology 2002; 97**:** 197 – 202.

7- Seyfeli E, Duru M, Kuvandık G, Kaya H, Yalcin F. Effect of obesity on P-wave dispersion and QT dispersion in women. Int J Obes 2006 ; 30(6):957-961.

8- Russo V, Di Meo F, Rago A, Papa A, Molino A, Mosella M et al. Paroxysmal atrial fibrillation in myotonic dystrophy type 1 patients: P wave duration and dispersion analysis. Eur Rev Med Pharmacol Sci; 19(7):1241-1248.

9- **Postema PG, Wilde AA.** The measurement of the QT interval. Curr Cardiol Rev 2014;10:287-94.

10- Gibson C, Cannon C, Daley W, Dodge Jr J, Alexander B, Marble S. et al. TIMI frame count: A quantitative method of assessing coronary artery flow. Circulation 1996; 93: 879 – 888.

11- **Dodge JT, Brown BG, Bolson EL, Dodge H.** Intrathoracic spatial location of specified coronary segments on the normal human heart: application in quantitative arteriography, assessment of regional risk and contraction, and anatomic display. Circulation 1988;78:1167.

12- **Okutucu S, Aytemir K, Oto A.** P-wave dispersion: what we know till now? JRSM Cardiovasc Dis 2016; 5: 2048004016639443.

13- **Kuyumcu M, Özbay M, Özen Y, Yayla, Ç.** Evaluation of frontal plane QRS-T angle in patients with slow coronary flow. Scand Cardiovasc J. 2020; 54 (1):20-25..

14- Dogan S, Yildirim N, Gursurer M, Aydin M, Kalaycioglu E, Cam F. P-wave duration and dispersion in patients with coronary slow flow and its relationship with Thrombolysis in Myocardial Infarction frame count. J Electrocardiol. logy 2008, 41(1): 55-59.

15- Tenekecioglu E, Karaagac K, Yontar O, Agca F, Ozluk O, Tutuncu, A. et al. Evaluation of Tp-Te Interval and Tp-Te/QT ratio in patients with coronary slow flow Tp-Te/QT ratio and coronary slow flow. The Eurasian J Med 2015, 47(2): 104-108.

16- Li Y, Wang Y, Jia D, Zhang Y, Guan Z, Ma C. Assessment of risk factors and left ventricular function in patients with slow coronary flow. Heart Vessels 2016 ; 31(3): 288-297.

17- Mahmoud K. Effect of coronary slow flow on dispersion of P-wave & QT-interval and its relationship with thrombolysis in myocardial infarction frame count. Egypt Heart J 2013; 65(3): 175-180.

18- Doğanay B, Kuyumcu M, Çetin M, Balbay Y. Blood group A predicts slow coronary flow in patients undergoing elective coronary angiography. Turkish J Clin Labor 2020; 11(2): 35-42.

19- Mahfouz R, Hasanein M, Farag E, Abdullah R. Non invasive predictors of coronary slow flow. Zagazig Univ Med J 2014; 20(4): 1-11.

20- Atak R, Turhan A, Sezgin T, Yetkin O, Senen K, Ileri M et al. Effects of slow coronary artery flow on QT interval duration and dispersion. Ann Noninvasive Electrocardiol. 2003; 8 (2): 107–111.