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Relationship between frontal QRS-T angle and coronary slow flow phenomenon

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Abstract

Background/Aim: Coronary slow flow phenomenon (CSFP) is termed as slow passage of contrast dye to distal portion of the coronary arteries, and can provoke angina pectoris, serious arrhythmias, or even sudden death. Previous reports suggested that frontal QRS-T angle (fQRSTa), measured by surface ECG may associate with ventricular arrhythmias and cardiac death. In this study, we aimed to assess the relationship between fQRSTa and CSFP.

Methods: In this case-control study, we retrospectively included 76 patients with CSFP [85.5% male; mean age 58.4 (9.2) years] and 50 patients with normal coronary flow (control group) [86.6% male; mean age 56.5 (10.1) years] between July 2017 and March 2019. CSFP was identified by TIMI frame count (TFC) method. Demographic, clinical and ECG characteristics were obtained from hospital records.

Results: The groups were similar concerning co-morbid cardiac conditions. Mean QTc interval and median fQRSTa were significantly greater in CSFP group compared with the controls [416.2 (34.5) vs 401 (36.3), P=0.020 and 51° (11° to 132°) vs 27° (4° to 92°), P<0.001; respectively].

Conclusion: The findings may suggest a possible distortion in cardiac electrical micropathways and indicate an increased likelihood of arrhythmia.

Keywords: Coronary slow flow phenomenon, Frontal QRS-T angle, Arrhythmia

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Ethics Committee Approval
Kırşehir Ahi Evran University Medical Faculty Clinical Research Ethics Committee approved the study design on 14/05/2019 with the decision number 2019-09 / 101
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

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Introduction

Coronary slow flow phenomenon (CSFP) is characterized by slow transmission of contrast dye towards distal vessel portions without overt stenosis in the epicardial coronary arteries [1]. Whereas the precise mechanism with which CSFP remains incompletely understood, its incidence was reported to between 1-5.5% in different studies [2-4]. Furthermore, the theme of CSFP was reported to associate with the de ventricular tachyarrhytmias, sudden cardiac death, atrial fibrillation, angina pectoris, acute coronary syndromes [5-7].

Previous evidence concerning the effect of CSFP on electrocardiographic (ECG) indices showed that the P wave dispersion and the parameters of ventricular repolarization, such as Tpeak-to-Tend interval (Tp-e), QT dispersion (QTd), T wave, Jpoint-to-Twave interval, and Tp-e/QT and Tp-e/QTc ratios, were significantly greater in patients with CSFP than the subjects with normal coronary flow [6, 8-10]. In addition, these parameters are known to associate with the atrial and ventricular tachyarrhythmia generation.

More recently, much attention has been devoted to the spatial QRS-T angle, a parameter that implies the angle between the directions of ventricular repolarization and depolarization vectors, since wider angles have been reported to indicate the risk of ventricular arrhythmias, sudden arrhythmic death, cardiovascular death and overall death in acute coronary events, heart failure and even in general population [11-14]. Since most physicians are not acquainted with the spatial QRS-T angle due to its lack of wide availability, frontal QRS-T angle (fQRSTa) is more appealing because it is readily available from a surface ECG and has a significantly well correlation with the spatial QRS-T angle [12].

In light of the afore-mentioned premises, we intended to assess the relationship between fQRSTa with CSFP in a comparable manner with normal epicardial flow.

Materials and methods

Study patients and design

This was a retrospectively designed study. Hospital database was scanned for coronary angiographies performed between July 2017 and March 2019, and a total of 76 patients who underwent coronary angiography that revealed CSFP in at least one of the main coronary arteries in the absence of overt stenosis or myocardial bridge were included. In addition, 50 sex- and age-matched patients whose angiography depicted normal coronary vessels with no CSFP were enrolled to comprise the control group. The reason for angiography in all patients was the presence of anginal chest pain or symptoms regarded as angina equivalent with positive non-invasive stress tests (treadmill test or myocardial perfusion scintigraphy). Demographic and clinical characteristics of the patients were also obtained from the hospital database. Exclusion criteria were as follows: Coronary stenosis, moderate-to-severe valvular heart disease, history of acute coronary syndrome, hearth rhythm other than normal sinus rhythm, bundle branch block, dilated or hypertrophic cardiomyopathy, history of myocarditis, malignancy, chronic obstructive pulmonary disease, severe kidney and liver dysfunction. All participants included in the study underwent a comprehensive echocardiographic evaluation using Vivid S5 (GE Vingmed Ultrason AS, Horten, Norway). Left ventricular ejection fraction (LVEF) was calculated using the modified Simpson's rule. The body-mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Obesity was defined as BMI >30 kg/m^2.

Kırşehir Ahi Evran University Medical Faculty Clinical Research Ethics Committee approved our study design on 14/05/2019 with the decision number 2019-09 / 101, and our study follows the rules of the Declaration of Helsinki.

Coronary angiography

Using the standard Judkins technique, coronary angiography was performed in each patient via Siemens Artis Zee (Siemens Medical Solution, Erlangen, Germany) either through the transfemoral or transradial routes. Assessment of the cineangiographic views recorded at 15 frames/sec was fulfilled using Axiom (Siemens Medical Solution, Erlangen, Germany) workstation by two independent and experienced cardiologists blinded to the study.

CSFP was diagnosed according to Thrombolysis in Myocardial Infarction (TIMI) frame count (TFC) method proposed by Gibson et al [15]. In brief, the total number of the cine-frames for contrast dye to reach the distal end of each major epicardial coronary artery, namely the left anterior descending artery (LAD), the left circumflex artery (Cx), and the right coronary artery (RCA) were identified with cine frame counters [15]. The distal ends were defined as distal bifurcation of LAD, distal bifurcation of Cx or obtus marginalis, whichever was longer, and the first branch of posterolateral artery of RCA [6,8,15,16]. Since LAD follows a longer course compared with Cx and RCA, a correction was made by dividing the total frame counts calculated for LAD by 1.7 to obtain the corrected TFC (cTFC) for LAD [15]. As previously described by Gibson et al. [15], the normal values of cTFC for LAD, Cx and RCA are 21.1 (1.5), 22.2 (4.1), and 20.4 (3.0), respectively. Mean TFC was computed as the sum of the respective TFCs for LAD, LCx and RCA divided by 3. Since the standardized rate for filming for these normal values was 30 frames/sec [15], we multiplied the TFCs we counted by 2. Calculation of TFCs was fulfilled by two blinded and independent cardiologists, and any disagreement occurring between these two cardiologists was resolved by a third independent and blinded cardiologist. Patients with TFC greater than these normal values were considered to have CSFP.

Electrocardiography

The 12-lead ECG was recorded (MAC 2000, GE medical systems, Milwaukee, WI, USA) at a paper speed of 50 mm/s and 10mm / mV amplitude. All ECGs were scanned and transferred to a computer and then used with x400 magnification by software. All intervals were measured from Lead 2 [17]. The QT intervals were corrected using Bazett's formula [18]. The mean of the three consecutive beats was calculated. These intervals were measured by two cardiologists blinded to the study data. Respective intra- and interobserver coefficients of variations were 3.2% and 4.8%.

The P wave, QRS complex and T wave angles were obtained from the intrinsic reports provided by the ECG device [19-21]. To compute the fQRSTa, absolute difference was taken between the QRS and T wave angles to obtain values between 0

and 180°. If the value exceeded 180°, it was subtracted from 360°.

Statistical analysis

All analyses were conducted using the SPSS 18.0 (SPSS Inc, Chicago, USA). Categorical variables were given as percentage, while continuous variables were presented as mean ± standard deviation and median. The normality of the distribution was tested using the Kolmogorov-Smirnov test. Student's t test was used to compare variables with normal distribution and Mann-Whitney U test was used to compare variables with non-normal distribution. The relationship between fQRSTa and TFC was analyzed with bivariate linear correlation analysis. Univariate and multivariate logistic regression analysis was performed to determine the risk factors predicting the presence of CSFP. A *P*-value of <0.05 was considered statistically significant.

Results

This study enrolled a total of 126 patients, of which 76 had CSFP (CSFP group) [85.5% male; mean age: 58.4 (9.2) years] and 50 had normal flow (control group) [86.6% male; mean age: 56.5 (10.1) years]. Demographic and clinical characteristics were presented Table 1. Two groups were similar concerning age, gender, and co-morbid factors such as family history of coronary artery disease, hypertension (HT), diabetes mellitus (DM), dyslipidemia, obesity, and smoking habit (*P*>0.05). There were no significant differences regarding LVEF between the groups.

Table 1: Demographic and clinical characteristics of the study population

	CSFP group (n=76)	Control group (n=50)	*P*-value
Age, years	58.4 (9.2)	56.5 (10.1)	0.275
Gender, male, %	85.5	86.6	0.868
Cardiac risk factors, %			
Family history of CAD	27.6	28.0	0.957
Hypertension	76.3	74.0	0.768
Diabetes mellitus	28.9	18.0	0.169
Dyslipidemia	64.4	52.0	0.162
Obesity (BMI >30 kg/m2)	19.7	20.0	0.967
Smoking history	34.1	31.3	0.744
Systolic BP, mm Hg	147.1 (25.6)	142.3 (30.1)	0.341
Diastolic BP, mm Hg	87.121 (5.2)	85.1 (14.4)	0.458
Heart rate, beats per min	81.8 (13.2)	79.2 (16.3)	0.336
LVEF, %	62.5 (4.9)	63.0 (3.8)	0.542
LA diameter, mm	37.6 (3.4)	36.9 (3.7)	0.277
LVEDD, mm	46.3 (3.3)	45.8 (2.4)	0.358
LVESD, mm	28.5 (3.1)	27.7 (2.9)	0.148
IVST, mm	10.5 (1.1)	10.7 (1.2)	0.337
PWT, mm	8.9 (1.1)	8.8 (1.0)	0.605
LVMI, g/m^2	81.4 (13.0)	80.5 (12.7)	0.701

Data are given as number (percentage) for categorical variables and mean (standard deviation) for continuous variables, BMI: body-mass index, BP: blood pressure, CAD: coronary artery disease, IVST: interventricular septal thickness, LA: left atrium, LVEF: left ventricular ejection fraction, LVEDD: left ventricle end-diastolic pressure, LVESD: left ventricle end-systolic pressure, LVMI: left ventricle mass-index, PWT: posterior left ventricle wall thickness

Angiographic data of the groups were given in Table 2. In the CSFP group, respective distributions based on the number of the vessels involved were as follows: 1-vessel involvement, 38.1%; 2-vessel involvement, 30.3%; and, 3-vessel involvement, 30.2%. Most of the patients in CSFP group had single vessel involvement. More specifically, LAD was the predominantly involved vessel (68.2%) in CSFP group, followed by RCA (64.2%) and LCx (56.9%). Compared with the controls, the patients in CSFP group possessed significantly higher cTFC for LAD [44.7 (11.3) vs 20.5 (4.4), *P*<0.001], higher TFC for Cx [42.1 (9.4) vs 19.3 (4.9), *P*<0.001], and higher TFC for RCA [40.8 (8.5) vs 20.2 (4.7), *P*<0.001]. Moreover, mean TFC was significantly higher in the CSFP group compared with the control group [43.93 (9.56) vs 20.0 (4.1), *P*<0.001].

ECG variables of the groups were also provided in Table 2. Both groups were similar with regards to mean heart rate, PR interval, QRS interval, and QRS complex axis (p>0.05 for all). On the other hand, mean QTc interval was longer, albeit with weak statistical significance, in the CSFP group compared with that of controls [416.2 (34.5) vs 401 (36.3), *P*=0.020]. Although median T-wave axis seemed greater than that of control group, this difference did not reach the level of statistical significance [47° (-73 to 135) vs 41° (-65 to 121), *P*=0.128]. However, CSFP group displayed a significant increase in median fQRSTa compared with the control group [51° (11 to 132) vs 27° (4 to 92), *P*<0.001].

Table 2: Comparison of the baseline electrocardiographic and angiographic characteristics of the study population

	CSFP group (n=76)	Control group (n=50)	*P*-value
Heart rate (beats/min)	71.4 (10.2)	69.5 (12.3)	0.346
PR interval (msec)	141.0 (23.2)	138.4 (19.8)	0.520
QRS interval (msec)	96.71 (1.2)	94.4 (10.1)	0.248
QTc interval (msec)	416.2 (34.5)	401(36.3)	0.020
QRS axis (°)	34 (-80 to 120)	23 (-53 to 80)	0.838
T-wave axis (°)	47 (-73 to 135)	41 (-65 to 121)	0.313
QRS-T angle (°)	51 (11 to 132)	27 (4 to 92)	<0.001
Arterial involvement			
LAD, %	68.2	-	
LCx, %	56.9	-	
RCA, %	64.2	-	
Vessel involvement			
1-vessel, %	38.1	-	
2-vessel, %	30.3	-	
3-vessel, %	30.2	-	
TIMI frame counts			
Corrected LAD	44.7 (11.3)	20.5 (4.4)	<0.001
LCx	42.1 (9.4)	19.3 (4.9)	<0.001
RCA	40.8 (8.5)	20.2 (4.7)	<0.001
Mean TIMI frame count	43.93 (9.56)	20.0 (4.1)	<0.001

Data are given as number (percentage) for categorical variables and mean (standard deviation) or median (IQR) for continuous variables. CSFP: coronary slow flow phenomenon, LAD: left anterior descending artery, LCx: left circumflex artery, RCA: right coronary artery, TIMI: thrombolysis in myocardial infarction

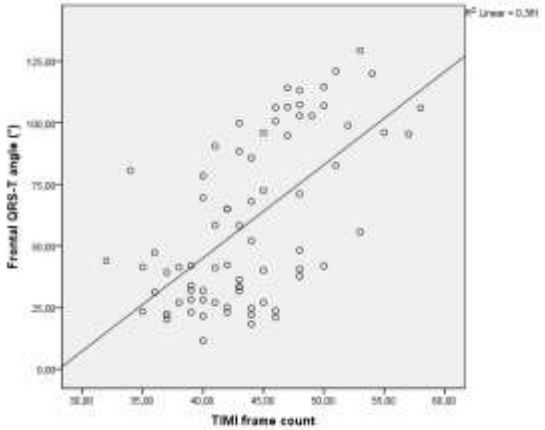
Pearson correlation analysis in SCFP group revealed significant correlation between the TIMI frame count and fQRSTa. (r=0.618, *P*<0.001) (Figure 1).

In the multivariate logistic regression model, frontal fQRSTa, together with smoking and DM, remained independently associated with CSFP (Table 3).

Table 3: Logistic regression model showing the variables associated with coronary slow flow phenomenon

	OR with 95% CI	*P*-value	OR with 95% CI	*P*-value
Age	1.02 (0.96–1.04)	0.851		
Male gender	1.45 (0.720- 3.02)	0.315		
Smoking	1.79 (1.13-2.90)	0.010	1.51 (1.10-2.21)	0.018
Heart rate	0.82 (0.79-1.03)	0.576		
Hypertension	1.08 (0.94-1.29)	0.124		
Diabetes mellitus	1.649 (1.07-2.52)	0.022	1.34 (1.01-2.01)	0.038
QRS-T angle	1.09 (1.02-1.17)	0.016	1.04 (1.01-1.06)	0.023

Figure 1: Pearson correlation analysis in SCFP group, between the TIMI frame count and fQRSTa



Discussion

The present study may contribute to the current literature with its main findings as follows: 1) Patients with CSFP were characterized with greater fQRSTa than the control subjects in the setting of angiographically-depicted normal coronary vasculature; 2) fQRSTa was significantly correlated with TFC and 3) Multivariate logistic regression analysis revealed significant and independent association of CSFP with fQRSTa, smoking and DM.

CSFP has a preponderance in smoking males [22]. The true pathophysiological mechanism underlying CSFP is yet to be completely resolved. However, a variety of hypotheses were proposed in this regard, including endothelial damage incurred by low plasma nitric oxide and high asymmetric dimethyl arginine concentrations [23,24]. Furthermore, histopathological examinations by Mosseri et al. [25] and Mangieri et al. [26] on ventricular biopsy specimens demonstrated presence of microvascular resistance against blood flow because of myointimal proliferation, fibromuscular hyperplasia, endothelial thickening and disruption in patients with CSFP. Accordingly, Pakdemir et al. [27] disclosed diffuse atherosclerosis in the coronary vasculature by means of intravascular ultrasound and fractional flow reserve.

There are a number of studies conducted with specific ECG markers for the anticipation of sudden death in patients with CSFP. Recent evidence suggested that some ECG parameters of ventricular repolarization such as Tp-e interval, QTd, and Tp-e/QT and Tp-e/QTc ratios were increased in the setting of CSFP [8-10]. Additionally, Yilmaz et al. [28] demonstrated a significant association between CSFP and QRS fragmentation. On the other hand, reports concerning the status of corrected QT interval (QTc) are conflicting. Sucu et al. [8] and Atak et al. [29] reported similar QTc interval between patients exhibiting CSFP and those with normal flow, whereas Karaman et al. [10] and Sezgin et al. [30] demonstrated a significant escalation in mean QTc in CSFP patients compared with those of normal coronary flow. However, Atak et al. [29] further reported an increase in the dispersion of QTc (QTcd) in their study despite similar maximum-QTc intervals. It has been well recognized from the previous studies that prolongation in QTc confers an increased risk of sudden cardiac death, and ventricular tachycardia and fibrillation [31]. In our study, mean QTc was greater in CSFP group compared with the controls, which is compatible with the findings of Karaman et al. and Sezgin et al. fQRSTa is a relatively novel ECG index utilized in the risk assessment of cardiac and overall deaths. It provides much more useful in risk stratification either QRS axis or T-wave axis alone [32]; however, there is no certain reference range for a normal fQRSTa owing to its variability by age and gender. In healthy persons possessing normal cardiac structure, fQRSTa is expected to be narrow. On the other hand, wider fQRSTa point out to more heterogeneity and distortion in the delicate balance between ventricular depolarization and repolarization, which translates into the presence of such cardiac fabric that is relatively more susceptible to ventricular arrhythmias [33-35]. Although the ultimate mechanism with which ventricular arrhythmias generate in the setting of CSFP remains unexplained due to scanty of relevant studies, deterioration in the aforementioned ventricular repolarization indices, namely QTcd, Tp-e, Tp-e/QT and Tp-e/QTc, were proposed as probable etiologies [10,29]. In this regard, significant widening of fQRSTa in our study may further contribute to the struggles to explain the arrhythmic mechanisms in CSFP.

Despite the definitive role of fQRSTa in the cardiac and overall mortality-risk stratification in a variety of conditions, the number of the studies regarding its potential role in diagnostic purposes is quite limited. Tanriverdi et al. [19] reported a significantly greater fQRSTa [47 (29.7°)] in non-dipper HT patients compared with those of dipping pattern [26.7 (19.6°)]. In addition, fQRSTa independently associated with the presence of non-dipping pattern. In another study, Gungor et al. [36] assessed fQRSTa in 307 patients in which angiography depicted normal coronary vasculature, and found that fQRSTa was independently associated with larger left main coronary artery caliper and presence of HT. Median fQRSTa in their study was 38°. In our study, we revealed a wider median fQRSTa 51° (11 to 132) in CSFP patients as compared with the patients with angiographically normal coronary flow [37.2 (26.5°)] in a cohort of 126 patients with apparent cardiac risk factors, and also demonstrated a significant and positive association between fQRSTa and CSFP. Not only are our findings consistent with findings of Gungor et al. but also extend their findings. Kuyumcu et al. [37] reported a significantly wider fQRSTa [69 (51°)] in SCFP patients compared with those of normal coronary artery patients [46 (36°)]. In contrast to our study, they found a significantly negative correlation (r= - 0.496) between TFC and fQRSTa. Also, the mean TFC was larger in normal coronary artery [32 (6)] patients than in SCFP patients [14 (4)]. In this study, the negative correlation between TFC and fQRSTa may be due to TFC being smaller in SCF patients than normal coronary artery patients.

Co-existence of prolonged QTc interval and wider fQRSTa in the present study is plausible, and attributable to distortion in the cardiac conduction patterns at microscopic level. Our premise is that myocardial ischemia due to widespread atherosclerosis and microscopic medial hypertrophy and fibrosis in the setting of CSFP serve as a main pathophysiological mechanism with which imbalance between ventricular depolarization and repolarization occurs.

Limitations

This study should be interpreted together with some limitations. The relatively small number of patients might have abated the statistical power of the study. Hence, our findings should be confirmed with future large-scale investigations. Secondly, prospective follow-up of the patients was not exercised so as to reveal possible arrhythmic or anginal complications in CSFP group. As a future perspective, studies with larger patient recruitment can be useful in search of association between fQRSTa and the frequency and severity of angina episodes in CSFP patients.

Conclusions

Subjects with CSF tend to be characterized with wider fQRSTa. Furthermore, fQRSTa is correlated with TFC, and independently associated with CSFP. Our findings may signify a possible distortion in the micropathways of ventricular electrical

activities, and hence an increase in the likelihood of arrhythmia generation. However, future large-scale and prospective studies may be able to establish such a relationship.

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