P-Wave and QT Dispersions on Electrocardiography in Coronary Artery Slow Flow Phenomenon

Mücahid Yılmaz[1], Hasan Korkmaz[2], Ökkeş Uku[1], Ertuğrul Kurtoğlu[1], Mehmet Nail Bilen[1], Mehmet Akbulut[2]

[1] Elazığ Training and Research Hospital, Clinic of Cardiology, Elazığ, Turkey
[2] Fırat University Faculty of Medicine, Department of Cardiology, Elazığ, Turkey

ABSTRACT

Introduction: The purpose of this study was to analyse P-wave and QT dispersions on electrocardiography in patients with CSFP and compare the findings with those of patients with NCA.

Patients and Methods: This study included a total of 82 patients (40 patients with NCA and 42 patients with CSFP). Coronary blood flow was calculated according to the thrombolysis in myocardial infarction (TIMI) frame count. Electrocardiograms were obtained at a rate of 50 mm/s and amplitude of 20 mV, including at least 3 QRS complexes for each derivation, and were taken with 12 standard deviations. The longest P-wave duration was defined as Pmax, and the shortest P-wave duration was defined as Pmin. The difference between Pmax and Pmin was defined as P-wave dispersion. QTc, which is the QT interval corrected for heart rate, was measured according to Bazett's formula. The difference between the longest QTc and shortest QTc was considered as QTc dispersion. All measurements were performed manually.

Results: This study demonstrated that P-wave dispersion (53.2 ± 5.35 and 46.07 ± 4.12, $p < 0.0001$, respectively), Pmax (106.2 ± 10.11 and 97.7 ± 8.17, $p< 0.0001$, respectively), maximum QTc (438.96 ± 16.77 and 426.13 ± 10.01, $p< 0.0001$, respectively) and QTc dispersion (68.99 ± 4.34 and 61.64 ± 4.15, $p< 0.0001$, respectively) were significantly prolonged in the CSFP group than the NCA group.

Conclusion: This study demonstrated that P-wave and QTc dispersions were prolonged in the CSFP group.

Key Words: Coronary slow flow; P-wave dispersion; QTc dispersion

Koroner Yavaş Akımda P Dalga Dispersiyon ve QTc Dispersiyonu

ÖZET

Giriş: Bu çalışmada koroner yavaş akım (KYA) tespit edilen hastalar ile normal koroner anatomi (NKA) tespit edilen hastalar arasında P dalga dispersiyonu ve QTc dispersiyonu karşılaştırıldı ve KYA ile P dalga dispersiyonu ve QTc dispersiyonu arasındaki ilişkinin belirlenmesi amaçlandı.

Hastalar ve Yöntem: Çalışmaya 40 NKA ve 42 KYA olmak üzere toplam 82 hasta alındı. Koroner kan akımı TIMI kare sayısına göre hesaplandı. Elektrokardiyografi çekimleri her derivasyon için en az 3 QRS kompleksi içerecek şekilde, 50 mm/saniye hızında, 20 mV amplitüdünde ve standart 12 derivasyonda çekildi. En uzun p dalgası Pmax ve en kısa p dalgası Pmin olarak kabul edildi. En uzun P dalgası ile en kısa p dalgası arasındaki farkı P dispersiyonu kabul edildi. QTc dispersiyonu ölçümleri için öncelikle kalp hızına göre Bazzet formülü ($QT/\sqrt{R\text{-}R}$) ile düzeltilmiş QT aralığı hesaplandı. En uzun QTc aralığı (QTcmax) ile en kısa QTc (QTcmin) aralığı arasındaki fark hesaplanarak ölçüldü. Bütün ölçümler manuel olarak yapıldı.

Bulgular: Bu çalışma KYA grubunda P dispersiyonu (sırası ile 53.2 ± 5.35 ve 46.07 ± 4.12, $p< 0.0001$), Pmax (sırası ile 106.2 ± 10.11 ve 97.7 ± 8.17, $p< 0.0001$), maksimum QTc (sırası ile 438.96 ± 16.77 ve 426.13 ± 10.01, $p< 0.0001$) ve QTc dispersiyonu (sırası ile 68.99 ± 4.34 ve 61.64 ± 4.15, $p< 0.0001$) sürelerinin NKA grubuna göre istatistiksel olarak anlamlı derecede uzun olduğunu göstermektedir.

Sonuç: Bu çalışma koroner yavaş akım da P dalga dispersiyonu ve QTc dispersiyonu sürelerinin arttığını göstermektedir.

Anahtar Kelimeler: Koroner yavaş akım; P dalga dispersiyonu; QTc dispersiyonu

Correspondence

Hasan Korkmaz

E-mail: hkorkmaz23@hotmail.com
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INTRODUCTION

Coronary slow flow phenomenon (CSFP) is defined as a slow progression of the contrast to distal vessels in the absence of any stenosis during angiography[1]. The increased QT dispersion (QTd) on ECG indicates non-uniform ventricular repolarisation 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[2]. It is believed that the homogeneity of the total duration of ventricular depolarisation and repolarisation prevents arrhythmias. The inhomogeneous conduction velocity of the ventricles along different areas or repolarisations is considered to be likely to result in severe ventricular arrhythmia through a re-entry mechanism, thereby leading to sudden cardiac death[3]. 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[2,4]. 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 predict which patients are most likely to develop life-threatening arrhythmias[5]. P-wave dispersion (Pd) is defined as the difference between the maximum and minimum P-wave durations. Pd is a simple electrocardiographic marker used in the evaluation of intraatrial and interatrial conduction times and inhomogeneous propagation of sinus impulses in the atrium, which is prone to fibrillation[6,7]. Prolonged Pd has been reported to be associated with stable angina pectoris and acute coronary syndrome[8].

The purpose of this study was to investigate the presence of a relationship between Pd (indicative of atrial conduction abnormalities) and QTcd [indicative of repolarisation inhomogeneity and CSF, which is considered as a variant of coronary artery disease (CAD)].

PATIENTS and METHODS

This cross-sectional study included 82 patients undergoing coronary angiography (CAG) at our hospital. The study population was divided into 2 groups: Group 1 included 40 patients with normal coronary arteries (NCA) and Group 2 included 42 patients with CSFP. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee.

Electrocardiograms were taken in 12 standard derivations and using a 3-channel synchronous Nihon Kohden electrocardiography (ECG) (Nihon Kohden, Tokyo, Japan) device at a rate of 50 mm/s and amplitude of 20 mV, including at least 3 QRS complexes for each derivation. During the acquisition of ECG, patients were instructed to breathe normally but not speak. For each derivation, P-wave durations were manually analysed using 10× magnifying glass. The onset of the P-wave was considered as the intersection of the isoelectric line with the P-wave, whereas the offset point was considered as the junction between the isoelectric line and the last point of the P-wave. The maximum P-wave duration was considered as the longest P-wave and the longest atrial conduction time. The difference between the longest and shortest P-wave durations was considered as Pd[9,10]. The QT interval was measured from the beginning point of the QRS complex to the end of the T-wave in each derivation. The end point of the QT interval was considered as the end point of the notch, if present, between the T and U waves. QTc, which is the QT interval corrected for heart rate, was calculated according to Bazett's formula ($QT/\sqrt{R\text{-}R}$). In each lead, the average of the QTc interval of 3 consecutive beats at each derivation was considered as the QTc interval of that lead. Patients with ECGs with measurable QTc intervals in at least 9 leads were included in this study. QTc dispersion was considered as the difference between the longest QTc interval (QTcmax) and shortest QTc interval (QTcmin). All measurements were performed manually using 10× magnifying glass. Each calculation was independently analysed in a single-blind fashion by 2 cardiologists, and the mean of 2 assessments was recorded. All electrocardiographic parameters were measured in milliseconds.

In all the patients, coronary angiography was performed using the Judkins technique with a Philips Integris Allura 9 C monoplane device (Philips Medical Systems, Eindhoven Netherland). Coronary arteries were displayed at the left anterior oblique and cranial, right anterior oblique and caudal and horizontal angles. Coronary angiographic scans were obtained by the injection of contrast medium, iohexol 350 mg l/mL (Amersham Health, Co. Cork, Ireland).

During the analysis of CSF, the "Thrombolysis in Myocardial Infarction (TIMI) frame count", which is defined as the number of cineframes required for the contrast to reach distal landmarks of the left anterior descending artery (LAD), was used to obtain an objective analysis of the index of coronary blood flow as a continuous quantitative variable. TIMI frame counting was performed using the Philips Integris Allura 9 C software VISUB cabinet-CDM digital imaging system. The mean of the measurements obtained from at least 3 projections was taken into consideration. The 'TIMI frame count' method developed by Gibson et al. was used for the measurement of contrast flow and the diagnosis of CSFP[11]. The first frame was defined as the frame in which the contrast medium was injected into the coronary artery ostium and the coronary artery was first visualised, whereas the last frame was considered as the one in which the contrast reached the distal end. The difference between the first and last frames was considered as the number of frames. Based on the frame count corrected for the difference in artery length, the mean reference values were 36 ± 1 for LAD, 22.2 ± 4.0 for circumflex artery (CX) and 20.4 ± 3 for right coronary artery (RCA)[11]. In this study, a value that was 2 standard deviations above the mean reference values was used: values of 38 for LAD, 30 for Cx and 26 for RCA were considered as CSFP. All angiographic analyses were performed by 2 experienced angiographists.

The exclusion criteria included patients with CAD who had atherosclerotic plaques in coronary arteries angiographically, acute coronary syndrome, heart failure, arrhythmia, valvular heart disease, chronic obstructive pulmonary disease, pulmonary hypertension, neurological disorder or renal dysfunction; pregnant women; patients receiving anti-arrhythmic therapy; patients using medications that may interfere with QTd (such as probucol, terfenadine, amiodarone, erythromycin and clarithromycin); patients with electrolyte imbalance, ventricular or atrial arrhythmias or pacemaker rhythm; patients with a QRS duration less than 120 ms on ECG; patients with a small P amplitude or patients with undetected P-wave end point.

Statistical Analysis

Data analysis was performed using SPSS 12. Continuous variables were presented as mean ± standard deviation, whereas categorical variables were presented as numbers and percentages. The Kolmogorov-Smirnov test was used to verify the normality of the distribution of the variables. In group comparisons, Student's t-test was used for normally distributed continuous variables and the Mann-Whitney U test was used for non-normally distributed continuous variables. The chi-square test was used for categorical variables. A p value of < 0.05 was considered statistically significant.

RESULTS

No significant differences in terms of age; sex; hyperlipidaemia; hypertension; diabetes mellitus; smoking habits; platelet count or levels of LDL cholesterol, triglyceride, Htc, glucose, urea, creatinine, sodium and potassium were found between the CSFP group and the NCA group (Table 1). The levels of WBC (7.75 ± 3.8 $10^3/\mu$l and 6.39 ± 1.8 $10^3/\mu$l, p = 0.045, respectively) and haemoglobin (14.1 ± 1.6 g/dL and 13.4 ± 1.47 g/dL, p= 0.038, respectively) were higher in the CSFP group than in the NCA group.

Pmax (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), QTcmax (438.96 ± 16.77 and 426.13 ± 10.01, p< 0.0001, respectively), QTcmin (370.60 ± 17.30 and 363.94 ± 9.25, p= 0.034, respectively) and QTcd (68.99 ± 4.34 and 61.64 ± 4.15, p< 0.0001, respectively) were higher in the CSFP group than in the NCA group (Table 2).

DISCUSSION

In this study, Pd, Pmax, QTcmax, QTcmin and QTcd values were significantly higher in patients with CSFP (Table 2).

CSFP is defined as an angiographic finding characterised by TIMI-2 flow in patients with angiographically normal or close to NCA in the absence of any ischaemic provocative manoeuvres such as angioplasty in at least 1 major epicardial arteries[12]. Even though the aetiology has not been fully elucidated, potential causes include endothelial dysfunction, microvascular dysfunction, preclinical atherosclerosis and small vessel disease. Exercise stress test and ECG abnormalities have been more commonly reported in patients with CSFP than in those with NCA[13]. The incidence of perfusion abnormalities has been reported to be between 28% and 75% by myocardial

Table 1. Characteristics of the groups

	Slow flow phenomenon	Normal coronary artery	p value
Sex (male/female)	21/21	19/21	0.99
Age	55.5 ± 7.28	54.35 ± 9.36	0.53
HL (n, %)	8 (19%)	5 (12.5%)	0.61
HT (n, %)	8 (19%)	6 (15%)	0.84
DM (n, %)	7(16.7%)	4 (10%)	0.57
Cigarette (n, %)	8 (19%)	6 (15%)	0.84
PLT	253.11 ± 63.3	261.5 ± 61.2	0.54
WBC	7.75 ± 3.83	6.39 ± 1.80	0.045
Htc	41.7 ± 4	40.4 ± 4	0.13
Hb	14.1 ± 1.6	13.4 ± 1.47	0.038
LDL	108.39 ± 30	112.97 ± 36	0.57
TG	174.2 ± 114	166.36 ± 77	0.73
Glucose	104 ± 22.7	103 ± 24	0.83
Na	138.6 ± 3.1	139.71 ± 2.8	0.12
K	4.3 ± 0.4	4.1 ± 0.35	0.064
Urea	36.7 ± 13.3	33.6 ± 9.5	0.24
Crea	1 ± 0.21	0.71 ± 0.15	0.10

PLT: Platelet, HL: Hyperlipidaemia, HT: Hypertension, DM: Diabetes mellitus, WBC: White Blood Cell, Htc: Haematocrit, Hb: Haemoglobin, LDL: Low-density lipoprotein, TG: Triglyceride, Na: Sodium, K: Potassium, Crea: Creatinine.

Table 2. Comparison of electrocardiographic findings in the groups

	Slow flow phenomenon	Normal coronary artery	p value
Pmax	106.2 ± 10.11	97.7 ± 8.17	< 0.0001
Pmin	53.95 ± 8.39	51.87 ± 5.92	0.20
Pd	53.2 ± 5.35	46.07 ± 4.12	< 0.0001
QTcmax	438.96 ± 16.77	426.13 ± 10.01	< 0.0001
QTcmin	370.60 ± 17.30	363.94 ± 9.25	0.034
QTcd	68.99 ± 4.34	61.64 ± 4.15	< 0.0001

Pmax: Maximum P-wave, Pmin: Minimum P-wave, Pd: P-wave dispersion, QTcmax: Maximum corrected QT, QTcmin: Minimum QT corrected QTcd, Corrected QT dispersion.

perfusion scintigraphy[14]. In a study of patients with CSFP, normal-appearing epicardial coronary arteries were assessed by FFR and IVUS[15]. The FFR values decreased despite maximal hyperaemic blood flow. IVUS revealed diffuse calcification along the epicardial coronary artery, predicting atherosclerosis. As a result of these findings, it can be concluded that in patients with CFSP, CAD was present in the small arterioles and main epicardial coronary arteries and endothelial dysfunction was responsible for the increased resistance in the setting of atherosclerosis.

Pd is associated with non-homogeneous and irregular propagation of sinus impulses. Pd, which is a non-invasive method, has been reported to be a valuable tool in predicting which patients are most likely to develop atrial fibrillation[16-18]. Several electrophysiological studies have demonstrated significantly prolonged interatrial and intraatrial conduction during sinus rhythm in patients with paroxysmal atrial fibrillation, resulting in prolonged P-wave duration[19-21]. This electrophysiological characteristic leads to increased Pd on electrocardiographic measurements. Thus, Pd can be used to identify patients at increased risk for the development of AF during sinus rhythm[22]. Repolarisation heterogeneity, which has been reported to cause atrial arrhythmia, is multifactorial, and its main reasons include the size of cardiac cavities, myocardial fibrosis, myocardial ischaemia and the autonomic nervous system[23]. Some authors have reported that prolonged Pd is associated with stable angina pectoris and acute coronary syndrome[24]. Considering that CSFP is a variant of ischaemic heart disease, Pd is likely to be increased in patients with CSFP. In this study, we observed that Pd and Pmax were higher in the CSFP group than in the NCA group.

The QT interval, a non-invasive diagnostic method in clinical practice, which is measured by ECG, is a parameter that is believed to reflect ventricular repolarisation duration, and QTc dispersion, defined as the heterogeneity of QT interval observed in ventricular repolarisation asynchronism in ECG, is now being used in clinical practice[25]. Regional heterogeneity of action potential duration in the ischaemic cardiac muscle tissue can initiate and propagate constant arrhythmias. The presence of these arrhythmias in a healthy heart can lead to haemodynamically unstable ventricular tachycardia or fibrillation, resulting in death[26]. Because sudden cardiac death a major complication of most cardiac diseases, including ischaemic heart disease, it is of vital importance to differentiate patients at high risk. Regional repolarisation heterogeneity can be detected by comparing intramyocardial ECG recordings and by recording electrical signals directly obtained from the heart surface. In light of the data obtained by measuring regional action potentials, the QTc interval on standard ECG and epicardial ECG, it has been concluded that QTc dispersion is an important predictor of cardiac arrhythmias and mortality[27]. It is well known that the QT interval on standard 12-lead ECG is sensitive to myocardial ischaemia. Several studies have reported a relationship between increased QTc dispersion and CAD[28,29]. It has been known that ischaemic attacks lead to a temporary increase in QTcd in patients diagnosed with CAD[30]. Acute or chronic myocardial ischaemia causes regional shortening of action potential duration, resulting in prolonged QTcd. Several studies have emphasised that an increased QTcd in patients with CAD is likely to reduce cut-off values in severe arrhythmias such as ventricular tachycardia and ventricular fibrillation[31]. It can be inferred from these data that QTcd can be prolonged in patients with CSFP. Likewise, it was found in this study that QTCmax, QTCmin and QTcd were significantly higher in patients with CSFP than in those with NCA without ischaemia.

CONCLUSION

In this study, Pd, which is an ECG marker indicating impaired atrial conduction, and QTc dispersion, which is a vital ECG marker of ventricular repolarisation homogeneity as well as ventricular instability, were significantly greater in patients with CSFP than in those with NCA.

LIMITATIONS

The first limitation was of this study the small number of patients enrolled in the study. The second and one of the most important limitations of this study was the lack of investigation of a correlation between TIMI frame count and P-wave and QTc dispersion in patients with CSFP.

CONFLICT of INTEREST

The authors reported no conflict of interest related to this article.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: MY, HK, ÖU, EK, MB, MA

Analysis/Interpretation: MY, HK, ÖU, EK, MB

Data Acquisition: ÖU, EK, MB

Writting: MY

Critical Revision: MY, HK, MA

Final Approval: All of authors

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