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The Association between QT Dispersion-QT Dispersion Ratio and the Severity-Extent of Coronary Artery Disease in Patients with Stable Coronary Artery Disease

Stabil Koroner Arter Hastalığında QT Dispersiyonu-QT Dispersiyon Oranının Koroner Arter Hastalığı Yaygınlık-Ciddiyeti ile İlişkisi

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Abstract / Özet

Objective: To evaluate QT dispersion in patients admitted with suspicion of coronary artery disease (CAD) and to study the association between QT parameters and the extent and severity of CAD.

Methods: Prospective study, recording electrocardiograms at 50 mm/s in patients with and without CAD. Single-blind analysis for QT parameter and angiography scoring measurements. Results are expressed as QT dispersion (QTd), corrected QT dispersion (QTcd), the QT dispersion ratio (QTdR), and vessel and Gensini scores for the evaluation of CAD extent-severity.

Results: QTd, QTcd, and QTdR were all higher in patients with CAD than without CAD (58.9±14.6 msec vs 32.9±10.8 msec, 63.2±15.5 msec vs 35.0±10.7 msec, 68.3±18.7 msec vs 37.7±12.5 msec; p<0.001, p<0.001, p<0.001, respectively). QTd, QTcd, and QTdR values had a tendency to increase significantly from the 1-vessel toward 3-vessel disease group (50.71±15.02 msec vs 60.03±13.24 msec vs 70.91±7.02 msec, 53.54±15.04 msec vs 64.41±14.06 msec vs 76.68±7,10 msec and 57.04±17.06 msec vs 69.53±16.77 msec vs 83.54±12.64 msec, respectively, in 1-, 2-, and 3-vessel disease groups). Gensini score was also significantly correlated with QTd, QTcd, and QTdR (p<0.001, p<0.001, p<0.001, respectively).

Conclusion: QT parameter increases in patients with CAD than those with normal coronary arteries may provide important information in our clinical practice. The regression analysis showed that the main factor affecting QTd, QTcd, and QTdR is the Gensini score-in other words, the extent and severity of CAD.

Key Words: Coronary artery disease, QT dispersion, QT dispersion ratio, Gensini score, vessel score

Amaç: Koroner arter hastalığı (KAH) şüphesi ile başvuranlarda QT dispersiyonunun ve QT parametreleri ile KAH yaygınlık-ciddiyetinin degerlendirilmesi.

Yöntemler: KAH olan ve olmayanlarda 50 mm/sn hızla çekilen elektorkardiyogramların okunmasına dayanan prospektif çalışma. QT parametreleri ve anjiyografi skorları için tek kör analiz yapıldı. Sonuçlar QT dispersiyonu (QTd), düzeltilmiş QT dispersiyonu (cQTd), QT dispersiyon oranı (QTdR) ve KAH yaygınlık-ciddiyet için Vessel ve Gensini skorları kullanıldı.

Bulgular: QTd, QTcd ve QTdR KAH grubunda KAH olmayanlara göre daha yüksekti (58,9±14,6 msn'e karşı 32,9±10,8 msn, 63,2±15,5 msn'e karşı 35,0±10.7 msn, 68,3±18,7 msn'e karşı 37,7±12,5 msn; sırasıyla p<0,001, p<0,001, p<0,001). QTd, QTcd, QTdR değerleri 1-damar hastalarından 3-damar hastalarına doğru gidildikce artma eğilimindeydi (50,71±15,02 msn'e karşı 60,03±13,24 msn'e karşı 70,91±7,02 msn, 53,54±15,04 msn'e karşı 64,41±14,06 msn'e karşı 76,68±7,10 msn ve 57,04±17,06 msn'e karşı 69,53±16,77 msn'e karşı 83,54±12,64 msn; 1-2-,3-damarhastalarında sırasıyla). Gensini skoru QTd, QTcd ve QTdR ile kuvvetli korele saptandı (sırasıyla p<0,001, p<0,001, p<0,001).

Sonuç: KAH saptadışımız QT parametre artışı günlük klinik pratiğimizde yardımcı olacaktır. REgresyon analizleride QT parametrelerini etkileyen en önemli etmenin Gensini skoru, yani KAH yaygınlık ve ciddiyeti olduğunu gösterdi.

Anahtar Kelimeler: Koroner arter hastalığı, QT dispersiyonu, QT dispersiyon oranı, Gensini skoru, Vessel skoru

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Introduction

Cardiovascular diseases are the leading cause of morbidity and mortality worldwide (1). The cardiovascular mortality rate is expected to increase from 28.9% to 36.3% in 2020 (1). Sudden cardiac death is responsible from nearly 50% of adult deaths due to coronary artery disease (CAD) in industrialized societies, and ventricular arrhythmias are the first recorded rhythm in 75%-80% of patients who are admitted with sudden cardiovascular collapse (2, 3).

QT interval, a non-invasive diagnostic method in clinical practice measured by electrocardiogram (ECG), is a parameter that is believed to reflect ventricular repolarization duration (4, 5). Ventricular repolarization can be directly measured from epicardial monophasic action potentials and correlated with QT interval on surface ECG (4, 5). Clinical and experimental studies suggested lengthened QT interval as a marker of ventricular arrhythmias and sudden death (6, 7). The difference between the longest and shortest QT intervals is recognized as QT dispersion (Qtd) (8). QTd is a measure of myocardial repolarization heterogeneity and a potential prognostic tool to predict ventricular arrhythmia development and associated mortality (9). QT dispersion ratio (QTdR), defined as QTd divided by cycle length, is proposed as a more beneficial marker than QTd in predicting ventricular arrhythmias (10). To date, several studies have shown increased QTd in patients with acute myocardial ischemia and in patients with induced ischemia, such as atrial pacing and balloon angioplasty; however, there is no sufficient number of studies evaluating the association between QTd and the extent and severity of CAD (10-16).

Our aim was to evaluate the association between QT parameters and the extent-severity of CAD in patients with stable CAD that would give precious data about the prognosis and would help to prevent ventricular arrhythmias and sudden cardiac death.

Methods

Patients

We included 100 consecutive patients who were admitted to our Cardiology Institute with suspected CAD. We performed routine tests and echocardiography and decided to perform coronary angiography (CAG) due to related guidelines to 87 of these patients. All patients were given the necessary information about the study. İstanbul University Cerrahpaşa Faculty of Medicine ethics committee approved our study, and written informed consent was obtained from all patients. The demographic features and echocardiography findings of all patients were recorded.

ECG

Resting 12-lead surface ECG with a speed of 50 mm/sec was taken from all patients before CAG. A 3-channel Hewlett Packard E-300 ECG device was used. All ECG measurements were performed using a magnifying glass and a ruler by a physician blinded to patient characteristics. QT interval was measured from all derivations as the time interval from the beginning of QRS to the end of the T-wave. The endpoint of the T-wave was considered as the point where the T-wave turns to the isoelectrical line. ECGs with measurable QT intervals in at least 8 derivations and at least 3 QT intervals in a derivation were included.

The longest (QT max) and shortest (QTmin) QT intervals were recorded, and corrected QT (cQT) intervals were measured by taking the QT interval and dividing it by the square root of the R-R interval to allow an assessment of the QT interval independently of heart rate. QT dispersion (QTd) was defined as the difference between cQTmax and cQTmin (QTd=QTmax - QTmin). QT dispersion ratio (QTdR) was calculated by dividing the QTd by the cycle length and expressed as a percentage. The formula is: (Qtd/R-Rmsec)x100.

Patients with nonreliable QRS and T-waves on ECG, paroxysmal atrial fibrillation, rhythm-conduction disturbances, valvular diseases, thyroid diseases, cardiomyopathy, congenital heart disease, pulmonary disease, pulmonary hypertension, acute coronary syndromes, congestive heart failure, myocardial infarction history, permanent pacemakers, serum electrolyte disturbances, and antiarrhythmic drug therapy were excluded from the study.

CAG

CAG was performed using the femoral approach with standard Judkins method. A Philips Integris H 3000 (USA) was used as the cineangiography device. All of the angiograms were recorded to compact discs in DICOM format and evaluated 'off-line' and visually later. Patients with at least 50% of stenosis in at least 1 epicardial coronary artery was considered as having CAD and formed the patient group, whereas others formed the control group.

Coronary artery scoring

CAD was evaluated according to number of diseased vessels (the vessel score) and the severity and localization of the stenosis (Gensini score) (17, 18).

1. Vessel score: 1 point is given for each epicardial coronary artery with significant stenosis (≥50% vessel lumen narrowing). Score ranges from 0-3, depending on the number of vessels involved (17).
2- Gensini score: This scoring system depends on the degree of the stenosis: 1 point for 1%-25% stenosis, 2 points for 26%-50%, 4 points for 51%-75%, 8 points for 76%-90%, 16 points for 91%-99%, and 32 points is given for 100% stenosis. Then, these points are multiplied by the coefficient, which is given for each main coronary artery and each segment, and the sum of all gives the total score (18).

Statistical analysis

All of the statistical analyses were performed using SPSS for Windows 15.0 statistical package program. Demographic features were analyzed by arithmetic averages, and standard deviations were measured (mean±SD). Categorical variables were evaluated with chi-square and student t-tests. P-value lower than 0.05 was considered statistically significant. The association between two quantitative variable was assessed by correlation test and Pearson-Bravais correlation coefficient (r value) was used. Negative "r" value referred to inverse relation and positive "r" value referred to relation in the same direction. While absolute "r" values less than 0.250 were considered an indicator of ignorable weak commitment, absolute "r" values ≥0.5 sought to mention casuality links. One-way Anova test was used for analysis of more than two variables. Effects of related variables were evaluated by linear regression test.

Results

We enrolled 100 consecutive patients with coronary artery disease suspicion and performed CAG in 87: 60 out of 87 patients had at least 50% stenosis in at least one coronary artery and formed the patient group with coronary artery disease, and 27 out of 87 patients had normal coronary arteries and formed the control group.

Clinical and demographic features of patient groups

Demographic features of the groups are given in Table 1. The mean age was 56.9±10.6 and 53.7±9.6 in the patient and control groups, respectively. Male patient percentage was significantly higher in the patient group than the control group (n=44 (73.3%) and n=12 (44.4%), p=0.009). Age, history of diabetes, history of hypertension, and smoking status were not statistically different among groups. Left ventricular ejection fraction values were significantly higher in the control group than the patient group (58.6±3.6%, 55.0±5.4%, p=0.002).

Table 1. Demographic features of the patients

	Patient group n:60	Control group n:27	p
Age	56.9±10.6	53.7±9.6	NS
Male sex	44 (73.3%)	12 (44.4%)	0.009
Diabetes mellütus	25 (41.7%)	11 (40.7%)	NS
Hypertension	35 (58.3%)	14 (51.9%)	NS
Smoking	27 (45.0%)	8 (29.6%)	NS
EF%	55.0±5.4	58.6±3.6	0.002

p<0.05: statistically significant; NS: not significant; EF: left ventricular ejection fraction

Evaluation of electrocardiographic QT parameters

The results of QT parameter calculations are given in Table 2. While heart rate and QTmin were not different among groups, QTmax, QTd, and QTcd intervals were significantly longer and QTdR was significantly higher in the patient group than the control group (p=0.017, p<0.001, p<0.001, p<0.001, respectively).

Comparison of QT parameters and demographic and clinical features of the patient group is given in Table 3. Accordingly, although QT parameters were not significantly related to age, sex, diabetes, hypertension, and ejection fraction values, smoking was related with QTd and QTcd in the patient group (r: 0.369, p=0.004 and r:0.290, p=0.024, respectively). Control group's QT parameters were not associated with the demographic and clinical features (Table 4).

The association between QT parameters and CAD extent and severity

a) The association between QT parameters and number of affected vessels (Vessel score):

22 (39%) out of 60 patients with CAD had 1-vessel disease, 26 (41%) had 2-vessel disease, and 12 (20%) had 3-vessel disease. We also evaluated the relationship between QT parameters and number of affected vessels. Qtcd and QTdR values had a tendency to increase significantly from the normal coronary artery group towards the 3-vessel disease group (Figure 1). QT parameter distribution among groups due to vessel score is given in Table 5. QTd was significantly higher in patients with 1-, 2-, and 3-vessel disease compared to the control group (p<0.001). While QTd was not significantly different between patients with 1-vessel and 2-vessel disease and 2-vessel and 3-vessel disease, it was significantly higher in patients with 3-vessel disease than 1-vessel disease (p<0.001). QTcd was significantly higher in patients with 2- and 3-vessel disease than 1-vessel disease (p=0.031, p<0.001, respectively). Furthermore, QTcd was significantly higher in patients with 3-vessel disease compared to 2-vessel disease (p=0.045). QTdR was significantly higher in patients with 1-, 2-, and 3-vessel disease compared to control group (p=0.002, p<0.001, and p<0.001, respectively) and was borderline significantly increased in patients with 2-vessel disease than 1-vessel disease (p=0.049). At the same time, QTdR was significantly increased in patients with 3-vessel disease than 1-vessel disease (p<0.001) and not significantly different between patients with 2- and 3- vessel disease.

b) The association between QT parameters and CAD severity and localization (Gensini score)

The lowest Gensini score was 20, the highest was 117, and the average score was 36.6±26 in the patient group. Gensini score was significantly correlated with QTd, QTcd, and QTdR (p<0.001, p<0.001, p<0.001, respectively).

As a result, lengthened QT parameters indicate higher vessel and Gensini scores (p<0.001) (Table 6).

Table 2. QT parameters of the groups

ECG parameter	Patient groups	n	Mean±SD	p
Heart rate(beat/min)	CAD	60	70.17±12.63	NS
	Control	27	69.41±13.40	
QT max (msec)	CAD	60	434.13±40.63	0.017
	Control	27	398.56±34.74	
QT min (msec)	CAD	60	357.23±37.89	NS
	Control	27	345.59±32.80	
QTd (msec)	CAD	60	58.90±14.62	<0.001
	Control	27	32.96±10.88	
QTcd (msec)	CAD	60	63.20±15.56	<0.001
	Control	27	35.06±10.74	
QTdR (%)	CAD	60	68.35±18.74	<0.001
	Control	27	37.70±12.54	

p<0.05: statistically significant, NS: not significant, SD: standard deviation, msec: miliseconds; ECG: electrocardiogram; CAD: coronary artery disease

Table 3. Association between QT parameters and demographic features of the patient group

	QTd		QTcd		QTdR	
	r	p	r	p	R	p
Age	0.056	NS	0.105	NS	0.149	NS
Male sex	0.121	NS	0.068	NS	0.006	NS
Diabetes	0.143	NS	0.132	NS	0.222	NS
Hypertension	0.237	NS	0.232	NS	0.208	NS
Smoking	0.369	0,004	0.290	0,024	0.178	NS
EF	-0.125	NS	-0.087	NS	-0.087	NS

p<0.05: statistically significant , NS: not significant; Pearson Correlation: r value >0.250: statistically significant; EF: left ventricular ejection fraction

Table 4. Association between the QT parameters and demographic features of the control group

	QTd		QTcd		QTdR	
	r	p	r	p	R	p
Age	-0.371	NS	-0.270	NS	-0.135	NS
Male sex	0.157	NS	0.146	NS	0.120	NS
Diabetes	-0.357	NS	-0.350	NS	-0.300	NS
Hypertension	-0.135	NS	-0.143	NS	-0.122	NS
Smoking	0.040	NS	0.090	NS	0.112	NS
EF	0.180	NS	0.091	NS	-0.002	NS

p<0.05: statistically significant; NS: not significant
Pearson Correlation: r value >0.250: statistically significant; EF: left ventricular ejection fraction

Discussion

Coronary artery disease is a major cause of morbidity and mortality all over the world. Diagnosis of silent CAD and determining the prognosis and cardiovascular risks by non-invasive tests are of great importance.

The ECG is the most commonly used diagnostic tool in cardiology practice. In a standard ECG, the time between the beginning of the Q wave and the termination of the T wave is recognized as the QT interval, which reflects the time between depolarization of

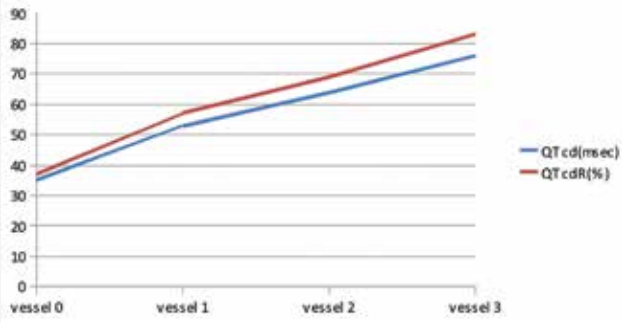

Figure 1. QT parameter distribution due to Vessel score

Table 5. QT parameter distribution among groups due to Vessel score

QT parameter	Vessel score		n	Mean±SD
QTd (msec)	Control group		27	32.96±10.88
	CAD	1	22	50.71±15.02
		2	26	60.03±13.24
		3	12	70.91±7.02
QTcd (msec)	Control group		27	35.06±10.74
	CAD	1	22	53.54±15.04
		2	26	64.41±14.06
		3	12	76.68±7.10
QTdR (%)	Control group		27	37.70±12.54
	CAD	1	22	57.04±17.06
		2	26	69.53±16.77
		3	12	83.54±12.64

SD: standard deviation; KAD: coronary artery disease; msec: miliseconds; CAD: coronary artery disease

Table 6. Correlation analysis of Vessel and Gensini score and QT parameters

		Vessel score	Gensini score
QTd	r	0.726	0.636
	p	<0.001	<0.001
QTcd	r	0.738	0.661
	p	<0.001	<0.001
QTdR	r	0.697	0.636
	p	<0.001	<0.001

p<0.05: statistically significant

myocardial cells with the incoming activation wave and electrical recovery: in other words, repolarization (8). A standard ECG derivation with the shortest QT interval reflects the region of myocardium that repolarizes the earliest, like vice the derivation with the longest QT interval reflects the region of the myocardium that repolarizes the latest. The difference between these two QT intervals is known as QTd. QTd provides the measurement of ventricular repolarization dispersion and is an indicator of electrical instability (8,19). Lengthened QT dispersion indicates the heterogeneity of ventricular repolarization, and this nonhomogeneous repolarization provides ventricular arrhythmia development (19).

QTmax was significantly prolonged in the patient group than control group (p=0.017). Several studies have also shown prolonged QTmax values in patients with CAD, and this finding is thought to be linked with atherosclerosis associated-sympathetic system activation (20, 21).

Several studies have already shown increased QTd in different patient groups, such as acute myocardial infarction, acute coronary syndromes, vasospastic angina, syndrome X, exercise-induced myocardial ischemia, heart failure, ventricular hypertrophies, and even during balloon angioplasty (10-16, 22, 23). Also, QTd increments have already been shown to be associated with malignant ventricular arrhythmias and sudden cardiac death (24). To our knowledge, there are not enough studies in the literature pointing out the association between the resting QT parameters and the severity and extent of CAD in patients with newly diagnosed stable CAD.

QTd and QTdR were significantly higher in patients with angiographically proven CAD than patients with normal coronary arteries, regardless of traditional cardiovascular risk factors in our study, and we also revealed an association between the severity and extent of CAD and QT parameters. Our results showing different resting QT parameter measurements in patients with and without CAD are concordant with other published studies; however, none of these studies analyzed the association between QT parameters and the extent and severity of CAD (10, 25). On the other hand, our findings slightly contradict the findings of another study in which the investigators could not show significant resting QT parameter differences between patients with and without CAD (26). They were able to show significant QT differences only after inducing acute myocardial ischemia by atrial pacing. This difference between studies may be due to the characteristics of the patients included in the study. In addition, the low number of patients in this study may simply not have been enough for a statistically significant difference.

The variability of regional myocardial repolarization is the main factor determining the QTd. The enhancement of QTd in patients with CAD is thought to be associated with regional ischemia that happens before other myocardial functional deterioration. Accordingly, in chronic ischemia, the existing ischemic regions and/or fibrosis seems to cause heterogeneous repolarization and myocardial irregularity, resulting in enhanced QTd and QTdR (27).

The patient group was further divided into 3 sub-groups according to the number of diseased vessels and compared in terms of QT parameters. QT parameters were significantly higher in all sub-groups than control group (p<0.001), and the main difference was observed between the 1- and 3-vessel disease subgroups (p<0.001). In our opinion, these findings suggest that the main factor affecting QT parameters is ischemia. The higher the ischemia is, the longer the QT parameters are.

The most important cause of adult sudden cardiac death (SCD) in developed countries is CAD. The first recorded rhythm is ventricular fibrillation in 75%-80% of patients who are admitted with sudden cardiovascular collapse (3). Most fatal arrhythmias seen in

patients with CAD are not related to acute coronary artery obstruction; however, ost SCD patients have serious coronary artery lesions (28). Observation of QT parameter lengthening in patients with severe CAD, together with our knowledge from other studies about the relationship between prolonged QT parameters-specifically QTd and QTdR-and malignant arrhythmias proves the prognostic importance of our findings.

Although age, sex, diabetes, hypertension, and EF values were not associated with QT parameters in the patient group, smoking was significantly associated with QTd and QTcd ($p=0.004$, $p=0.024$). The reason why we could not show an association between QT parameters and other traditional CAD risk factors may be due to the small sample size of our patient group.

Correlation analysis revealed only a reverse relationship between EF values and QT parameters within all demographic features. We observed an increase in QTd and QTdR as the extent and severity of CAD increased, probably due to the increase in ischemic areas. This finding may be considered as due to impaired global left ventricular functions rather than coronary ischemia itself; however, we did not include patients with serious left ventricular dysfunction (EF<45%).

The results of our statistical analysis revealed some degree of association between QT parameters and smoking, EF values, and Gensini score. Therefore, we further performed linear regression test in order to reveal which one of these factors mainly influenced the QT parameters. The regression analysis showed that the main factor affecting QTd, QTcd, and QTdR is the Gensini score ($p<0.001$, $p=0.002$ and $p<0.001$, respectively-in other words, the extent and severity of CAD.

The main limitations of our study were its single-centered basis and relatively small patient population size, making the power of the study limited. We did not follow up patients; however, long follow-up would reveal arrhythmia and sudden death incidence and more precious data.

Conclusion

QT dispersion and QT dispersion ratio, a non-invasive diagnostic tool in clinical practice, is not routinely used in our daily practice; however, it is associated with various CAD clinical forms. Several published studies have also emphasized using QT parameters as a non-invasive marker of CAD in patients with suspected CAD (29). We showed a significant association between QTd and QTdR and CAD extent and severity. Evaluating QTd and QTdR in stable CAD and determining the association between the extent-severity of CAD may provide important prognostic information and help to predict ventricular arrhythmia and sudden cardiac death risk. More studies performed on larger populations are needed for QT parameters to become a diagnostic marker.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of İstanbul University Cerrahpaşa Faculty of Medicine (23.06.2009/20768).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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