Original Article / *Özgün Araştırma*

A Remarkable Point: QT Dispersion in Trained Athletes

Önemli Bir Ayrıntı: Antrenmanlı Sporcularda QT Dispersiyonu

Mutlu Ekici, Özgür Tanrıverdi*, Murat Beyaz**

Siyami Ersek Thorax, Heart and Vascular Surgery Training and Research Hospital, Cardiovascular Surgery Intensive Care Unite, İstanbul, Turkey

**Şafak Health Group Göztepe Hospital, Department of Internal Medicine and Emergency Service, İstanbul, Turkey*

***Memorial Hospital, Department of Sports Medicine, İstanbul, Turkey*

Abstract

Aim: It is a common belief that QT dispersion can increase the risk of cardiac death by inducing arrhythmias and that this is a consequence of changes in the ventricular muscles due to heavy training. The aim of the present study was to compare the presence of QT dispersion among trained athletes and healthy individuals, who exercised without formal training.

Methods: A total of 53 trained athletes (Group 1, 16 females), and 32 healthy sedentary individuals (Group 2, 14 females) were included in the study. QT dispersion at rest and after stress tests using the Bruce protocol or athletes' protocol was measured for all patients.

Results: QT dispersion in subjects in Group1 was significantly higher than those in Group 2 ($p<0.05$). QT dispersion in trained males was significantly higher than in females ($r=0.106$, $p<0.05$).

Conclusion: Further studies on a larger sample size, recruiting younger sportsmen and utilizing echocardiography to determine tissue blood flow and left ventricular hypertrophy are required to assess the QT dispersion phenomenon. *(The Medical Bulletin of Haseki 2010; 48: 72-5)*

Key Words: Sports, QT dispersion, sudden death, cardiac death, arrhythmias

Özet

Amaç: QT dispersiyonu, aritmiler ve ağır egzersiz sonucu ventrikül kasında oluşan birtakım değişiklikleri içeren nedenlerle kardiyak ölüm riskini artırabilir. Bu çalışmada antrenmalı sporcular ile sedanter bireylerde QT dispersiyonu karşılaştırılmıştır.

Yöntemler: Antrenmalı 53 sporcu (Grup 1) ve 32 sedanter sağlıklı birey (Grup 2) çalışmaya alındı. Tüm hastalarda, istirahatte ve Bruce ya da sporcu protokolü ile yapılan stres testinde QT dispersiyonu belirlendi.

Bulgular: Grup 2 ile karşılaştırıldığında Grup 1 hastalarda QT dispersiyonu daha uzundu ($p<0.05$). Antrenmanlı erkeklerde kadınlara oranla daha uzun QT dispersiyonu saptandı ($p<0.05$).

Sonuç: Çalışmanın, genç sporcularda sol ventrikül hipertrofisi ve doku kan akımının ölçüldüğü ekokardiyografik incelemeler ve daha geniş hasta serileri ile yapılması önerilmiştir. *(Haseki Tıp Bülteni 2010; 48: 72-5)*

Anahtar Kelimeler: Sporcular, QT dispersiyonu, ani ölüm, kardiyak ölüm, aritmiler

Introduction

Sudden cardiac death (SCD) of trained athletes became a very impressive health problem in the last few years. Because of arrhythmias, QT dispersion (QTd) can increase the cardiac death risk (1). Likewise, it is commonly thought that extension or shortening of QT interval occurs as a result of changes in ventricular muscle, which is related with hard effort, especially the left ventricular hypertrophy (LVH) (2-5). Many studies investigated the relationship between LVH, QTd and arrhythmia and evaluated the SCD risk by comparing trained athletes and control group. Our aim is to compare QTd rates in trained athletes and healthy sedentary individuals.

Methods

A total of 53 trained athletes (Group 1, 16 females, 14 volleyball players, 12 basketball players, and 23 footballers), and 32 healthy sedentary individuals (Group 2, 14 females) were included in the study. There were no significant differences in the body mass index and lipid profiles among patients. All patients had resting and stress tests

Address for Correspondence/Yazışma Adresi: Dr. Özgür Tanrıverdi
Adnan Menderes Üniversitesi Medikal Onkoloji Bilim Dalı, Aydın, Turkey
Phone: +90 256 444 12 56/340
Fax +90 256 214 40 86 E-mail: ozgurtanriverdi@hotmail.com
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using the Bruce protocol or athletes' protocol. QT measurements were made on test ECGs at rest and post-stress. A 12-lead ECG was recorded at a speed of 50 mm/sec. QT interval was measured between the onset of the QRS complex to the end of the T wave, and in case of T and U waves, to the lowest point between the T and U waves. Subjects were included in the study if and when the QT interval could be measured on at least 7 leads. QT dispersion was defined as the difference between maximum and minimum QT intervals. Furthermore, the QT interval corrected for heart rate (QTc) was calculated with QT/RR1/2 formula. All numerical variables were expressed as median and range. Non-numerical variables were expressed as frequency and percentage. All analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 11.0 for Windows. All data were expressed as mean±SD or median range, as appropriate. A two-tailed p-value of <0.05 was considered statistically significant. While the individual numbers and gender characteristics between athletes (Group 1) and the control group (Group 2) were compared using the chi-square test, the averages of the numeric variables between two groups were compared using the student t-test. Correlation analyses were performed with the Pearson's bivariate correlation analysis.

Results

The mean ages of subjects in Group 1 and 2 were 26.4±4.8 years and 28.7±8.2 years, respectively. All groups were similar with regard to age and sex (p>0.05).

At resting ECG measurements, QT dispersion in subjects in Group1 (QTd=0.055±0.027 sec) was significantly higher than those in Group 2 (QTd=0.033±0.023 sec), (p=0.0018). Similarly, at effort-ECG measurements, QT dispersion in subjects in Group1 (QTd=0.044±0.019 sec) was significantly higher than those in Group 2 (QTd=0.021±0.019 sec), (p=0.0001). QT dispersion in trained males (QTc=0.459±0.023 sec) was significantly higher than in females (QTc=0.431±26 sec), (r=0.106, p<0.05). QT dispersion increased with the time of exercise (r=0.208, p<0.05); for football players (r=0.0187, p<0.05) (Table 1).

QT dispersion increased proportionally with age in Groups 1 (r=0.124, p<0.05). We observed that the longer effort is spent, the bigger QTds (p<0.05, r=0.208) occur. QTd values determined in football players (QTd=0.061±0.021 sec) increased significantly when compared with basketball players (QTd=0.057±0.024 sec), (p<0.05, r=0.208). In addition, QTd values found in women who play basketball (QTd=0.053±0.018 sec) increased significantly when compared with women who play volleyball (QTd=0.49±0.017 sec), (p<0.05, r=0.108) (Table 1).

Discussion

Sudden cardiac death of sportsmen occurs rarely and is mainly attributed to cardiovascular diseases (1,4). The most common cause of SCD, which is one of the leading reasons for death, is cardiac arrhythmia (4).

For sportsmen who are younger than 35 years old, the most common cardiac reason related with sudden death is hypertrophic cardiomyopathy (5,6). Other common causes, which are thought to be responsible for SCD, are right ventricular dysplasia, transmission system problems, mitral valve prolapse, idiopathic SVH, anomalies of coroner arteries, premature atherosclerosis, arrhythmogenic right ventricle, myocarditis, and Marfan syndrome (5,7). Rare reasons, especially in post mortem studies, are dilated cardiomyopathy, aortic rupture, nonatherosclerotic coroner arterial disease, aortic valve stenosis, pulmonary embolism (7). Those causes are published with different rates in different studies (1,4,5- 8). In a study by

Table 1. QT measurements of patients at Group 1 and 2. (*A two-tailed p value of <0.05 was considered statistically significant)

	Parameters (sec)	Group 1 Trained sports	Group 2 Sedentary controls	p value*
Resting	RR	0.713±0.115	0.761±0.112	0.084
	QTd	0.055±0.027	0.033±0.023	0.0018
	QTcd	0.062±0.026	0.036±0.024	0.004
	QT	0.377±0.044	0.343±0.032	0.014
	QTc	0.422±0.028	0.401±0.021	0.034
Efforts	RR	0.455±0.071	0.438±0.086	0.21
	QTd	0.044±0.019	0.021±0.019	0.0001
	QTcd	0.069±0.021	0.027±0.02	0.0001
	QT	0.313±0.027	0.288±0.029	0.02
	QTc	0.451±0.031	0.429±0.028	0.014

Burks et al., no significant difference in atherosclerosis was found between people who play sports and who does not (6). In their study, Maron et al. examined totally 158 sudden deaths of hard-effort sportsmen and they published that in 15% of the events, sudden death was because of non-cardiac reasons. In addition, the average age of 134 sudden deaths from cardiac causes was determined as 17 (between, 12-40 ages); 90% of subjects were male and 52% of them were of the white race (8). Also in the same study, it was mentioned that the most sportive conditions were basketball and football, and those 2 sports were responsible for 68% of sudden deaths (8). Sudden cardiac death was seen in 78 of those cases after workout, and in 43, during formal sporting matches (8).

Athlete's heart has its own morphological and functional differences because of regular physical exercise (9). Different kind of cardiac adaptation mechanisms, which develop as a result of regular exercise, can be designated with ECG examination and is related with different factors (10). The most common adaptations can be determined by measuring QT intervals, and QTd is the preferred parameter. Sex related parameters of a sportsman are also associated with the type and duration of physical exercise (5). QTd parameter is a measurement of non-homogeneous repolarization of myocardium and shows the increase in ventricular arrhythmia probability risk (11-14). Generally, 50ms and over is accepted as significant (15). QTd can be used as a non-invasive method in trained sportsmen to show pathological cases (12). As in a study, the decreased QTd indicated homogeneous repolarization and therefore, explained the decreased cardiac mortality of individuals who make exercises regularly (16).

The most important cardiac constitutional change in athlete's heart is known as LVH (17). We can say that there is a significant relationship between QTd and LVH. Cardiac hypertrophy, with its general meaning, is a response to stress increasing the cardiac mass (17). LVH is an important risk factor for cardiovascular complications in hypertensive patients (18). LVH increases the probability of ventricular arrhythmia and sudden death risk. Because of this reason, it has a clear influence on cardiovascular mortality (12). Patients with LVH have high QTd levels and this makes sudden cardiac risk higher (18). For mean and heavy hypertrophies, electrically transmission time shows similar changes (17). In mean and early hypertrophies, cardiac myosin contractile performance is normal or increased, while in heavy hypertrophy (related with heart failure), when contraction time of muscle increases, its amplitude decreases (17). The sudden death after exercises in a sportsman, who does not have a specific sickness, can be directly related with cardiac hypertrophy and changed repolarization (17). In a study, it was noted that patients with LVH (stenotic aortic valve, hypertension) has higher QTcd results than sportsmen and healthy control group ($p<0, 05$) (12). Myocardial hypertrophy seen in hard-effort sportsman can be related with QTd, as seen in patients with systemic hypertension. In some studies, it was possible to find a relationship between LVH and major increase in QT length or with QTd (19,20).

Another result found in our study is that as they make harder physical exercises, QTd values of basketball and football players was significantly higher than those of volleyball players (OR 1.4, $r=0.208$, $p<0.05$ and OR 1.8, $r=0.234$ and $p<0.05$, respectively).

We believe that additional studies should be conducted, including bigger patient database, more sportsmen, determination of tissue's blood flow by echocardiography and determination of left ventricular hypertrophy.

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