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Can QT dispersion predict multi-vessel coronary artery disease in patients with acute coronary syndrome?

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ABSTRACT

Objectives. This study was planned to evaluate the relation between QT dispersion (QTd) and multi-vessel coronary artery disease in acute coronary syndrome. ***Methods.*** Two hundreds and twenty-five consecutive patients with a diagnosis of non-ST segment elevation acute coronary syndrome were included. Three groups were defined as a single vessel, two vessels, and three vessels. Echocardiographic, biochemical and electrocardiographic parameters of the groups were compared. ***Results.*** QTd, corrected QT dispersion (QTcd) and corrected QT max values significantly increased in patients with the three-vessel disease compared to the patients in the single-vessel disease group (60±27 vs. 45±28 ms, 68±32 vs. 50±32 ms and 471±52 vs. 443±48 ms; p=0.002, p=0.001, and p=0.001, respectively). Also, QTd and QTcd were statistically more increased in patients with the two vessels disease than single-vessel disease group (57±29 vs. 45±28 ms; p=0.045, and 63±32 vs. 50±32 ms; p=0.046, respectively). ***Conclusion.*** In a patient with acute coronary syndrome and diffuse vessel disease, changes in the myocardial cells due to ischemia cause more of the QT dispersion in the patients with a multi-vessel disease than those with single-vessel disease.

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Keywords: Acute coronary syndrome, coronary artery disease, non-ST segment elevation, myocardial infarction, QT dispersion

Introduction

Ischemic heart disease is the leading cause of mortality worldwide [1]. In patients with multi-vessel coronary artery disease (CAD), hospitalized or not, ischemic events and other related complications may be frequently reported [2]. Therefore, multi-vessel CAD disease has to be defined, and a treatment plan should be made as soon as possible. QT dispersion (QTd) increase by ischemia is reported in various publications [3].

This study was planned to evaluate the relation between QTd and multi-vessel CAD in acute coronary syndrome (ACS).

Methods

Following the permission of the Local Ethics Committee, the patient files of the Cardiology Department of Bursa Yuksek Ihtisas Training and Research Hospital were reviewed. Between January

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2013 and January 2014, all patients administered to the hospital due to ACS were enrolled in the study. Acute coronary syndrome was defined as presentation with symptoms of ischemia in association with electrocardiographic changes and positive cardiac enzymes. Patients diagnosed with non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA) were included in this study. Clinical information included data on systemic hypertension (HT), diabetes mellitus (DM), dyslipidaemia, smoking, previous history of CAD, including coronary angioplasty or myocardial revascularization, and early family history of CAD. DM was determined by physician report and was based on a fasting blood sugar level ≥126 mg/dl or use of antidiabetic medication. Hypertension was physician-reported for systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg or use of antihypertensive agents. Hyperlipidaemia was physician reported for total cholesterol ≥200 mg/dl, low-density lipoprotein level ≥130 mg/dl, or use of cholesterol-lowering medication. Smoking included active or previous tobacco use over ten pack years.

Coronary angiographies were performed in our clinic using the standard Judkins technique [4]. Significant CAD was defined when >70% luminal diameter narrowing of a major coronary artery in any projection. Congestive heart failure, cardiogenic shock, valvular heart disease, left ventricular hypertrophy, severe HT, uncontrolled DM, serious arrhythmias with hemodynamic instability or heart failure, patients receiving class I or class III antiarrhythmic agents were excluded from the study.

Measurement of QRS Intervals from the 12-Lead ECG

Twelve-lead ECG was recorded using a Schiller AT2 PLUS at a paper speed of 50 mm/sec and a gain of 10 mm per mV with the patient lying supine. QT interval was measured manually from onset of the QRS complex to the end of the T wave, defined as the point of return of the T wave to the isoelectric line. QT interval was corrected for heart rate following Bazett's formula: QTc = QT/square root of RR. QT dispersion (QTd) was calculated as the difference between the longest (QT max) and the shortest QT (QT min) intervals recorded. Corrected QT dispersion (QTcd) was defined as the difference between the maximum and the minimum QTc for a given heart rate [5]. The study protocol was approved by the institutional review board of our Center, and informed consent was obtained from all patients.

Statistical Analysis

SPSS 16.0 statistical program (SPSS Inc., Chicago, IL, USA) was used for the statistical analysis. All values are given as mean ± standard deviation. Mean values of continuous variables were compared between the groups using the Student t test or Mann–Whitney U test, according to whether normally distributed or not, as tested by the Kolmogorov–Smirnov test. A *p* value of less than 0.05 was considered as significant.

Results

During the above described period, a total number

Table 1. Comparison of biochemical variables of groups.

	Group I (n=76)	Group II (n=58)	Group III (n=91)	*p*
Age(Years)	58±12	61±13	60±12	0.94
Gender				
Male	57	44	66	0.73
Female	19	14	25	0.8
Height (cm)	168±6	168±6.5	168±7	0.61
Weight (kg)	79±12	77±11	76±10	0.82
BMI	28±4	27±4	27±3,5	0.34
Smoking	31 (40%)	24 (41%)	32 (35%)	0.25
DM	16 (21%)	17 (29%)	26 (28%)	0.8
HT	35 (46%)	29 (50%)	51 (55%)	0.35
Total cholesterol (mg/dl)	194±52	185±43	205±53	0.23
LDL (mg/dl)	120±44	117±40	127±46	0.22
HDL (mg/dl)	41±12	40±12	44±17	0.18
Triglyceride (mg/dl)	157±224	145±106	160±148	0.16

Data are presented as means ± SD. BMI=Body mass index, DM=diabetes mellitus, HDL=High-density lipoprotein, HT=hypertension, LDL=Low-density lipoprotein

Table 2. Comparison of electrocardiographicalfeatures of groups.

	Group I (n=76)	Group II (n=58)	Group III (n=91)	*p* (I-II)	*p* (I-III)	*p* (II-III)
R-R (ms)	857±187	831±155	809±155	0.11	0.11	0.79
QTmax (ms)	405±42	411±50	421±52	0.14	0.56	0.9
QTc max (ms)	443±48	453±48	471±52	0.56	0.001	0.69
QTmin (ms)	360±35	353±40	361±45	0.19	0.10	0.74
QTcmin (ms)	393±35	390±36	403±40	0.88	0.19	0.31
QTd (ms)	45±28	57±29	60±27	0.045	0.002	0.34
QTcd (ms)	50±32	63±32	68±32	0.046	0.001	0.55

Data are presented as means ± SD. Max=maximum, Min=minimum, QTc=corrected QT, OTd=OT dispersion, OTcd=corrected OT dispersion

of 225 consecutive patients with a diagnosis of non-ST segment elevation ACS (NST-ACS) were hospitalized in our hospital. The study population was classified according to their number of lesions of coronary artery disease. Seventy-six patients with the single-vessel disease were defined as the group I, fifty-eight patients with the two-vessels disease were defined as group II and ninety-one patients with three-vessels disease were defined as group III.

The demographic, clinical, echocardiographic, biochemical, electrocardiographic and angiographic data of the three groups were compared (Table 1). There were no statistically significant differences in age, gender, weight, height, smoking, BMI, hypertension and diabetes mellitus among all three groups.

When ECG parameters are examined; comparison of group III to the group I, QTd (60±27 versus 45±28 ms; p=0.002), QTcd (68±32 versus 50±32 ms; *p*=0.001), QTc max (471±52 versus 443±48 ms; *p*=0.001) values seem significantly high. On the other hand, the comparison between group II and the group I shows us that group II's QTd (57±29 versus 45±28 ms; *p*=0.045) and QTcd (63±32 versus 50±32 ms; *p*=0.046) statistical values are obviously higher. When the three-vessels disease group compared with the two-vessels disease group, all ECG parameters didn't show a noticeable difference. And also, all three groups R-R, QT max, QT min, QTc min values did not have any difference (Table 2).

Discussion

Our study showed that QTd, QTcd, and QTc max values significantly increased in patients with the three-vessel disease compared to the patients with single-vessel disease group. In addition to this, QTd and QTcd were more increased in patients with the two-vessel disease than single-vessel disease group.

Cardiovascular disease is the most significant cause of mortality and morbidity all around the world [1]. All ischemic events develop rapidly in the myocardial tissue and cause myocardial injury in ACS patients [6, 7]. Therefore, mortality, morbidity, and prognosis were mostly determined by the number of diseased vessels in acute coronary syndrome patients. In 2007, Mallika *et al.* [6] reported a study including modification of the classic ischemic cascade concept, demonstrating in 100% of cases studied that the earliest event in ischemia is QTc interval prolongation and its increased dispersion. The early appearance of QTc interval abnormalities is precisely one of its greatest advantages for ACS diagnosis since it provides evidence of the disease when, in many cases, ST-segment abnormalities have not yet been demonstrated [3].

QT interval shows the ventricle muscle repolarization and depolarization times. The difference between QT max and QT min interval named as QT dispersion. This term is used to describe heterogeneous ventricular repolarization, and an increase in QTd has been shown to heighten the risk of serious arrhythmias and sudden cardiac death. Some studies reported that patients with the triple-vessel disease had higher QTd [8]. A study in Buenos Aires, Argentina, involving patients with typical symptoms and enzyme abnormalities compatible with ischemia but without ST changes on EKG, found prolonged QTc interval and increased dispersion, primarily in cases with adverse clinical events [3].

Most clinical and experimental studies show that primary and secondary QT intervals constitute a substantial predisposition factor for ventricular

arrhythmia [9, 10]. Elongation of the repolarization time increases the risk of arrhythmia but not always cause ventricular arrhythmias. For the ventricular arrhythmia, main trigger factor is the repolarization's non-uniform character. For the first time, the relationship between the repolarization's non-uniform character and a decrease of the ventricular fibrillation threshold was shown by Han and Moe in 1964 [11]. In electrophysiological studies, the connection between heterogeneous repolarization and arrhythmia induction were supported [12, 13].

In the setting of cardiac ischemia, evidence suggests that QT prolongation in the surface ECG represents delayed and non-uniform recovery of repolarization in areas of ischemia or infarction. The underlying mechanism is due to elevation of extracellular potassium level, acidosis, and anoxia occurring during ischemia. These conditions cause a reduction in membrane excitability, shortening of action potential duration, and prolongation of excitability recovery following an action potential [14].

The Limitations of the Study

The weak points of our study include a small number of patients, lack of classification according to scoring systems (i.e. Syntax), and detailed measurements using digital devices.

Conclusions

In our study, we found that in patients with ACS and diffuse vessel disease, changes in the myocardial cells due to ischemia (ischemic damage, delay of the electrical message, elongation of the action potential) cause more change in the QT dispersion in patients with a multi-vessel disease than those with single-vessel disease. A thorough search of the English literature available to us revealed that our study is probably the first study concerning determining the number of diseased vessels and by using QT dispersion. Higher QTd values may alert the physician to possible multi-vessel disease and patients requiring prompt invasive measures.

We conclude that further studies with larger number of patients may prove useful information concerning the value of QTd in treating ACS patients.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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