ORIGINAL ARTICLE

Effect of uncomplicated obesity on QT interval in young men

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KEY WORDS

corrected QT, prolonged QT interval, uncomplicated obesity

ABSTRACT

INTRODUCTION QT prolongation and obesity are associated with ventricular arrhythmia and sudden cardiac death. The relationship between uncomplicated obesity and QT interval prolongation is not clear.

OBJECTIVES The aim of the study was to investigate the effects of uncomplicated obesity on QT interval in young men.

PATIENTS AND METHODS A total of 122 men, including 59 obese patients and 63 controls, were recruited into the study. Patients with hypertension, diabetes mellitus, and ischemic heart disease were ineligible. Body mass index (BMI) of all patients was calculated. QT interval was measured from the precordial lead – V_5, and corrected QT (QT_c) was calculated using the Bazett's formula.

RESULTS Mean age, BMI, and waist circumference (WC) of obese patients and controls were as follows: 22.0 ±3.0 years, 36.2 ±2.2 kg/m², and 114 ±8.1 cm; 22.6 ±2.9 years, 24.7 ±2.5 kg/m², and 81.6 ±7.5 cm, respectively. There was a statistically significant difference between the obese and control groups with regard to BMI and WC (P <0.001). Furthermore, statistically significant differences were observed between the 2 groups in terms of QT_c (407.9 ±17.1 ms vs. 397.7 ±14.0 ms, respectively, P <0.001), systolic (126.9 ±8.2 mmHg vs. 114.2 ±11.1 mmHg, respectively, P <0.001) and diastolic blood pressure (78.3 ±4.5 mmHg vs. 66.9 ±10 mmHg, respectively, P <0.001). There was a positive correlation between QT_c interval and both WC (r = 0.357, P <0.001) and BMI (r = 0.424, P <0.001). There was no association between QT_c and blood pressure.

CONCLUSIONS Uncomplicated obesity in young men is associated with QT interval prolongation. Weight gain may inversely affect cardiac repolarization in uncomplicated obesity.

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INTRODUCTION Several conventional risk factors, including hypertension, hyperglycemia, and hyperlipidemia, are commonly present in obese patients.[1] Furthermore, sudden cardiac death may also occur in this patient group before the development of heart diseases.[2] It has been shown that obesity is an independent risk factor, and a higher body mass index (BMI) increases coronary mortality when evaluated together with other coronary risk factors in the Framingham study.[3] It has been demonstrated that coronary mortality increases with a higher BMI both in men and women.[4]

QT interval prolongation is associated with ventricular arrhythmia and sudden cardiac death.[5] It has also been shown to be associated with increased mortality in ischemic heart disease and diabetic nephropathy.[6,7] Obesity has been reported as the cause of QT interval prolongation.[8,9] However, studies conducted in patients with uncomplicated obesity have demonstrated no effect of weight gain on cardiac repolarization.[10]

Because female sex is associated with cardiac repolarization abnormalities, the majority of studies have been conducted in middle-aged women. To our knowledge, there have been no studies evaluating the effects of obesity on cardiac repolarization in young men. Therefore,

we investigated whether uncomplicated obesity causes QT interval prolongation in this patient group.

PATIENTS AND METHODS The study was conducted in Beytepe Military Hospital, Ankara, Turkey, between January 2005 and January 2008. It was a cross-sectional study that involved 122 men (59 with obesity and 63 healthy controls). Exclusion criteria were as follows: hypertension (≥140/90 mmHg), diabetes mellitus, elevated fasting glucose (≥100 mg/dl), ischemic heart disease, arrhythmia, complete left and right bundle branch block (BBB), thyroid, liver, and kidney disease, and use of such medications as antiarrhythmic agents, tricyclic antidepressants, antipsychotics, and antihistaminics. All participants were recruited during military draft at first medical examination. At the time of examination, patients were not involved in any regular physical activity. Informed consent was obtained from all participants and the study protocol was approved by the local ethics committee.

On admission, we performed routine clinical measurements including blood pressure, height, and weight. BMI was calculated using the formula of BMI = kg/m^2. Obesity was considered if BMI was ≥30 kg/m^2.[11] The control group comprised nonobese subjects with BMI <30 kg/m^2.

Blood samples were collected after overnight fast and were analyzed without freezing. Laboratory tests included fasting plasma glucose, renal function, liver enzymes, sodium and potassium in plasma, insulin, thyroid-stimulating hormone, and lipid profile. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula: LDL-C = (total cholesterol – high-density lipoprotein cholesterol) – (triglicerydes/5).[12] Insulin sensitivity was evaluated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) with the formula of HOMA-IR = (fasting blood insulin [μU/ml] × fasting blood glucose [mmol/l]/22.5).[13] No universal HOMA-IR cut-off value has been established for insulin resistance. High levels of HOMA-IR indicate lower sensitivity to insulin.

Twelve lead electrocardiographic (ECG) recordings were taken at a rate of 25 mm/s and 10 mm/mV amplitude. The ECGs were transferred to a personal computer by a scanner and then magnified ×400 by the Corel Paint Shop Pro X software. The onset of the Q wave was regarded as the starting point of the QT interval. The point where the T wave returned to the isoelectric TP segment was accepted as the end of the QT interval. QT interval was manually measured by calculating the average of sequential 3 QT distances from the precordial lead of V_5. Corrected QT (QT_C) was calculated using the Bazett's formula of $QT_C = QT/\sqrt{RR}$.[14] In addition, all measurements were reviewed by a cardiologist who was unaware of the clinical characteristics of the subjects.

The data were analyzed using the SPSS software. We used the U Mann-Whitney test for nonparametric unpaired variables when comparing the 2 groups, and the Student's t test for para-

TABLE Clinical and laboratory data of the study groups

	Obese group (n = 59)	Control group (n = 63)	*P*
age, yrs	22.0 ±3.0	22.6 ±2.9	>0.05
BMI, kg/m^2	36.2 ±2.2	24.7 ±2.5	<0.001
waist circumference, cm	114.0 ±8.1	81.6 ±7.5	<0.001
heart rate, beats/min	71.7 ±8.6	65.7 ±7.9	<0.001
systolic BP, mmHg	126.9 ±8.2	114.2 ±11.1	<0.001
diastolic BP, mmHg	78.3 ±4.5	66.9 ±10.0	<0.001
QT_C, ms	407.9 ±17.1	397.7 ±14.0	<0.001
FPG, mmol/l	4.9 ±0.3	4.8 ±0.2	>0.05
insulin, μU/ml	9.6 ±2.9	4.5 ±2.1	<0.001
HOMA-IR	2.14 ±0.6	0.9 ±0.4	<0.001
total cholesterol, mg/dl	178.2 ±27.8	148.1 ±20.7	<0.001
LDL-C, mg/dl	109.2 ±28.2	88.2 ±18.3	<0.001
HDL-C, mg/dl	37.4 ±6.8	45.6 ±7.8	<0.001
TG, mg/dl	152.8 ±53.1	77.6 ±39.3	<0.001
Na, mmol/l	140.1 ±1.8	140.2 ±2.0	>0.05
K, mmol/l	4.0 ±0.1	4.0 ±0.1	>0.05
Ca, mg/dl	9.1 ±0.2	9.2 ±0.3	>0.05
TSH, μU/ml	1.3 ± 0.7	1.2 ± 0.4	>0.05

Data are given as mean ± standard deviation; Student's t-test was used

Abbreviations: BMI – body mass index, BP – blood pressure, FPG – fasting plasma glucose, HOMA-IR – Homeostatic Model Assesment of Insulin Resistance, HDL-C – high-density lipoprotein cholesterol, LDL-C – low-density lipoprotein cholesterol, QT_C – corrected QT, TC – total cholesterol, TG – triglycerides, TSH – thyroid-stimulating hormone

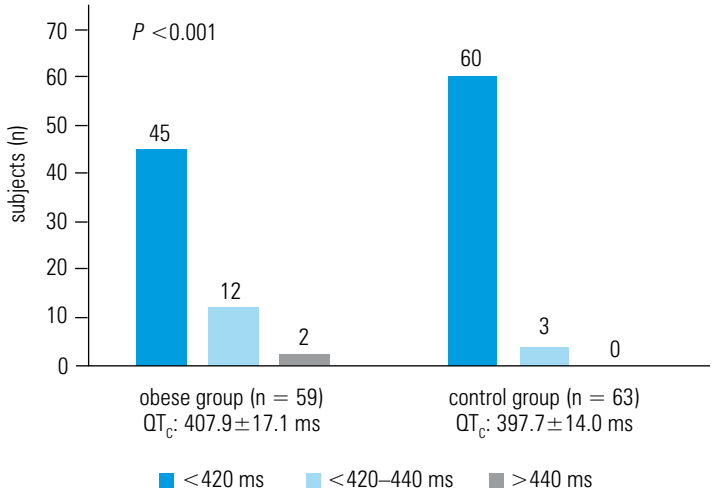

FIGURE Distribution of subjects according to corrected QT (QT_C)

metric variables. The analysis of correlations was performed using the Spearman's rank correlation test for nonparametric data and using the Pearson's correlation test for parametric data.

RESULTS Clinical characteristics of both groups are summarized in the **TABLE**. The mean age of the obese and the control groups was similar (22.0 ±3.0 years vs. 22.6 ±2.9 years, respectively). BMI and waist circumference (WC) values were higher in obese subjects compared with controls (36.2 ±2.2 kg/m^2 and 114.0 ±8.1 cm vs. 24.7 ±2.5 kg/m^2 and 81.6 ±7.5 cm, respectively). The HOMA-IR values were also significantly higher in obese patients compared with controls (2.14 ±0.6 vs. 0.9 ±0.4, respectively; P <0.001). Similarly, QT_C values were higher in obese subjects compared with controls (407.9 ±17.1 ms vs. 397.7 ±14 ms, respectively; P <0.001). We also observed increased blood pressure in obese patients compared with the control group, although hypertensive patients were excluded.

Relationships between QT_C and obesity and metabolic parameters We noted that QT_C values were well correlated with BMI, WC, and HOMA-IR (r = 0.357, P <0.001; r = 0.424, P <0.001; r = 0.413, P <0.001, respectively). However, there was no correlation between QT_C and blood pressure. We measured serum electrolytes to determine all the factors that might potentially influence the QT interval in obesity. No difference was observed between the groups in terms of plasma electrolytes. Also, the 2 groups did not differ in terms of the proportion of current smokers (49.2% in the obese group and 50.8% in the control group, P >0.05). In the obese group, 3 patients had incomplete right BBB, 2 had left anterior fascicular block (LAFB), and 1 had left posterior fascicular block (LPFB). In the control group, 1 patient had incomplete right BBB, and 3 had LAFB. There was no difference between the groups in terms of the presence of incomplete right BBB, LAFB, and LPFB.

DISCUSSION There is growing evidence that prolonged QT and/or QT_C intervals and an increase in QT dispersion predict and precede morbidity and mortality in various diseases.[5,15,16] To our knowledge, this study has been the first to demonstrate that uncomplicated obesity in young men without known cardiovascular disease is associated with QT interval prolongation. This finding suggests that obesity impairs cardiac repolarization even in young men.

There are many congenital, idiopathic, and iatrogenic reasons for prolonged QT interval. QT interval depends on the heart rate in an obvious way (the faster the heart rate, the shorter the QT interval). The standard clinical correction is to use the Bazett's formula which calculates the heart rate-corrected QT interval of QT_C with the formula of $QT_C = QT/\sqrt{RR}$.[17] Sagie et al.[18] provided a new correction formula from the linear regression model (for a reference RR interval of 1 second): $QTL_C = QT + 0.154(1\text{-}RR)$. Although the authors claimed that this equation corrects QT more reliably than the Bazett's formula, the latter is more commonly used in clinical practice.

A key mechanism underlying the present observation is most likely related to a developing cardiac repolarization disorder.[19] Long QT syndrome is present in subjects in whom QT_C is above 440 ms. The values of QT_C between 440 and 420 ms are accepted as a transient, and those below 420 ms are considered normal.[20,21] In the present study, the mean QT_C values for both groups were within the normal range. Distribution of subjects according to QT_C intervals is shown in the **FIGURE**.

The effect of age on cardiac repolarization and QT interval remains controversial. While a number of authors reported that aging prolongs QT interval,[22-24] others demonstrated that it has no effect.[25,26] QT interval in women is longer compared with men because of the effect of sex hormones on cardiac electrophysiology.[27] In order to evaluate the effect of obesity on cardiac repolarization, we only recruited men into the study. Therefore, our data is independent of sex and age. QT prolongation in obese men may also be the result of altered sex hormone metabolism due to an increase in total body fat.

Obesity causes significant abnormalities in cardiac morphology including left atrial enlargement, left ventricular geometric changes, and diastolic dysfunction.[28-30] Obesity may lead to atrial and ventricular repolarization anomalies in addition to the morphological changes discussed above.

In a previous study, Giralo et al.[10] reported no difference between patients with uncomplicated obesity and the controls in terms of QT interval and QT dispersion values. However, these authors recruited middle-aged women and men. Additionally, no data was provided regarding the levels of blood pressure in the study and control groups. The authors concluded that uncomplicated obesity does not affect cardiac repolarization parameters. In contrast to these findings, we observed that QT_C interval in young men with uncomplicated obesity is longer than in healthy subjects of the same age. The discrepancy between our study

and that of Giralo et al.[10] may result from different study populations and designs.

The present study showed that even though systolic and diastolic blood pressure were within the normal range in both groups, the blood pressure was higher in obese patients compared with controls. This difference however did not correlate with QT_c interval prolongation.

Our results are consistent with other studies in subjects with cardiovascular disease showing obesity to prolong QT interval and/or increase QT dispersion. A number of these studies demonstrated also that weight loss may normalize cardiac repolarization parameters.[8,9,31-33] We observed a stronger correlation between QT_c and WC than between QT_c and BMI. This result indicates that abdominal obesity expressed as WC is a more important predictor of cardiac risk than BMI in obese patients.[34-37] Additionally, and similarly to other studies, WC was also well positively correlated with the HOMA-IR.[36]

The main limitation of our study is that we did not measure sex hormone status in our subjects. A larger patient group and evaluation of the hormone status, including estrogen and testosterone levels, might help determine hormone factors related to obesity and QT_c.

In conclusion, our results indicate that the problem of uncomplicated obesity in young men should not be ignored. In a younger age group, weight gain and a higher BMI may inversely affect cardiac repolarization, even if there are no obesity-related disorders, such as diabetes, hypertension, and coronary artery disease. Effects of obesity on cardiac repolarization and ventricular susceptibility to arrhythmias should be further evaluated in large clinical trials.

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Wpływ niepowikłanej otyłości na długość odstępu QT u młodych mężczyzn

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otyłość niepowikłana, skorygowany odstęp QT, wydłużony odstęp QT

STRESZCZENIE

WPROWADZENIE Wydłużenie odstępu QT i otyłość związane są z występowaniem komorowych zaburzeń rytmu serca oraz nagłą śmiercią z przyczyn sercowych. Związek pomiędzy niepowikłaną otyłością a wydłużeniem odcinka QT jest niejasny.

CELE Celem badania była analiza wpływu niepowikłanej otyłości na długość odstępu QT u młodych mężczyzn.

PACJENCI I METODY W badaniu wzięło udział 122 mężczyzn, w tym 59 otyłych pacjentów i 63 stanowiących grupę kontrolną. Z badania wykluczono pacjentów z nadciśnieniem tętniczym, cukrzycą oraz chorobą niedokrwienną serca. Obliczono wskaźnik masy ciała (*body mass index* – BMI) dla wszystkich uczestników badania. Odstęp QT był mierzony z odprowadzenia przedsercowego V_5, a skorygowany odstęp QT (*corrected QT* – QT_C) obliczono za pomocą wzoru Bazetta.

WYNIKI Średni wiek, BMI i obwód pasa (*waist circumference* – WC) wynosiły odpowiednio: 22,0 ±3,0 lata, 36,2 ±2,2 kg/m² i 114 ±8,1 cm u otyłych pacjentów; 22,6 ±2,9 lat, 24,7 ±2,5 kg/m² i 81,6 ±7,5 cm w grupie kontrolnej. Zaobserwowano statystycznie istotne różnice pomiędzy grupami nie tylko pod względem BMI i WC (P <0,001), ale też QT_C (407,9 ±17,1 ms *vs* 397,7 ±14,0 ms, P <0,001), ciśnieniem skurczowym (126,9 ±8,2 mm Hg *vs* 114,2 ±11,1 mm Hg, P <0,001) i ciśnieniem rozkurczowym krwi (78,3 ±4,5 mm Hg *vs* 66,9 ±10 mm Hg, P <0,001). Stwierdzono także istnienie dodatniej korelacji między długością odstępu QT_C a WC (r = 0,357; P <0,001) i BMI (r = 0,424; P <0,001). Nie zaobserwowano korelacji pomiędzy wartościami QT_C i ciśnienia tętniczego krwi.

WNIOSKI Występowanie niepowikłanej otyłości wiąże się z wydłużeniem odstępu QT w grupie młodych mężczyzn. Wzrost masy ciała może wpływać negatywnie na repolaryzację mięśnia sercowego w niepowikłanej otyłości.

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