Journal of Electrocardiology 53 (2019) 36–39

Contents lists available at ScienceDirect

Journal of Electrocardiology

journal homepage: www.jecgonline.com

Evaluation of electrocardiographic ventricular repolarization parameters in extreme obesity

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ARTICLE INFO

Keywords:
Arrhytmia
Extreme obesity
Tp-e interval
Tp-e/JTc ratio
Tp-e/QTc ratio

ABSTRACT

Background and objectives: The risk of sudden death and cardiac arrhythmia increases in morbidly obese patients. We aimed to evaluate the marker of arrhythmias such as Tp-e/QT, Tp-e/QTc, Tp-e/JT and Tp-e/JTc ratios in extreme obesity.

Methods: The study included 41 extremely obese patients and 41 control subjects. QTmax, QTmin, QRS, JT and Tp-e intervals were measured od 12-lead electrocardiographies. In addition, Tp-e/QT, Tp-e/QTc, Tp-e/JT and Tp-e/JTc rates and QTc, cQTd and JTc intervals were calculated.

Results: Tp-e interval (79.2 ± 9.7 ms (milisecond) vs. 68.6 ± 8.1, $p < 0.001$), QTc interval (395.9 ± 18.8 vs. 377.9 ± 19.3 ms, $p < 0.001$), JTc interval (317.1 ± 27.0 vs. 297.4 ± 23.2 ms, $p = 0.001$), Tp-e/QT ratio (0.22 ± 0.03vs. 0.19 ± 0.02, $p < 0.001$), Tp-e/QTc ratio (0.20 ± 0.26vs. 0.18 ± 0.02, $p = 0.001$), Tp-e/JT ratio (0.29 ± 0.04 vs. 0.25 ± 0.03, $p < 0.001$), TPe/JTc ratio (0.25 ± 0.04 vs. 0.23 ± 0.03, $p = 0.018$), QTd (32.8 ± 10 vs.15 ± 6.4 ms, $p < 0.001$) and cQTd (70.0 ± 30.1 vs. 31.3 ± 22.4 ms, $p < 0.001$) were significantly higher in obese patients.

Conclusion: Compared to healthy subjects potential ECG repolarization predictors were significantly increased in extremely obese patients.

Introduction

Obesity is one of the most important health problems of today which is becoming an epidemic condition [1].

The definition of obesity has been made by the World Health Organization (WHO) as an accumulation of excess fat in the body to the extent that it can impair health. Obesity is defined as a BMI ≥ 30.0 kg/m^2 and classified as extreme in case of BMI ≥ 40 kg/m^2 (Table 1) [2].

Obesity is associated with various comorbidities such as diabetes, hypertension, cardiovascular diseases, metabolic syndrome and cardiopulmonary diseases. It's also associated with an increased risk of morbidity and reduced life expectancy [3].

The interval between the beginning of the QRS complex and the end of the T wave in the surface electrocardiogram (ECG) reflects ventricular depolarization and repolarization. Ventricular repolarization (VR) is a complex electrical phenomenon. Cardiac electrical changes during VR may lead to lethal arrhythmias [4].

The QT interval (QT), corrected QT interval (QTc) and QT dispersion (QTd) have been shown to predict ventricular arrhythmic events and sudden death in various clinical situations [5,6].

There are studies showing that the JT interval is a more specific repolarization marker than the QT interval. QT interval is composed of depolarization and repolarization components and it is also affected by QRS period [7]. However, JT interval is the component of the QT interval that reflects ventricular repolarization alone [8].

Tp-e interval, which is predictive of ventricular arrhythmias and sudden death even in patients with normal QTc, is a relatively new ECG parameter showing ventricular repolarization [9,10].

Tp-e/QT ratio has also recently been used as new electrocardiographic marker for ventricular repolarization distribution [11]. Both Tp-e interval and Tp-e/QT ratio have also been associated with malignant ventricular arrhythmias [12].

In this study, we aimed to evaluate potential ventricular arrhythmia predictors of surface ECG, namely Tp-e interval, Tp-e/QT, Tp-e/QTc, Tp-e/JT and Tp-e/JTc ratios in patients with extreme morbid obesity.

Material and methods

Study population

This is a cross-sectional study conducted at Bolu Abant Izzet Baysal University Hospital between October 2017 and June 2018. The study protocol was approved by the local Ethics Committee. After evaluation of 72 extremely obese patients 41 of them (31 female, 10 male, mean age 35 ± 7 years) having extreme obesity based on the WHO classification were included in the study. The patients were aged between 21 and 45 years. Patients older than 45 years were not included due to

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https://doi.org/10.1016/j.jelectrocard.2018.12.003
0022-0736/

Table 1
Classification of overweight and obesity by body mass index[2].

	Body mass index, kg/m^2
Underweight	<18.5
Normal	18.5–24.9
Overweight	25.0–29.9
Obesity, class	
I	30.0–34.9
II	35.0–39.9
III (extreme obesity)	≥40

increased probability of unknown atherosclerosis and comorbidities that may affect ECG. Physical examination, medical history of patients and blood biochemistry were evaluated in all groups to exclude systemic diseases. After matching for age and major cardiovascular risk factors like smoking, hypertension, diabetes and hyperlipidemia 41 subjects were enrolled in control group. The control group consisted of 41 volunteers (29 females, 12 males, mean age 35 ± 5 years). To minimize their effects on ECG, subjects who were known to have diabetes mellitus or hypertension longer than one year were not included neither in obesity group nor in control group.

Due to hemodynamic effects on heart and increased distance from electrodes obesity may be associated with several changes in the ECG like increased heart rate, low QRS voltage, increased QRS duration, left-axis deviation, hypertrophic findings, nonspecific flattening of the T waves, left atrial abnormalities and ST segment depression. These ECG changes may affect ventricular repolarization. Therefore we've excluded such changes. Among 31 patients excluded: ventricular conduction abnormalities were not clearly analyzed in ECGs of 3 patients, 5 patients had left bundle branch block (LBBB), 1 patient had right bundle branch block (RBBB), 3 patients had chronic lung disease, 1 patient had congestive heart failure, 6 patients had thyroid dysfunction, 1 patient had hepatocellular carcinoma, 5 patients had ST segment depression on ECG and 6 patients were taking beta blocker or nondihidopyridine calcium antagonists. Other exclusion criteria included liver dysfunction, hemolytic disorders, concomitant inflammatory diseases (infections and autoimmune diseases), known coronary or peripheral artery disease, atrial fibrillation, valvular diseases, electrolyte imbalances and use of antiarrhythmic drugs.

Electrocardiography

Twelve-lead ECGs were obtained after a 10-minute rest, with 10 mm/mV amplitude and 25 mm/s rate with standard lead positions in a supine position using commercially available machine (Nihon Kohen Cardiofax ECG-1950 VET). ECG length is 10 s, therefore depending on heart rate there were 4 to 6 beats per lead. ECGs were manually measured by the use of a magnifying Glass (TorQ 150 mm Digital Caliper LCD) by two blinded cardiologists having no information about the patients. QT intervals were taken beginning from the onset of the QRS complex to the end of the T wave, which was defined as its return to the TP baseline. If U waves were present, the QT interval was measured to the nadir of the curve between the T and U waves. The R-R interval was measured and used to compute the heart rate and to correct QT interval (QTc) with Hodges's formula [13,14]. QT dispersion (QTd) was determined as the difference between the maximum and minimum QT interval in different leads. The Tp-e interval was defined from the peak of T wave to the end of T wave. Measurements of Tp-e interval were performed from precordial leads. Rate QTc and corrected QT dispersion (cQTd) was calculated by using the Hodges's formula [$QTc = QT + 0.00175 \times (HR - 60)$]. JT intervals were measured from the end of the QRS complex (J point) to the end of the T wave (JTend interval). JTc was calculated by using the Hodges's formula [$JTc = JT + 0.00175 \times (HR - 60)$]. Tp-e/QT, Tp-e/QTc, Tp-e/JT and Tp-e/JTc ratios were also calculated. Higher rate of flat T-waves in obesity may lead to potentially longer QT measurements and/or JT due to measurement error. Therefore flat T waves were not included in our measurements. No patient had less than nine measurable leads. The intraobserver and interobserver variations for measurements were <5%.

Statistical analysis

Analyses were carried out using SPSS 15.0 Statistical Package Program for Windows (SPSS Inc., Chicago, Illinois, USA). Quantitative variables are expressed as mean ± standard deviation (SD), and qualitative variables as numbers and percentages. Differences between independent groups were assessed by Student *t*-test for normally distributed quantitative variables and Mann-Whitney's *U* test for variables without normal distribution and Chi-square test for qualitative variables. A *p* value lower than 0.05 was considered as statistically significant.

Results

Frequencies of diabetes mellitus, smoking, hypertension and hyperlipidemia were not significantly different between study patients and control group (Table 2).

Compared to control group, electrocardiographic repolarization parameters were significantly higher in obese patients (Tp-e interval: 79.2 ± 9.7 ms (vs. 68.6 ± 8.1, $p < 0.00$; QTc interval: 395.9 ± 18.8 vs. 377.9 ± 19.3 ms, $p < 0.001$; JTc interval:317.1 ± 27.0 vs. 297.4 ± 23.2 ms, $p = 0.001$; Tp-e/QT mean ratio: 0.22 ± 0.03vs. 0.19 ± 0.02, $p < 0.001$; Tp-e/QTc ratio: 0.20 ± 0.26vs. 0.18 ± 0.02, $p = 0.001$; Tp-e/JT ratio: 0.29 ± 0.04 vs. 0.25 ± 0.03, $p < 0.001$; Tp-e/JTc ratio: 0.25 ± 0.04 vs. 0.23 ± 0.03, $p = 0.018$; QT dispersion: 32.8 ± 10 vs.15 ± 6.4 ms, $p < 0.001$ and cQTd: 70.0 ± 30.1 vs. 31.3 ± 22.4 ms, $p < 0.001$) (Table 3). Intraobserver difference between cardiologists for JT, QT and Tp-e measurements were 2.5%, 3.4% and 4.3% relatively. This systematic error between cardiologists was similar for both obesity and control groups.

Discussion

In this study we have found that obesity was significantly associated with increased Tp-e interval, Tp-e/QT, Tp-e/QTc, Tp-e/JT and Tp-e/JTc ratios on surface ECG which have known to be associated with ventricular arrhythmias and sudden death. As far as we know there is no other study in the literature searching relationship between Tp-e interval and Tp-e/QT, Tp-e / QTc, Tp-e/JT and Tp-e/JTc ratios in extreme morbid obesity.

Obesity is an increasingly emerging epidemic health problem. The increased frequency of development of cardiovascular diseases (CVD), type 2 diabetes, obstructive sleep apnea (OSA) and various cancers in obese individuals may be responsible for the declined life expectancy [15,16].

It has been reported that sudden cardiac death (SCD) occurs in obese persons even without structural heart disease [17]. In the Framingham Heart Study, obesity was found to be a strong predictor of SCD [18]. The annual sudden cardiac mortality rate in obese men and women was estimated to be about 40 times higher than the rate of unexplained

Table 2
General characteristics of the study groups.

Baseline characteristics	Control group (n = 41)	Extreme obesity (n = 41)	*p* value
Age (mean ± SD) (years)	35 ± 5	35 ± 7	0.236
Male/female	12/29	10/31	0.618
Hypertension (%)	5(12%)	6 (14.6%)	0.32
Smoking	14(34%)	12(29%)	0.261
Diabetes mellitus	4(10%)	5(12%)	0.353
Hyperlipidemia	2(5%)	3(7%)	0.343
BMI	19.3 ± 1.5	45.6 ± 6.7	<0.001

BMI: body mass index, SD: standard deviation.

Table 3
Electrocardiographic findings of the study population.

	Control group (n = 41)	Extreme obesity (n = 41)	*p* value
Heart rate (bpm)	69.5 ± 12.4	81.2 ± 17.1	0.001
Tp-e ms	68.6 ± 8.1	79.2 ± 9.7	<0.001
Qtmin ms	353.8 ± 24.3	342.4 ± 26.6	0.068
Qtmax ms	368.5 ± 25.2	375.2 ± 29.8	0.274
QTc ms	377.9 ± 19.3	395.9 ± 18.8	<0.001
JT ms	280.0 ± 27.1	280.0 ± 26.8	0.905
JTc ms	297.4 ± 23.2	317.1 ± 27.0	0.001
Tp-e/QT	0.19 ± 0.02	0.22 ± 0.03	<0.001
Tp-e/QTc	0.18 ± 0.02	0.20 ± 0.26	0.001
Tp-e/JT	0.25 ± 0.03	0.29 ± 0.04	<0.001
Tp-e/JTc	0.23 ± 0.03	0.25 ± 0.04	0.018
QTd ms	15.0 ± 6.4	32.8 ± 10.0	<0.001
cQTd ms	31.3 ± 22.4	70.0 ± 30.1	<0.001
QRS ms	87.4 ± 11.2	90.4 ± 10.5	0.083

bpm: beat per minute, ms: millisecond, Tp-e: T peak and end interval, QTmin: minimum QT, QTmax: maximum QT, QTc: corrected QT interval, JT interval (JT): were measured from the end of the QRS complex (J point) to the end of the T wave (JTend interval), JTc: corrected JT interval, QT dispersion (QTd) was determined as the difference between the maximum and minimum QT interval, cQTd: corrected QT dispersion.

cardiac arrest in a matched nonobese population [19]. Ventricular tachycardia has been the most common reported arrhythmia for sudden death [17,19,20]. Potentially lethal ventricular arrhythmia may be the first clinical presentation or may be the cause of sudden death. Therefore it is important to determine potential objective predictors of those malign arrhythmias. 12-lead ECG is a simple and widely used method that can be helpful for that purpose. Accordingly, some studies have reported improvement in ventricular repolarization after weight loss in obese subjects [23].

Myocardial repolarization has been evaluated by various methods including QT, Tp-e, JT intervals and their ratios. Prolongation of QT and QTc intervals have been suggested as a risk factor for ventricular arrhythmia and death [24,25]. Obesity has been found to be associated with a high prevalence of a prolonged QT interval [21,22] which represents electrical depolarization and repolarization. In the present study we have also found that QTc duration is prolonged in obesity (395.9 ± 18.8 vs 377.9 ± 19.3 ms). Similar to our age criteria Hemendra-Suthar et al. [26] studied 50 obese subjects (age: 18–40 years, BMI > 30 kg/m^2) and 50 matched controls. There was no significant difference between mean QTc in normal weight subjects (383.72 ± 3.2 ms) and obese subjects (394.80 ± 20.71 ms). However, meta-analysis by Omran et al. [27] showed that QTc was significantly longer and QT or QTc dispersion was significantly greater in obese or overweight patients than in normal weight controls. Isik and et al. [28] reported that QTc and QTd were increased in obese or overweight patients compared to age and gender-matched normal weight controls (QTc: 434.6 ± 36.0 vs. 409.0 ± 20.4 ms, $p < 0.001$ and QTd: 67.7 ± 13.7 vs. 42.7 ± 7.2 ms, $p < 0.001$). Blood pressure was an independent predictor of QTc and QTc dispersion. In the present study, to eliminate their effects on depolarization and repolarization parameters we have excluded subjects having hypertension and diabetes longer than one year. Both QTc and QT dispersion have been correlated positively and significantly with CVD and all-cause mortality [29]. However, until now, it is unclear whether obesity-related QT interval prolongation is associated with the increased risk of cardiac arrhythmias which can lead to sudden cardiac deaths [30].

Tp-e interval is the interval between the peak and the end of T wave on ECG. The earliest part of the repolarization is the repolarization of the epicardial action potential corresponding to the peak of the T wave. The last part of the repolarization is the repolarization of the M cells corresponding to the end of the T wave. Thus, the Tp-e interval can be a marker of total dispersion of repolarization [31,32]. It has been related to ventricular arrhythmias and sudden cardiac death [9,10]. Electrophysiological studies have shown a relationship between long Tp-e interval and ventricular tachycardia (VT) induction and spontaneous VT formation [33]. Tp-e/QT ratio remains relatively stable between heart rates of 60–100 beats per minute [34] and has also been associated with malignant ventricular arrhythmias [12]. Compared to the single use of Tp-e or QT intervals the Tp-e/QT ratio is considered to be a more sensitive index of arrhythmogenesis [34].

The QT interval is largely determined by the duration of repolarization which corresponds to the JT interval. Therefore, the JT interval has been proposed as a more appropriate measure of ventricular repolarization than the QT [35]. Furthermore, the risk of incident cardiovascular events was better predicted with measurements of the JT rather than the QT interval [7,36,37]. The heart rate-corrected JT (JTc) interval has also been proposed as a more appropriate measure of ventricular repolarization than the QTc interval in individuals with the increased QRS duration [7,38].

Most studies assessing VR in obese patients have used the QT and QTc intervals and/or QT or QTc dispersion. Relatively few have employed the JT or JTc interval and its variants, the Tp-e interval or the Tp-e/QT ratio for this purpose [27]. In association with prolonged QT duration we have found that Tp-e and JTc intervals and Tp-e/QTc and Tp-e/JTc ratios are increased in morbidly obese subjects. We hope that clinical significance of this finding for the prediction of malignant arrhythmias will be evaluated in future studies.

Limitations

Manual calculation of measurements instead of computer-assisted calculations is an important limitation. Automated measurement systems have been developed for QT measurement, but problems with these systems currently exist [30]. Manual identification of T-end is also problematic, cardiologist dependent and poorly reproducible. Therefore automated methods may be preferred [39]. Weight loss in obese patients can improve or prevent most obesity-related risk factors for cardiovascular disease and also arrhythmias. Long-term ambulatory ECG monitorization methods might be valuable for documentation of association between studied surface ECG parameters with arrhythmias. Small sample size and lack of clinical follow up of the patients in our study for mortality and morbidity is an important limitation.

Conclusions

Previous studies have shown that QT prolongation is important in terms of malignant ventricular arrhythmias in morbid obese patients [21,23]. However, Tp-e, JT, JTc intervals and Tp-e/QTc and Tp-e/JTc ratios were not measured in morbid obesity. This study shows that these relatively new repolarization indices and potential electrocardiographic predictors of ventricular arrhythmias are significantly increased in extremely obese patients. Long-term follow-up and large-scale prospective studies are needed to confirm our results.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Conflicts of interest

There are no conflicts of interest.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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