

CHRONIC MYOCARDIAL INFARCTION INFLUENCE OF OBESITY ON DISPERSION PARAMETERS

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REZUMAT

Obiectiv: Aprecierea influenței obezității asupra parametrilor de dispersie a activității ventriculare. **Material și metodă:** 50 de pacienți cu infarct miocardic cronic, împărțiți în două loturi: obezi (10) și normoponderali (40) au fost investigați electrocardiografic, calculându-se indicii de variabilitate a intervalului QT corecțat în funcție de frecvența cardiacă (CFC): QTdc (dispersia QT CFC), JTdc (dispersia JT CFC), QRSdc (dispersia QRS CFC), precum și valorile medii ale intervalelor QT (QTm) și JT (Jm). **Rezultate:** Asocierea obezității a determinat o creștere semnificativă statistic doar a dispersiei QRS ($p=0,0027$), dar nu și a celorlalți indici ai variabilității QT. **Concluzii:** Se poate spune că la pacienții coronarieni obezi există o afectare a conducerii intraventriculare, motiv pentru care se impune ajustarea valorilor dispersiei QRS în funcție de indicele de masă corporală.

Cuvinte cheie: obezitate, infarct miocardic cronic, dispersia QT, dispersia QRS

ABSTRACT

Objective: Evaluation of obesity influence on ventricular activity dispersion parameters. **Material and method:** Fifty chronic myocardial infarction patients, divided in two groups: obese and normal weight patients, underwent electrocardiographic investigation to calculate the heart rate corrected (HRC) QT interval variability indices: QTdc (HRC QT dispersion), JTdc (HRCJT dispersion), QRSdc (HRC QRS dispersion), and the average values of the QT (QTm) and JT (Jm) intervals. **Results:** Obesity increases significantly only QRS dispersion ($p=0.0027$), but not the other variability QT indices. **Conclusions:** Intraventricular conduction is affected in obese coronary patients and QRSdc should be adjusted to the body mass index in coronary patients.

Key Words: obesity, chronic myocardial infarction, QT dispersion, QRS dispersion

INTRODUCTION

As weight increases, the blood volume and the work of the heart augment too, leading, in time, to left ventricular hypertrophy and affecting myocardial contraction and relaxation. Obesity is frequently associated with diabetes, increasing the cardiovascular risk, and represents a major risk factor for coronary artery disease, independent of the existence of other associated risk factors. Increase of the abdominal mass impairs the function of the diaphragm, which, together with the sleeping apnea, reduces the oxygen supply, the result being arrhythmia and even sudden death during sleep. Hypoxia can cause pulmonary hypertension and right heart failure.

The ventricular activity dispersion parameters are considered markers of arrhythmia and of risk for sudden death.¹

Considering the conclusions of other studies, that obesity increases QT dispersion, the aim of this study was to evaluate the influence of obesity on the dispersion parameters in chronic myocardial infarction (MI) patients, knowing the correlation between obesity and coronary death.² This objective can be achieved, evaluating the significance of the differences between the dispersion parameters calculated in obese MI patients, compared to those obtained for the normal weight MI patients.

MATERIAL AND METHOD

Fifty patients with a history of myocardial infarction were investigated (the clinical characteristics of the patients are presented in Table 1) by performing 12-lead ECG, at a paper speed of 25 mm/s, using a Siemens-Megacart electrocardiograph; the existence of a chronic myocardial infarction was demonstrated, considering the criteria of the Joint European Society of Cardiology and of the American College of Cardiology Committee for the Redefinition of

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Received for publication: Feb. 17, 2005. Revised: Jun. 17, 2005.

Myocardial Infarction: any QR wave ≥ 30 ms in leads V_1 - V_3 , abnormal Q wave in lead I, II, aVL, aVF or V_4 - V_6 , in any two contiguous leads, and at least 1 mm in depth.³ The following parameters were studied: QT dispersion (QTd - the difference between the maximal and minimal QT interval value in the 12 leads), QTdc (heart rate corrected QTd) and the average QT duration (QTdm). The following parameters were calculated for the JT interval: JT dispersion (JTd - the difference between maximal and minimal duration of the JT interval), JTdc (heart rate corrected JTd) and the average duration of the JT interval in the 12 leads (JTdm). In a similar way: QRSd (the difference between QRSmax and QRSmin) and QRSdc (heart rate corrected QRS dispersion), were calculated.

Table 1. Clinical characteristics of the investigated chronic myocardial infarction patients

Age	67 \pm 20 years
Sex	44 men and 6 women
MI ancientness	1 - 9 years
MI location	Anterior: 20 Inferior: 24 Anterior and inferior: 6
Cardiovascular risk factors	Smoking: 10 Type 2 diabetes mellitus: 5 (4 obese) Hypertension: 15 (8 obese) Obesity: 10
Arrhythmias (in antecedents or in the evolution, not in the moment of recording)	Premature contractions - Atrial: 4 - Juxtanothal: 1 - Ventricular: 6 Sinus tachycardia: 5 Atrial flutter: 2 Atrial fibrillation: 1
Associated disorders	Heart failure: 12 Left atrial hypertrophy: 1 Left ventricular hypertrophy: 5 Right ventricular hypertrophy: 1 Unstable angina: 3 Ventricular aneurysm: 2 Mitral regurgitation: 3 Chronic Obstructive Pulmonary Disease: 5
Medication	Angiotensin conversion enzyme inhibitors: 20 Calcium blockers: 4 Beta-blockers: 4 Class III antiarrhythmic drugs: 8 Digitalis: 4

The dispersions were calculated considering at least eight leads for each patient, excluding the leads in which the end of the T wave could not be determined exactly. The end of the T wave was defined as its return to the

isoelectric line. If the end of the T wave could not be determined, the tangential method was applied: the end of the T-wave was at the intersection of the isoelectric line and the tangent through the peak of the T wave and the point of the maximal slope of the T wave. If the U wave was present, the end of the T wave was defined as the minimal point between the T and U wave. For the QRS dispersion, sometimes the end of the QRS complex was difficult to determine, because of a slow slope through a plateau. The duration of this complex was measured up to the intersection of the S wave with the isoelectric line.⁴

In each lead, two QT intervals were measured and the average was used. The obtained values were heart rate corrected, according to the Bazett formula ($QTc = QT/\sqrt{RR}$) and QTdc was obtained. QTm and JTm were calculated for each patient as an average of the QT and JT interval duration in each lead. Each ECG was analysed by two observers, without knowing any clinical data.

Values over 30 kg/m² of the body mass index (BMI) can define the existence of obesity. BMI is a weight/height ratio: $BMI = \text{Weight (in kg)} / [\text{Height (in meters)}]^2$. The BMI was chosen because it reflects the adipose tissue mass of the organism and it is the most used method to define obesity. BMI can be considered a significant predictive factor of coronary deaths.⁵

RESULTS

The 50 patients with chronic myocardial infarction investigated in the study, were divided in two groups: obese (with a BMI > 30 kg/m², 10 patients) and normal weight patients (40 patients). The values obtained for the main variability indices of the QT interval may be seen in Table 2.

Table 2. The values obtained for QTdc, JTdc, QRSdc, QTm and JTm in the obese and normal weight chronic myocardial infarction patients.

Variability index	In obese patients (ms)	In normal weight patients (ms)
QTdc	35 \pm 23	33 \pm 25
JTdc	22 \pm 18	27 \pm 18
QRSdc	32 \pm 27	17 \pm 12
QTm	362 \pm 81	378 \pm 122
JTm	233 \pm 78	285 \pm 106

The t Student test was used to estimate the significance of the differences of the QTdc, JTdc, QRSdc, QTm and JTm in the obese and normal weight chronic myocardial infarction patients. (Table 2)

The differences were not statistically significant for either the dispersion parameter ($p < 0.05$ was considered significant), excepting QRSdc.

Although in non-coronary patients obesity increases QTd, in chronic MI patients, obesity doesn't increase additionally significant QTdc. It is known that coronary disease causes a more important increase of QTd compared to obesity. In other words, electrocardiographic parameters in coronary obese patients do not differ significantly from those of the normal weighted coronary patients. This is the reason why QTdc, JTdc, QTm and JTM don't have to be adjusted in coronary obese patients to the BMI. QRSdc should be adjusted to BMI in coronary patients.

DISCUSSIONS

The absence of any correlation between QTd and the antropometric parameters reflecting the distribution of the adipose tissue (BMI, waist circumference) emerges from an Italian study carried out on patients with uncomplicated obesity without cardiovascular diseases, diabetes, hormonal or electrolyte disorder, who were not taking any medication able to modify repolarisation.⁶

Another study found an increase of the QRS duration, of the QTc interval and even of the QRS voltage in leads DI, DII and DIII in obese patients, and a pathological left axial deviation, changes who were independent of the patients' age, sex or blood pressure.⁷

An American study, indicated that BMI didn't affected QRS duration, but QTd was significantly higher in normal weight coronary patients compared to the obese.⁸ This study didn't find statistically significant differences for QTd, JTm and QRSm (mean QRS interval value) in coronary obese patients compared to the normal weight patients. The differences from the present study could be due to the fact that the American group included not only chronic myocardial infarction patients, but also unstable angina patients, none of the patients were on class III antiarrhythmic therapy or cardiotonics, and electrolyte imbalances, as medical conditions that could affect the QT interval, haven't been excluded. QTd is significantly higher in MI patients compared to those with unstable angina. The effect of class III antiarrhythmic drugs on QT dispersion is controversial: while some authors found a decrease of QTd, others considered that QTd is not modified.⁹⁻¹³ Furthermore, the percent of hypertensive patients is higher in this study (37.5%) compared to the American study (13.78%), and QTdc is significantly higher in hypertensive patients compared to healthy controls¹⁴. Angiotensin converting enzyme inhibitors (ACEI) are known to decrease QTd, and 50% of the patients of this study were given ACEI and only 2.24%

of the patients in Nomura's study.¹⁵ Beta-blockers also reduce QTd, and 16.66% of the American patients were given this type of drugs and only 10% of the Romanian.¹⁶ 12.5% of the patients in the current study had also diabetes, and only 1.92% in the American study, and a high prevalence of increased QT interval duration and dispersion has been found in type 2 diabetic patients.¹⁷

QTd decreases in obese patients on a liquid protein diet, after weight loss. Shortening of the minimum QT interval causes the increased QT dispersion in obesity.² The decrease of the QTc interval and of QTdc during a long lasting weight loss program, mainly in women with visceral obesity, could have clinical significance, reducing cardiovascular risk, including life threatening arrhythmias and sudden death.¹⁸

According to an Italian study, QTc interval positively correlated with the circulating free fatty acids in obese women, and reduction of the body weight significantly decreases the QTc interval and QTdc, strictly correlated with the decrease of the plasma free fatty acid concentration.¹⁸ The same study has demonstrated a significant relation between QTc interval, respective QTd, and plasma epinefrin and norepinefrin concentration, suggesting a disfunction of the autonomic nervous system as a possible mechanism of the QTc interval increase in visceral obesity. QT interval is influenced by the autonomic tonus: sympathetic stimulation unopposed by vagal activity, might induce electrical ventricular instability, resulting in a risk of arrhythmia and sudden death in obese patients.

Hyperinsulinemia and glucose intolerance were associated with QT interval increase in obese, possible secondary to sympathetic activation.⁸

The increase of the BMI impairs myocardial contraction and relaxation, due to the existence of an obese-specific remodeling.¹⁹

Obese MI patients have a lower mortality and risk for major events after coronary angioplasty compared to those with a normal weight.²⁰

Table 3. The values for p obtained when comparing QTdc, JTdc, QRSdc, QTm and JTM in obese and normal weight MI patients.

	QTdc	JTdc	QRSdc	QTm	JTM
p	0.696	0.28	0.0027	0.518	0.48

CONCLUSIONS

Although in patients without a coronary disease, obesity significantly increases QT dispersion, in chronic MI patients, obesity influences significantly only QRSdc, and not QTdc, or JTdc. Ventricular

depolarisation is mostly affected by obesity, and repolarisation seems to be not affected.

In other words, although in patients without a coronary disease obesity increases QTd, in chronic myocardial infarction patients, obesity doesn't additionally increase QTdc significantly. The explanation is that the effects of the myocardial injury counteract the changes present in obese patients.

As a conclusion, the presence of obesity in chronic myocardial infarction patients, does not significantly increase the dispersion parameters of ventricular depolarisation, but affects intraventricular conduction, demonstrated by the higher average QRSdc values in obese patients and by the statistic significant differences of QRSdc between the obese and normal weight MI patients. The arrhythmia and sudden death risk in obese and normal weight MI patients have to be reevaluated.

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