

The Characteristics of QT Interval Dispersion in Obesity-related Hypertensive Patients

H Rui¹, B Yuan², X Wang¹, Z Wang¹

ABSTRACT

Objective: To find the characteristics of QT interval dispersion (QTd) in obesity-related hypertensive patients.

Methods: The study comprised 73 obesity-related hypertensive patients (group A), 58 non-obesity-related hypertensive patients (group B) and 105 healthy persons as control (group C). The maximum QT interval (QTmax), minimum QT interval (QTmin) and QTd were compared.

Results: The QTmax, QTmin and QTd of groups A and B were higher than those in group C; the difference was statistically significant ($p < 0.05$). The QTmax, QTmin and QTd of group A were higher than those in group B; the difference was statistically significant ($p < 0.05$).

Conclusion: High cholesterol and high blood pressure of long standing cause blood lipid metabolic disorders and may lead to blood flow that affects the myocardial cells to varying degrees. QT interval dispersion can be used as a forecast indicator of malignant arrhythmia, so great importance should be given to obesity-related hypertensive patients by timely and effective treatment to improve the survival rate.

Keywords: Hypertensive patients, non-obesity-related, obesity-related, QT interval dispersion

Características de la dispersión del intervalo QT en pacientes con hipertensión relacionada con la obesidad

H Rui¹, B Yuan², X Wang¹, Z Wang¹

RESUMEN

Objetivo: Encontrar las características de la dispersión del intervalo QT (QTd) en pacientes con hipertensión relacionada con la obesidad.

Métodos: El estudio abarcó 73 pacientes con hipertensión relacionada con la obesidad (grupo A), 58 pacientes hipertensos no relacionados con la obesidad (grupo B), y 105 casos de personas sanas como control (grupo C). Se compararon el intervalo máximo (QTmáx), el intervalo mínimo (QTmín), y la dispersión del intervalo QT (QTd).

Resultados: Los valores de QTmáx, QTmín, y QTd de los grupos A y B fueron mayores que los del grupo C; la diferencia fue estadísticamente significativa ($p < 0.05$). Los valores de QTmáx, QTmín, y QTd del grupo A fueron mayores que los del grupo B; la diferencia fue estadísticamente significativa ($p < 0.05$).

From: ¹Henan Province Hospital of TCM (The Second Affiliated Hospital of Henan University of Traditional Chinese Medicine), Zhengzhou, 450014, China and ²Henan University of Traditional Chinese Medicine, Zhengzhou, 450014, China

Correspondence: Dr Z Wang, Department of Cardiology, Henan Province Hospital of TCM (The Second Affiliated Hospital of Henan University of Traditional Chinese Medicine), Zhengzhou, 450014, China. Email: shifenglizz@163.com

Conclusión: *El colesterol alto y la presión arterial prolongada causan trastornos metabólicos de los lípidos de la sangre, y pueden conducir a un flujo sanguíneo que afecte las células del miocardio en diversos grados. La dispersión del intervalo QT se puede utilizar como un indicador para pronosticar la arritmia maligna, de manera que debe concederse gran importancia a que los pacientes con hipertensión relacionada con la obesidad reciban tratamiento oportuno y eficaz para mejorar su tasa de supervivencia.*

Palabras claves: Pacientes hipertensos, relacionados con la no obesidad, relacionados con la obesidad, dispersión del intervalo QT

West Indian Med J 2016; 65 (4): 634

INTRODUCTION

Obesity has been reported as a health killer in many parts of the world, and its prevalence has increased rapidly since 1980 (1). It is estimated that by the year 2030, 38% of the world's adult population will be overweight. Significant global health strategies must reduce the morbidity and mortality associated with the obesity epidemic (2). It has been established in the literature that obesity is associated with an increased risk of developing cardiovascular disease, hypertension, coronary artery disease, heart failure, stroke and death (3–8). Several pathways linking obesity and cardiovascular disease have been described. In a 2016 study, the complex relationship between obesity and cardiovascular disorders, in particular coronary atherosclerosis and heart failure, was proposed (9).

Some studies have shown that QT interval dispersion (QTd) is an independent predictor of adverse cardiovascular outcomes, including death (10–12). QT interval dispersion can predict the risk of arrhythmia. Numerous studies have confirmed the effects of QTd on the evaluation of mortality of coronary artery disease, congestive heart failure, hypertension and other diseases. In this paper, by comparing the QTd differences among obese hypertensive patients, non-obese hypertensive patients and healthy individuals, we gained an understanding into the QTd characteristics of obese hypertensive patients. This will provide the basis for clinical treatment and prognosis.

SUBJECTS AND METHODS

Subjects

A total of 131 hypertensive patients who were treated at the Department of Cardiology of the Second Affiliated Hospital of Zhengzhou University were selected, 78 of whom were male and 53 female, with an average age of 60 ± 9.4 years. These patients satisfied Chinese

Hypertension Prevention Guide recognized standard 2010 (13), and excluded patients with secondary hypertension, electrolyte disorders or cardiomyopathy, and who were not taking drugs that would affect ventricular repolarization. Based on their body mass index (BMI), they were classified as obese (group A) and non-obese (group B).

A total of 105 healthy persons were selected who had had a medical examination in our hospital in the same period (group C), 62 of whom were male and 43 female, with an average age of 53 ± 8.3 years.

Body mass index

This is obtained by dividing the body weight (kg) by height (m) squared (*ie* kg/m^2). The weight and height of the subjects were measured by professional doctors. A BMI of ≥ 25 was taken as the obesity standard.

Measurement of QT intervals

The electrocardiogram (ECG) used was produced by Nihon Kohden: synchronized 15-lead ECG was used for the subjects of the three groups A, B and C in a quiet state and supine position. The maximum QT interval (QTmax) and the minimum QT interval (QTmin) were manually measured. The calculation of QTd (QTmax – QTmin) was done by professionals. Determination of the QT interval indices was from the starting point of the QRS complex to the intersection of the T-wave descending part of equipotential lines, the unclear T-wave or the T, U fusion leads were removed; each measurement needed at least eight leads. Three measurements were taken and the average of the measurements used.

Statistical analysis

All data were analysed statistically using SPSS 16.0 software. Measurement data were presented as mean \pm standard deviation (\pm s) and *t*-test was used; $p < 0.05$ was considered as statistically significant.

RESULTS

The QTmax, QTmin and QTd in group A were markedly higher than those in group C; the difference was statistically significant ($p < 0.05$). The t -value of QTd was 8.606 in group A when compared with group C. It means that QT abnormal rates in group A were higher than those in group C (Table 1).

Table 1: Comparison of QTmax, QTmin and QTd between groups A and C

Group	QTmax	QTmin	QTd
Group A	449 ± 1.9	409 ± 2.3	51 ± 1.7
Group C	388 ± 26.3	362 ± 15.6	36 ± 3.3
t	5.255	6.668	8.606
p	0.001*	0.002*	0.001*

*Compared with group C, $p < 0.05$.

The QTmax, QTmin and QTd in group B were markedly higher than those in group C; the difference was statistically significant ($p < 0.05$). The t -value of QTd was 6.271 in group B when compared with those in group C. It means that QT abnormal rates in group B were higher than those in group C (Table 2).

Table 2: Comparison of QTmax, QTmin and QTd between groups B and C

Group	QTmax	QTmin	QTd
Group B	444 ± 2.7	404 ± 2.5	47 ± 2.3
Group C	388 ± 26.3	362 ± 15.6	36 ± 3.3
t	4.818	6.128	6.271
p	0.001*	0.002*	0.005*

*Compared with group C, $p < 0.05$.

The QTmax, QTmin and QTd in group A were markedly higher than those in group B; the difference was statistically significant ($p < 0.05$). The t -value of QTd was 2.375 in group A when compared with Group B. It means that QT abnormal rates in group A were higher than those in group B (Table 3).

Table 3: Comparison of QTmax, QTmin and QTd between groups A and B

Group	QTmax	QTmin	QTd
Group A	449 ± 1.9	409 ± 2.3	51 ± 1.7
Group B	444 ± 2.7	404 ± 2.5	47 ± 2.3
t	3.506	3.283	2.357
p	0.008*	0.011*	0.046*

*Compared with group B, $p < 0.05$.

The QTmax, QTmin and QTd in groups A and B were markedly higher than those in group C; the difference was statistically significant ($p < 0.05$). The QTmax,

QTmin and QTd in group A were significantly higher than those in group B; the difference was statistically significant (Fig. 1).

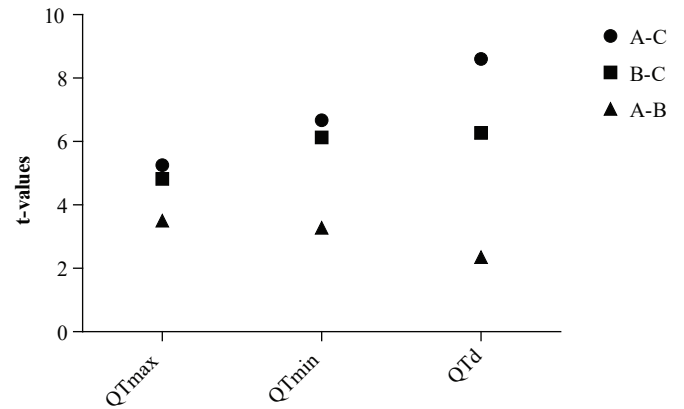


Fig. 1: Comparison of QTmax, QTmin and QTd in groups A, B and C.

DISCUSSION

QT interval dispersion is the difference between the QTmax and QTmin of synchronization leads: QTmax – QTmin (14). It was first proposed by Campbell in 1985.

Day *et al* found a long QTd (15), long QT interval syndrome, and put forward that QTd was an electrocardiology indicator which could reflect cardiac repolarization heterogeneity and reflect the risk of ventricular arrhythmias. Then there were other reports that confirmed the role of QTd in predicting mortality in essential hypertension, congestive heart failure, coronary heart disease and a long QT interval syndrome in populations, fully affirming the clinical value of QTd (16). The greater the QTd value, the greater the instability and asynchronization of cardiac repolarization and the higher arrhythmia risk. Therefore, detection of QTd has an important value in predicting malignant arrhythmic events clinically.

The QT interval was mainly influenced by myocardial ischaemia, autonomic nerve activity, heart rate, drugs and many other factors. The autonomic nervous system plays an important role in regulating cardiac arrhythmia (17, 18). The results of this study showed that QTd values in obese and non-obese hypertensive patients were higher than those in the control group, and higher in obese hypertensive patients than those in non-obese hypertensive ones. This was possibly due to the long-term high blood pressure of patients, increased venous return, ventricular filling and ejection volume increases, resulting in increased ventricular muscle tension and increased myocardial oxygen consumption. At the same time, it may be due to the long-term ejection volume increases, causing myocardial cell proliferation,

ventricular hypertrophy (19), leading to extremely uneven myocardial cell repolarization, QTd increases, causing ventricular tachycardia, ventricular fibrillation and other malignant arrhythmias.

Obesity can contribute to a large number of inflammatory cytokines, and adipokines (20, 21). The blood lipids of obese patients with hypertension increase, and lipid factors released into the blood vessel wall are likely to cause damage to the blood vessel elasticity, accumulation of body fat but also increase the stiffness of the blood vessel wall (22, 23). Blood stasis and lipid metabolism have different effects on haemodynamics, ventricular action potential duration and conductivity changes on myocardial cells, ventricular repolarization disorder, heterogeneity and instability increased, leading to prolonged QTd.

Maintaining a proper body weight is an effective measure in controlling high blood pressure. Despite the small sample size and the lack of patient follow-up, the above study showed that in order to reduce the incidence of malignant cardiac events and improve survival in patients with hypertension, weight control is imperative, which should be based on clinical characteristics. Early intervention is of paramount importance.

REFERENCES

1. Popkin BM, Doak CM. The obesity epidemic is a worldwide phenomenon. *Nutr Rev* 1998; **56**: 106–14.
2. Smith KB, Smith MS. Obesity statistics. *Prim Care* 2016; **43**: 121–35.
3. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. *New Engl J Med* 1999; **341**: 1097–105.
4. Diehr P, Bild DE, Harris TB, Duxbury A, Siscovick D, Rossi M. Body mass index and mortality in nonsmoking older adults: the cardiovascular health study. *Am J Public Health* 1998; **88**: 623–9.
5. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003; **138**: 24–32.
6. Kenchaiah S, Evans JC, Levy D, Wilson PW, Benjamin EJ, Larson MG et al. Obesity and the risk of heart failure. *New Engl J Med* 2002; **347**: 305–13.
7. Dunlap SH, Sueta CA, Tomasko L, Adams KF Jr. Association of body mass, gender and race with heart failure primarily due to hypertension. *J Am Coll Cardiol* 1999; **34**: 1602–8.
8. Katzmarzyk PT, Janssen I, Ardern CI. Physical inactivity, excess adiposity and premature mortality. *Obes Rev* 2003; **4**: 257–90.
9. Mandviwala T, Khalid U, Deswal A. Obesity and cardiovascular disease: a risk factor or a risk marker? *Curr Atheroscler Rep* 2016; **18**: 21.
10. Cin VG, Celik M, Ulucan S. QT dispersion ratio in patients with unstable angina pectoris (a new risk factor?). *Clin Cardiol* 1997; **20**: 533–6.
11. Malik M, Batchvarov VN. Measurement, interpretation and clinical potential of QT dispersion. *J Am Coll Cardiol* 2000; **36**: 1749–67.
12. Somberg JC, Molnar J. Usefulness of QT dispersion as an electrocardiographically derived index. *Am J Cardiol* 2002; **89**: 291–4.
13. Chinese Hypertension Prevention Guide Revision Committee. Chinese Hypertension Prevention Guide 2010. *J Cardiol* 2011; **30**: 579–615.
14. Campbell RWF, Gardiner P, Amos PA, Chadwick D, Jordan RS. Measurement of the QT interval. *Eur Heart J* 1985; **6**: 81–3.
15. Day CP, McComb JM, Campbell RW. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J* 1990; **63**: 342–4.
16. Zabel M, Portnoy S, Franz MR. Electrocardiographic indexes of dispersion of ventricular repolarization: an isolated heart validation study. *J Am Coll Cardiol* 1995; **25**: 746–52.
17. Fink GD. Does our fat tell our brain what to do? A 'sympathetic' appraisal. *J Physiol* 2010; **588**: 1389–90.
18. Grassi G, Seravalle G, Dell'oro R. Sympathetic activation in obesity: a noninnocent bystander. *Hypertension* 2010; **56**: 338–40.
19. Ichkhan K, Molnar J, Sombery J. Relation of left ventricular mass and QT dispersion in patients with systematic hypertension. *Am J Cardiol* 1997; **79**: 508–12.
20. Shi Z, Chen WW, Xiong XQ, Han Y, Zhou YB, Zhang F et al. Sympathetic activation by chemical stimulation of white adipose tissues in rats. *J Appl Physiol* 2012; **112**: 1008–14.
21. De Boer MP, Meijer RI, Wijnstok NJ, Jonk AM, Houben AJ, Stehouwer CD et al. Microvascular dysfunction: a potential mechanism in the pathogenesis of obesity-associated insulin resistance and hypertension. *Microcirculation* 2012; **19**: 5–18.
22. Song HB, Liu ZD, Lu FH. Effects of different types of primary arterial stiffness in obese patients with hypertension. *Chin J Arterioscl* 2013; **22**: 737–606.
23. Pierdomenico SD, Di Nicola M, Esposito AL, Di Mascio R, Ballone E, Lapenna D et al. Prognostic value of different indices of blood pressure variability in hypertensive patients. *Am J Hypertens* 2009; **22**: 842–7.