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A study of QRS complex in ECG in asymptomatic obese young patients

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Abstract

Obesity causes several haemodynamic changes such as increased blood and stroke volume, and an increase in pulmonary and left atrial pressure. These changes cause structurally altered cardiac tissue such as left atrial enlargement and remodelling, and ventricular hypertrophy. These may ultimately result in obesity-induced left ventricular diastolic and systolic dysfunction and right and left ventricular heart failure.

Keywords: Haemodynamic, changes, cardiac, ECG, obesity

Introduction

Obesity problem was only there in the urban world and the western countries but since the globalization has taken place at tremendous speed its complications are felt not only in the urban dwelling population of our country but also studies have been reported in the rural areas also. Obesity causes several haemodynamic changes such as increased blood and stroke volume, and an increase in pulmonary and left atrial pressure^[1, 2]. These changes cause structurally altered cardiac tissue such as left atrial enlargement and remodelling, and ventricular hypertrophy^[1, 2]. These may ultimately result in obesity-induced left ventricular diastolic and systolic dysfunction and right and left ventricular heart failure^[1, 2].

Some obesity-induced adverse effects on cardiac function can be identified on a 12-lead electrocardiogram (ECG). This includes an increased P-wave duration and dispersion^[3-6], prolongation of the PR interval^[3-7], low QRS voltage in the limb leads^[7-9], leftward shift of the heart axis^[7-11], various markers of left ventricular hypertrophy^[12-14] and prolongation of the corrected QT interval and prolonged QT-interval duration^[8]. Many of these electrocardiographic abnormalities have been reported to be reversible with substantial weight loss thereby reinforcing the association between BMI and electrocardiographic changes^[2, 8].

These electrocardiographic changes are well-documented in obese individuals. However, to which extent these electrocardiographic changes are associated with BMI in healthy young individuals with a normal BMI (18.5–25 kg/m²) is largely unknown. In addition, subtle physiological changes in these individuals are of particular interest in early phase pharmaceutical research because they help differentiate between normal physiological changes or potentially harmful or unknown pharmacodynamic effects. The aim of the present analysis was to evaluate the association between obesity and QRS complex derangements in young asymptomatic adults.

Aims and Objectives

The aim of the present analysis was to evaluate the association between obesity and QRS complex derangements in young asymptomatic adults.

Materials and Methods

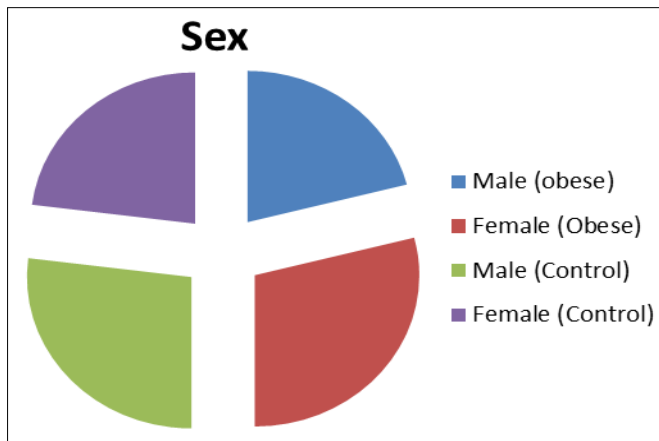
This study was done in the Department of Physiology, MIMS Mandya. This study was done from Feb 2018 to Jan 2019. One hundred candidates participated in the study. Out of these 50 were so selected who were obese according to BMI charts. The other fifty were so selected who fell in the normal BMI group.

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Height (HT) was measured in barefoot to the nearest 0.1cm using a vertical height scale. Body weight (WT) was recorded to the nearest 0.1 kg using a portable weighing machine. Body mass index (BMI) was calculated as weight divided by height squared (kg/m²).

Standard 12-lead electrocardiogram (Philips Company Page Writer 300pi) was taken after 10 minutes of rest. QRS interval and frontal plane QRS axis were measured using standard techniques.⁹ Voltage was recorded as the maximum amplitude of the R wave and Q or S waves in leads I, II and III to identify low voltage patterns. Data was analyzed by applying appropriate statistical tests by using SPSS package (version 14). Data was expressed in terms of mean ± SD. Unpaired 't'-test was used to study the changes in ECG variables. P value < 0.05 was taken as significant.

Results



Graph 1: Sex Distribution

Table 1: Age

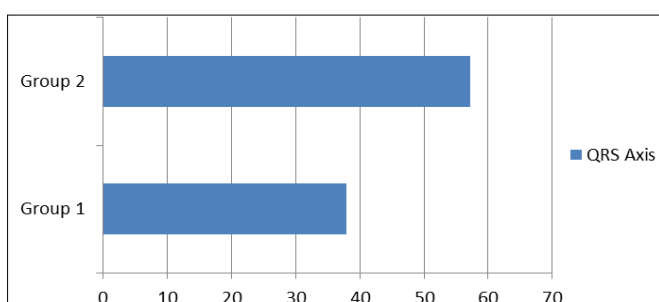
	Mean Age	Std. Deviation
Group 1	26.68	3.79
Group 2	29.76	6.86

Table 2: BMI

	Mean BMI	Std. Deviation
Group 1	31.87	1.87
Group 2	23.84	1.22

Table 3: QRS Study

Mean QRS Axis	Case	37.98
	Control	57.28
QRS Amplitude	Case	0.008
	Control	0.008
QRS Duration	Case	0.85
	Control	0.84



Graph 2: QRS Complex

Discussion

Until now, the only electrocardiographic items in which a normal trend with age has been recognized are those which relate to the electrical heart position: QRS axis, heart index, and the position indicated by the unipolar limb leads. But even with these items the reported age trends are inadequate to establish norms because of lack of definition of other characteristics (for example, body build and relative obesity), as well as the lack of satisfactory statistical analysis. In regard to the effect of body weight, it is well known that overweight tends to be associated with a left axis shift and that underweight persons tend to show right axis deviation. These tendencies are usually explained in terms of mechanical changes in the heart position but they have not been systematically studied or analyzed statistically.

Left atrial enlargement (LAE) is associated with an increased prevalence of atrial fibrillation, cardiovascular events and death^[13]. Obesity is found to be the most important risk factor for LAE development in the general population, and is dependent on the extent of obesity^[2-6]. Furthermore, LAE is also independently related to age, hypertension, BMI, waist circumference, and metabolic syndrome. Additionally, obesity is the strongest predictor of LAE in hypertensive patients, and is under the influence of race and gender. These structural changes can be observed on the twelve-lead surface ECG through increased P-wave duration, P-wave area, and P-wave dispersion^[2-6]. Obesity-associated electrocardiographic changes such as an increased P-wave duration (5–22 ms) and P-wave dispersion (14–25 ms)^[3-6], increased PR interval (5–13 ms)^[3-7] and a leftward shift of the heart axis (11–37 degrees) compared with adults with a normal BMI were reported^[7-11]. In the present analysis, we found a relation between BMI and these indices of atrial size. Although no left atrial measurement was performed these results suggest that atrial size may also be related to BMI in healthy individuals with a normal BMI (18.5–25.0 kg/m²).

Presumably, increased epicardial and pericardial fat, which are increased in obesity, further induce these changes^[2]. Cardiac fat depositions were found to have metabolic and inflammatory functions which can contribute to the fibrotic remodelling of the atrial tissue^[1,2]. These fat depositions are significantly increased in obesity and are believed to induce the abovementioned electrocardiographic changes^[1,2]. Hypothetically, the volume of epicardial and pericardial fat is also dependent on BMI in young, non-obese individuals. This may be an additional explanation for the association that was observed in the present analysis between BMI and the above-mentioned electrocardiographic changes.

Leftward shifts of the P-wave, QRS and T-wave axes (11–37 degrees) are reported in obese patients compared with healthy controls^[7-11]. The cause of these shifts is uncertain, but may be related to a leftward and more horizontal orientation of the heart attributed to the diaphragmatic pressure from central obesity, independent from left ventricular hypertrophy^[7-11]. This explains the association between lower BMI and rightward P-wave and QRS axes and independently from left ventricular mass^[11]. This is in line with our findings and presumably, the leftward change in heart axis that was observed in the present analysis is caused by an increase in diaphragmatic pressure which is dependent on BMI.

Conclusion

This study concludes that apparently healthy obese individuals may have higher anthropometric values and abnormal ECG findings.

References

1. Alpert MA, Omran J, Bostick BP. Effects of Obesity on Cardiovascular Hemodynamics, Cardiac Morphology, and Ventricular Function. *Curr Obes Rep* 2016;5(4):424–434. Doi: 10.1007/s13679-016-0235-6.
2. Lavie CJ, Pandey A, Lau DH, Alpert MA, Sanders P. Obesity and Atrial Fibrillation Prevalence, Pathogenesis, and Prognosis: Effects of Weight Loss and Exercise. *J Am Coll Cardiol* 2017;70(16):2022–2035. Doi:10.1016/j.jacc.2017.09.002.
3. Vaidean GD, Manczuk M, Magnani JW. Atrial electrocardiography in obesity and hypertension: Clinical insights from the Polish-Norwegian Study (PONS). *Obesity* (Silver Spring. MD 2016;24(12):2608–2614.
4. Babcock MJ, Soliman EZ, Ding J, A Kronmal Ding R, Goff DC. Pericardial fat and atrial conduction abnormalities in the Multiethnic Study of Atherosclerosis (MESA) *Obesity* 2011;19(1):179–184. Doi:10.1038/oby.2010.121.
5. Liu T, Fu Z, Korantzopoulos P et al. Effect of obesity on p-wave parameters in a Chinese population. *Ann Noninvasive Electrocardio* 2010;15(3):259–263. Doi: 10.1111/j.1542-474X.2010.00373.x.
6. Magnani JW, Lopez FL, Soliman EZ et al. P wave indices, obesity, and the metabolic syndrome: the atherosclerosis risk in communities study. *Obesity*. Silver Spring Md.) 2012;20(3):666–672. Doi: 10.1038/oby.2011.53.
7. Frank S, Colliver JA, Frank A. The electrocardiogram in obesity: statistical analysis of 1,029 patients. *J Am Coll Cardiol* 1986;7(2):295–299. Doi: 10.1016/S0735-1097(86)80494-6.
8. Fraley MA, Birchem JA, Senkottaiyan N, Alpert MA. Obesity and the electrocardiogram. *Obes Rev* 2005;6(4):275–281. Doi:10.1111/j.1467-789X.2005.00199.x.
9. Graner M, Pentikainen MO, Siren R et al. Electrocardiographic changes associated with insulin resistance. *NMCD* 2014;24(3):315–320.
10. Eisenstein I, Edelstein J, Sarma R, Sanmarco M, Selvester RH. The electrocardiogram in obesity. *J Electrocardiol* 1982;15(2):115–118. doi: 10.1016/S0022-0736(82)80003-4.
11. Kurisu S, Ikenaga H, Watanabe N et al. Electrocardiographic characteristics in the underweight and obese in accordance with the World Health Organization classification. *IJC Metabolic. Endocrine* 2015;9:61–65.
12. Okin PM, Jern S, Devereux RB, Kjeldsen SE, Dahlof B. Effect of obesity on electrocardiographic left ventricular hypertrophy in hypertensive patients: the losartan intervention for endpoint (LIFE) reduction in hypertension study. *Hypertension* 2000;35(1 Pt 1):13–18. Doi: 10.1161/01.HYP.35.1.13.
13. Lavie CJ, De Schutter A, Parto P, et al. Obesity and Prevalence of Cardiovascular Diseases and Prognosis-The Obesity Paradox Updated. *Prog Cardiovasc Dis* 2016;58(5):537–547. Doi: 10.1016/j.pcad.2016.01.008.
14. Kurisu S, Ikenaga H, Watanabe N, et al. Implications of World Health Organization classification for body mass index on the correlations between common electrocardiographic indexes for left ventricular hypertrophy and left ventricular mass. *Clin Exp Hypertens* 2016;38(8):715–720. Doi: 10.1080/10641963.2016.1200604.