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A comparative study of electrocardiographic QRS complex in asymptomatic obese young adults

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Abstract

Background: Standard 12-lead ECG is widely used initial screening test for non-invasive detection of cardiovascular changes in obesity. It is seen that obesity is associated with leftward shift of the P wave, QRS and T wave axis, various changes in the P wave morphology, and low QRS voltage, which remain asymptomatic and hence undiagnosed for a longer period of time.

Objectives: To study and compare the QRS axis (in degrees), QRS duration (in seconds) and QRS amplitude (in mV) in asymptomatic obese young adults and apparently healthy normal subjects.

Material and Methods: Asymptomatic obese 150 young adults in the age group of 18 – 39 years with BMI ≥ 30 kg/m^{2}as study subjects, age and sex matched 150 healthy individuals with BMI <22 kg/m^2 were taken as controls from the general population. The weight in kilograms and height in meters was measured and Body Mass Index (BMI), Body Surface Area (BSA) and Waist-Hip Ratio (WHR) were calculated using standard methods. A 12-lead ECG was taken after 10 minutes of rest. Data was expressed in terms of mean ± SD. Unpaired 't'-test was done to study the difference in QRS complex.

Results: Mean QRS axis (in degrees) in study subjects was 38.9±33.6 vs 56.5±33.4 in controls and was statistically significant. QRS duration (in seconds) in study subjects was 0.09 ± 0.01 vs 0.08±0.01 in controls and was statistically significant. QRS amplitude was increased in cases but was not statistically significant.

Conclusion: Our results suggest that obese individuals, being asymptomatic, have higher anthropometric values and abnormal QRS complex, which needs a regular check to reduce the chances of their manifestation, at a future date.

Keywords: Anthropometry, Electrocardiogram, Body Mass Index, Cardiovascular Diseases, QRS complex.

Introduction

Obesity is the current serious public health problem with established cardiovascular co-morbidities and a major cause of sudden death in developed as well as developing countries.[1]From 1980 onwards, the incidence of obesity started growing to epidemic proportions.[1] Based on data from the 2007 National Family Health Survey (NFHS)[2], 12.1% males and 16% females are overweight or obese. In Karnataka, 14% males and 17.3% females are obese or overweight. This "epidemic" is mainly because of rapid lifestyle changes involving physical inactivity, eating habits like eating in-between meals, preference to sweets, refined foods and less or no exercise at all.[3]

Obesity is associated with a wide variety of electrocardiographic (ECG) abnormalities like the leftward shifts of the P wave, QRS and T wave axes, various changes in the P wave morphology, low QRS voltage, various markers of left ventricular hypertrophy, particularly the Cornell voltage and product, T wave flattening, lengthening of the corrected QT interval and prolonged QT interval duration.[4] Computer assisted analysis of the small high-frequency ECG potentials (1 to 20μV) by signal averaged-electrocardiography (SAECG)[5] seen at the end of the QRS complex and into the ST segment have been correlated with increased risk of ventricular arrhythmias and sudden cardiac death.

Two major determinants of ECG's sensitivity are age and obesity. In cardiac medicine, the resting ECG has proved its value as a diagnostic tool for detecting heart disease and a prognostic tool in apparently healthy subjects[6]. Interplay between several factors, such as displacement of the heart horizontally by the elevated diaphragm, cardiac hypertrophy, increase in the distance between the heart and the electrodes and coexisting sleep apnea-hypoventilation syndrome, tend to modify the ECG in obese patients. The electrocardiographic voltage is attenuated by its passage through a thickened and fat-laden chest wall and is related to many factors, including anatomic position of the heart in the thorax, amount of fatty infiltration of the myofibrils, type of electrocardiographic leads for measuring voltage, degree of associated chronic lung disease and left ventricular muscle mass.[7]

Very few studies have been conducted in India. So, this study was taken up to detect any significant electrocardiographic changes occurring in obese young adults which usually goes undiagnosed for a long period of time until it becomes symptomatic.

Material and Methods

A comparative cross-sectional control study was done in which, subjects were screened from the general population in Bagalkot during health camps conducted by S. Nijalingappa Medical College and Hanagal Sri Kumareshwar Hospital and Research Centre. All the subjects were assessed clinically by thorough history taking and detailed clinical examination. Ethical clearance was obtained from the S. Nijalingappa Medical College Ethical Committee. Informed consent was taken in the form of signature on the informed consent form.

The sampling procedure was done by simple random sampling (lottery method) and150 subjects were randomly selected from this group who were asymptomatic obese young adults[8] in the age group of 18 – 40 years with BMI ≥ 30 kg/m^2. Age and sex matched 150 healthy individuals with BMI < 25 kg/m^2 were taken as controls from the general population. History taking included all present, past and recent illnesses of the subject. They were explained about the purpose and the procedure of the study. Subjects were taken into confidence to relieve their apprehension. The data was collected in the Research Laboratory of Department of Physiology.

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/m^2). Using a measuring tape, the waist circumference (WC) was measured at the level midway between the lowest rib margin and the iliac crest, the hip circumference (HC) at the widest level over the trochanters and the thigh circumference directly below the gluteal fold of the left leg. Waist-Hip ratio (WHR) was calculated as waist circumference divided by hip circumference. Body surface area (BSA) was measured by using Dubois Nomogram.

General Physical Examination as well as Systemic Examination was done to rule out the exclusion criteria. 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

Age distribution: The mean age (in years) of 150 cases was 31.6±5.5 and that of 150 controls was 31.9±6.8 (P = 0.68) and was statistically non-significant.

Gender distribution: Out of 150 cases and controls, 90 (60%) were males and 60 (40%) were females.

Anthropometric Characteristics: (Table 1)

The mean Body Mass Index (BMI) (in kg/m^2) in cases was 33.6±3.5 and in controls was 21.6±3.3 which was significant(P <0.00).

Mean Body Surface Area (BSA) in cases was 1.60±0.12 and in controls was 1.49±0.10 and was also significant(P<0.00).

Mean Waist Hip Ratio (WHR) in cases was 0.96±0.02 and in controls was 0.95±0.02 and was significant(P<0.00).

Table 1: Anthropometric Characteristics of two groups

	Group	Mean	S.D	t value	P value
BMI (kg/m^2)	Cases	33.6	3.5	30.78	0.00*
	Controls	21.6	3.3		
BSA (sq. m)	Cases	1.60	0.12	8.47	0.00*
	Controls	1.49	0.10		
WHR	Cases	0.96	0.02	5.37	0.00*
	Controls	0.95	0.02		

***P = 0.00 (Significant)**

Electrocardiographic parameters: (Table 2)

Various electrocardiographic parameters obtained in both case and control group were analyzed statistically by applying unpaired 't' test. The results were as follows.

1. Mean QRS axis (in degrees) in cases was 38.9±33.6 and in controls 56.5±33.4. This was statistically significant. (P = 0.00) **(Fig. 1)**
2. QRS duration (in seconds) in cases and in controls was 0.09±0.01 and 0.08±0.01 respectively, the difference being highly significant. (P = 0.00)
3. QRS amplitude (in mV) in cases was 0.85±0.16 and in controls, 0.85±0.27 and was not significant. (P = 0.87)

Table 2: Electrocardiographic parameters

	Group	Mean	S.D	t value	P value
Mean QRS axis (degrees)	Case	38.9	33.6	4.55	0.00*
	Control	56.5	33.4		
QRS duration (sec)	Case	0.09	0.01	5.92	0.00*
	Control	0.08	0.01		
QRS amplitude (mV)	Case	0.85	0.30	0.16	0.87
	Control	0.85	0.27		

***P = 0.00 (Significant)**

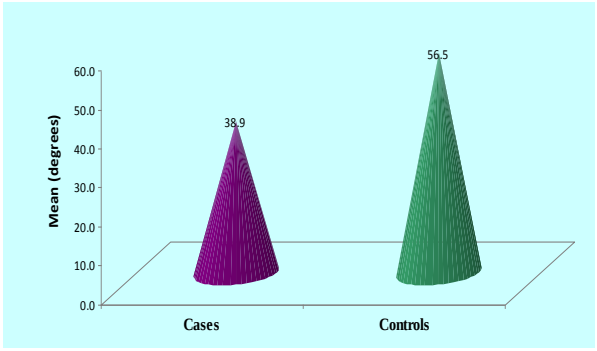

Fig. 1: QRS Axis in the two groups

Chi-square test was applied to study the nature of QRS axis. **(Table 3)**136 cases (90.7%) and 150 controls (100%) had normal QRS axis.12 cases (8%) had left axis deviation and 2 cases (1.3%) had right axis deviation. Chi-square value is 14.68, df = 2, P value is 0.001 and it is highly significant.

Table 3: Nature of QRS Axis

Nature of QRS Axis		Group		Total
		Cases	Controls	
Normal	No.	136	150	286
	%	90.7%	100%	95.3%
Left Axis Deviation	No.	12	0	12
	%	8%	0%	4%
Right Axis Deviation	No.	2	0	2
	%	1.3%	0%	0.7%
Total	No.	150	150	300
	%	100%	100%	100%
Chi-square	Df	P value		
14.68	2	0.001	Significant	

Discussion

In cardiac medicine, resting ECG has proved its value as a diagnostic tool for detecting "silent" heart disease.[10,11] Apart from its use in the clinical field, the ECG has also been employed as a prognostic tool in relatively healthy subjects. The intrinsic value of the ECG as a valuable diagnostic tool, according to Bayes' theorem, is dependent on the prevalence of findings in the general population.[12,13] Therefore, accurate estimation of the true prevalence of ECG abnormalities in large samples help in interpretation of the predictive value of ECG findings. The use of ECGs in epidemiological research has been greatly facilitated by the introduction of Minnesota code classification system.[14,15]

In this present comparative, cross-sectional study, we have compared the anthropometric characteristics and electrocardiographic parameters of 150 young obese asymptomatic subjects with that of apparently healthy age and sex matched normal weighing controls. The difference in the mean value of each parameter between the two groups is analyzed and discussed.

Anthropometric characteristics: There was statistically significant increase in BMI, BSA and WHR in the study group with mean BMI 33.6±3.5 kg/m^2, mean BSA 1.60±0.12 square meters and mean WHR 0.96±0.02. BMI is the most frequently used measure of obesity in the population health surveys. BMI does not however take into account the proportion of the weight which is related to the increased muscle mass or the excess distribution of fat within the body, both of which affect the health which are associated with obesity[16]. Several studies in adults have reported a stronger positive association between the cardiovascular risk factors such as hypertension, lipid and glucose concentrations with abnormal adiposity.[16,17,18]

Electrocardiographic Parameters

QRS duration and voltage: QRS duration in case and in controls was 0.09±0.01 seconds, and 0.08±0.01 seconds respectively and was highly significant, suggesting progressive QRS widening which is in accordance with the studies of Frank and his colleagues in 1029 obese subjects.[19] A prolongation of the QRS duration (>0.10 s) was associated with lower left ventricular ejection fraction and larger end-systolic and end-diastolic volumes and thus is a specific indicator of left ventricular dysfunction.[20] The perturbed depolarization associated with QRS prolongation may also play a direct role in sudden cardiac death via facilitation of reentrant tachyarrhythmias.[21]

QRS voltage in cases was 0.85±0.16 and in controls was 0.85±0.27 and was not significant, suggesting a slightly low voltage in obese individuals. Proger was the first to report low QRS voltage in obese patients in 1931.[22] Alpert et al reported a 11% incidence of low QRS voltage (< 0.5 mm in the standard leads and ≤ 10 mm in the precordial leads) in 100 normotensive morbidly obese patients compared to a 1% incidence in 100 normal lean subjects (P < 0.001).[23] In contrast Eisenstein and his colleagues reported no significant difference in the incidence of low voltage in moderately-to-severe obese subjects.[24] Frank and his co-workers reported a 3.9% incidence of increasing amplitude of QRS voltage with increasing percent overweight in 1029 subjects.[19] Speculation exists that low voltage, when present, is attributed to excessive chest wall fat and in some cases, increased epicardial fat, including anatomic position of the heart in the thorax, infiltration of fat in the myofibrils, type of electrocardiographic leads for measuring voltage, degree of associated lung disease and left ventricular muscle mass. Alpert et al[23] reported significant reduction of the frequency of low voltage (from 13% to 2%) with substantial weight loss in 60 normotensive morbidly obese patients. Brohetet al[25] evaluated electrocardiographic changes with weight reduction and noted a decrease in QRS amplitude, and concluded that the changes were too slight to be clinically apparent. In our study we did not estimate for left ventricular

hypertrophy (LVH) using the ECG criteria like Cornell product criteria,[26] Framingham criterion, Romhilt-Estes point score, left ventricular strain, Sokow-Lyon voltage criteria.[27]

QRS Axis: Out of 150 asymptomatic obese cases, 12 cases (8%) had left axis deviation and 2 cases (1.3%) had right axis deviation (Chi-square value 14.68, df = 2) and it was highly significant. Mean QRS axis in case was 38.9±33.6 degrees and in control group it was 56.5±33.4 degrees and was statistically significant. This is in accordance with many studies like Frank and his colleagues, who reported leftward mean QRS axis in 1029 obese subjects, which became more pronounced with increasing obesity.[19] Leftward shifts in the P wave, QRS and T wave axes were originally reported by Proger in 1931.[20] Subsequently, Eisenstein et al. reported that mean P wave, QRS and T wave axes were normal, but somewhat leftward in 144 obese subjects compared to 100 normal lean controls.[24] A subsequent study by these investigators demonstrated that substantial weight loss in 60 morbidly obese patients produced rightward movement of the mean P wave, QRS and T wave axes from their leftward location.[23] The cause of this axis shift is uncertain, but may relate to a leftward and more orientation of the heart horizontally attributed to the diaphragmatic pressure from visceral obesity.[23,24] Thus, obesity is associated with a leftward shift in QRS axis, that is directly related to the severity of obesity and is reversible with substantial weight loss.

Limitations

Sample size was too small. We did not correlate the age, sex and BMI with the ECG parameters. We did not estimate for left ventricular hypertrophy (LVH) using the ECG criteria.

Conclusion

Obesity is associated with a wide variety of ECG alterations like shift of QRS axis, mainly the left axis deviation, prolongation of the QRS duration and low QRS voltage suggesting left ventricular dysfunction and reentrant tachyarrhythmia. Thus, it can be concluded that apparently healthy obese individuals may have higher anthropometric values and abnormal ECG findings. Hence, a regular check on these parameters will help them in reducing the chances of its manifestation at a future date.

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