P wave Morphology and Dispersion in asymptomatic obese young adults – A comparative cross-sectional study

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Abstract

Background: Considerable variations in the P wave morphology are noted in association with obesity. Standard 12-lead ECG is still considered to be the initial screening test for non-invasive detection of cardiovascular changes.

Objective: To study the duration of P wave (milliseconds), P wave amplitude (mV) and dispersion of P wave (milliseconds) in obese subjects and to compare them with apparently healthy normal subjects.

Material and Method: Asymptomatic obese 150 young adults in the age group of 18– 39 years with BMI $\geq$ 30 kg/m^2, sex and age matched 150 healthy individuals with BMI less than or equal to 22 kg/m^2 were considered as controls from the general population. A standard 12-lead ECG after 10 minutes of rest was taken and duration of P wave and amplitude was noted. Minimum (min) and maximum (max) duration of P wave and dispersion of P wave (the difference between the max and min P wave duration) were measured by transferring the 12-lead ECG to personal computer via scanner. It was magnified 400 times by Adobe Photoshop software to pick up the smallest changes. Data was analyzed in terms of mean $\pm$ SD. Unpaired 't' test was used to study the changes in P wave.

Results: P wave duration was statistically extremely significant (104.3 ± 8.2 vs 95.7 ± 79, P value < 0.0001) and dispersion of P wave was statistically extremely significant (21.1 ± 3.4 vs 14.6 ± 3.9, P value < 0.0001) in obese subjects. P wave amplitude was statistically not significant.

Conclusion: Duration and dispersion of P wave was longer in asymptomatic obese individuals, increasing the risk of atrial flutter and fibrillation. Hence, a regular check on this parameter is needed to reduce its manifestation at a future date.

Keywords: Anthropometry, Electrocardiogram, Body Mass Index, P Wave Dispersion, Cardio-Vascular Diseases.

Introduction

Obesity is a serious public health problem and is a major cause for sudden death. It's a well-known fact that obesity is an independent risk factor for cardio-vascular accidents and is linked with hypertension, dyslipidemia, type-2 diabetes mellitus, coronary artery diseases and metabolic syndrome. Obesity is now an epidemic disease in the industrialized world and its prevalence is increasing in the developing countries like India.[1] The search for improved methods of electrocardiographic detection of cardiovascular abnormalities in the general population has gained lot of popularity in the recent years with increasing use of digital electrocardiogram (ECG).

Electrocardiogram (ECG)[2] is the algebraic sum of the potential fluctuations of the individual myocardial fibers during the cardiac cycle recorded extracellularly on the surface of the precordium. Two major determinants of ECG's sensitivity are age and obesity. The impact of obesity on cardiovascular changes manifests earlier in ECG according to several studies. Obesity is manifesting with a wide variety of ECG abnormalities like the left shifts of the P wave, QRS and T wave axis, changes in the morphology of P wave, low voltage of QRS, left ventricular hypertrophy determined by the Cornell voltage and product criteria, flattened T wave, lengthened corrected QT interval and prolonged duration of QT interval manifesting as life threatening ventricular tachyarrhythmia, atrial flutter and atrial fibrillation (AF). Recently, a new marker, dispersion of P wave, has been included as a predictor of atrial fibrillation (AF). P wave dispersion is the difference between maximum (max) and minimum (min) P wave duration and it signifies discontinuous, homogeneous propagation of sinus node impulses.[4]

This study was taken up to investigate P wave morphology and P wave dispersion of ECG in asymptomatic obese young adults, and thereby reducing its manifestations in the near future, as there are very few studies conducted in India.

Material and Method

This is a comparative cross-sectional study wherein the subjects were screened from the urban population of Bagalkot during health camps conducted by S. Nijalingappa Medical College and Hanagal Sri Kumareshwar Hospital and Research Centre. Ethical clearance from the Institutional Ethical Committee was obtained. The sampling procedure adopted was simple random sampling (lottery method) wherein 150 subjects were randomly selected who were asymptomatic obese young adults[5] in the age group of 18 – 39 years with BMI $\geq$ 30 kg/m^2. Sex and age matched 150 healthy individuals with BMI less than or equal to 22 kg/m^2 were

taken as control from the general population. They were explained about the purpose and the procedure of the study and informed consent was taken in the form of signature on the informed consent form. Subjects were taken into confidence to relieve their apprehension and the study was conducted in the Research Laboratory of Department of Physiology.

History taking included all present, past and recent illnesses of the subject. General Physical Examination as well as Systemic Examination was done to rule out the exclusion criteria. The subjects less than 18 years and more than 39 years with BMI 23-30 kg/m^2 were excluded from the study. Subjects previously diagnosed to have anaemia, thyroid dysfunction, electrolyte abnormalities, known valvular heart disease, coronary artery disease, heart failure, ECG abnormalities such as left bundle branch block, atrio-ventricular conduction defects and recent hospitalizations or any medications like antiarrhythmic agents, tricyclic antidepressants, antipsychotics and antihistaminics were excluded from the study.

Height (HT) was measured to the nearest 0.1cm barefoot using a vertical height scale. Body weight (WT to the nearest 0.1 kg was recorded using a weighing machine (Dolphin Company). Body mass index (BMI) as weight divided by height squared (kg/m^2) was calculated.

A Standard 12-lead ECG (Philips Company Page Writer 300pi) was recorded with a paper speed of 25mm/s with a calibration of 1 mV = 10mm, with the patient in the postprandial state and supine position. P wave morphology was studied mainly in lead II and V1. P wave duration, max P wave and min P wave duration and amplitude, were calculated using standard techniques.[6] Duration of P wave was defined as the time measured from the onset of the P wave to the end of the deflection of P-wave. Onset of P wave was considered as the junction between the iso-electric line and first visible upward or downward slope of the trace, the return of the trace back to its iso-electric line was considered to be the end of the P wave. P wave duration was measured by transferring the 12-lead ECG to a personal computer via a scanner and magnified 400 times to pick up the smallest variations by Adobe Photoshop software (Adobe Systems, Mountain View, CA). Max duration of P wave was defined as the longest duration of P wave and minimum duration of P wave was defined as the shortest duration of P wave. The difference between max and min P wave duration was P wave dispersion. By applying appropriate statistical tests using SPSS package (version14) data was analysed and expressed in terms of mean ± SD. Unpaired 't'-test was used to study the changes in P wave.

Results

This was a comparative cross-sectional study of electrocardiogram done in 150 asymptomatic obese young adults (BMI > 30 kg/m^2) and 150 non-obese (BMI

< 25 kg/m^2) controls. Out of 150 obese subjects and controls, 90 (60%) were males and 60 (40%) were females.

Max P wave duration (m sec) in obese subjects was 104.3 ± 8.2 and in controls was 95.7 ± 7.9. P value was < 0.0001 and was statistically extremely significant. Min P wave duration (m sec) was 83.8 ± 7.5 and in controls was 82.5 ± 6.8. P value was < 0.1168 and was statistically non-significant. P wave dispersion was 21.1 ± 3.4 and in controls 14.6 ± 3.9. P value was < 0.0001 and was statistically extremely significant (Table 1. Fig.1). P wave amplitude (in mV) in both obese subjects and controls was 0.11 ± 0.03 (Table 1).

Table 1: P wave in both groups

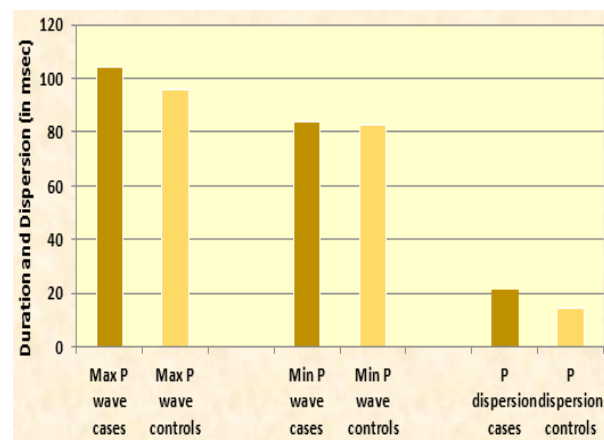|	Group	Mean	S.D	t value	P value
Max P wave duration (ms)	Case	104.3	8.2	9.25	0.0001*
	Control	95.7	7.9		
Min P wave duration (ms)	Case	83.8	7.5	1.57	0.1168
	Control	82.5	6.8		
P wave dispersion (ms)	Case	21.1	3.4	15.38	0.0001*
	Control	14.6	3.9		

*P = 0.0001 (Extremely Significant)

Fig. 1: P wave duration and Dispersion

Discussion

In cardiac medicine, resting ECG has proved its value as a diagnostic tool for detecting "silent" heart diseases.[7,8] Apart from its use in the clinical field, the ECG has also been employed as a prognostic tool in relatively healthy subjects. Likewise, Body Mass Index is the most frequently used obesity measure in the health surveys. However, BMI doesn't 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.[9] Several studies in adults have suggested a strong positive association between the risk factors of cardiovascular system such as hypertension, lipid profile

and glucose concentrations with abnormal adiposity.[9-11] In our study there was statistically significant increase in BMI with mean BMI 33.6 ± 3.5 kg/m^2.

P wave in the ECG signifies atrial depolarization and the P wave duration suggests the transmission of cardiac impulses from the SA node through the intra-atrial and inter-atrial tracts to the AV node, thereby depolarizing all the left and right atrial muscle fibers. It is known that obese patients have 50% risk of atrial fibrillation and flutter.[12] Left atrial enlargement leads to increase in the P wave duration and amplitude that causes atrial fibrillation. In addition, the autonomic control of the heart is abnormal in obese subjects due to predominance of sympathetic over parasympathetic drive. This affects intra-atrial and inter-atrial conduction time making the heart more prone to develop atrial arrhythmias, such as atrial flutter and fibrillation.[4] It has been studied that increased P wave duration and dispersion is associated with prolonged atrial conduction time.

In this present comparative, cross-sectional study, we have compared the P wave duration and dispersion and P wave amplitude 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 was analyzed. Max P wave duration and dispersion was extremely statistically significant in obese subjects. P wave amplitude in obese subjects and in controls was not statistically significant. Thus, it can be said that duration of P wave and dispersion is longer in obese individuals making them more vulnerable for atrial arrhythmias.

In obese patients, enlargement of the left atrium and electrical instability may be caused due to elevated plasma volume, diastolic dysfunction in left ventricle and enhanced neuro-hormonal activity. Many studies have shown that changes in left atrial pressure and dimension may influence P wave duration.[13,14] It was reported by Lars Frost et al that the risk of atrial flutter or fibrillation with increasing BMI was slightly smaller in women than in men.[15] Duru and his colleagues noted that P wave duration and dispersion, decreased significantly after substantial (10%) weight loss and the decrease in P wave dispersion clearly correlated with the amount of weight loss.[16]

The limitations of our study were that our sample size was too small; we did not correlate the age, sex and BMI with the ECG parameters, and there was no follow up study like weight reduction, done in these individuals; which is our future endeavor.

Concluding, obesity is associated with prolongation of the P wave duration, thus increasing the possibility of atrial enlargement and arrhythmias, more specifically determined by studying P wave dispersion. Thus, apparently healthy obese individuals who have higher anthropometric values can also develop 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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