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Original research article

Assessing the effect of obesity on P-wave duration and P-wave dispersion and possible relationship between P-wave measurements and the clinical and echocardiographic parameters

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Abstract

Aim: The aim of the present study was to evaluate P-wave duration and P dispersion (Pd) in obese subjects, and to investigate the relationship between P-wave measurements, and the clinical and echocardiographic variables.

Material & methods: The study population consisted of 52 obese and 30 normal weight control subjects. P-wave duration and P-wave dispersion were calculated on the 12-lead ECG. As echocardiographic variables, left atrial diameter (LAD), left ventricular end-diastolic, and end-systolic diameters (LVDD and LVSD), left ventricular ejection fraction (LVEF), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), and left ventricular mass (LVM) of the obese and the control subjects were measured by means of transthoracic echocardiography.

Results: Clinical and echocardiographic characteristics of 60 obese and 35 normal weight subjects were listed. Mean BMI for the obese and control groups were 35.5 ± 5.3 and 24.6 ± 1.6 ($P < 0.001$). Total cholesterol and LDL cholesterol levels, and triglycerides showed a trend toward higher values in the obese patients, but these values between both groups did not differ statistically. There were statistically significant differences between the obese and the control groups as regards to left atrial diameter (LAD), and left ventricular diastolic diameters (LVDD), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), and LVM ($P < 0.001$; for all parameters). Statistically significant differences were found in the values of Pmax and Pd between the obese and the control groups (109.7 ± 8.4 ms vs 102.4 ± 8.2 ms; $P < 0.001$ and 26.4 ± 5.0 ms vs 17.0 ± 4.3 ms; $P <0.001$). However, Pmin did not show any difference in obese patients compared to the controls (83.7 ±8.8 ms vs 83.6 ± 7.3 ms; $P > 0.05$; respectively).

Conclusion: In conclusion, we found that Pd values are elevated in obese patients and these increases in Pd were also correlated positively with BMI, LAD, LVDD, IVST, LVPWT, and LVM in obese patients. More importantly, increased Pd values in obese patients are closely associated with all of these parameters such as the clinical and echocardiographic parameters BMI, LAD, IVST, LVPWT and LVM.

Keywords: Obesity, BMI, ECG, P-wave, P-wave dispersion, atrial fibrillation

Introduction

Obesity is defined as a disease process in which excess body fat has accumulated to an extent that health may be adversely affected. According to WHO classification of body mass index (BMI) a person whose BMI is more than or equal to 30 Kg/m2 is obese and when BMI is between 18.5 to 24.99 then the person is considered normal [1]. Obesity is the first wave of a defined cluster of non-communicable diseases called 'New World Syndrome's creating an enormous socioeconomic and public health burden [2].

Obesity is one of independent risk factors for development of cardiovascular diseases, including essential hypertension and myocardial ischemia [3, 4, 5], and is also associated with sleep apnea syndrome [6] and insulin resistance [7]. It is well known that obesity is associated with left atrial enlargement and left ventricular filling abnormalities [8], both known predictor for atrial fibrillation. Age by itself is associated with increased AF frequency. P-wave dispersion (PD) is one of the essential non-invasive ECG markers for assessment of AF development [9, 10, 11, 12]. Compared to normal weight subjects; PD has been shown to be prolonged in obese subjects [13]. AF is more frequent, and PD is prolonged in patients with hypertension, diabetes and older age, and PD is prolonged.

P-wave dispersion (Pd) is defined as the difference between the maximum and the minimum P-wave duration in 12-lead surface electrocardiograms. Pd is considered to reflect the discontinuous and inhomogeneous propagation of sinus impulses and the prolongation of atrial conduction time. Increased

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Pd and maximum P-wave duration predict the development of AF in patients with various heart diseases [13]. However, P-wave alterations occurring in obese subjects have not been documented well in the literature.

Hence in the present study the aim was to evaluate the effects of obesity on P-wave duration and P-wave dispersion, and investigated possible relationship between P-wave measurements and the clinical and echocardiographic parameters.

Material & Methods

The study population consisted of 60 obese and 35 normal weight control subjects. P-wave duration and P-wave dispersion were calculated on the 12-lead ECG. As echocardiographic variables, left atrial diameter (LAD), left ventricular end-diastolic and end-systolic diameters (LVDD and LVSD), left ventricular ejection fraction (LVEF), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), and left ventricular mass (LVM) of the obese and the control subjects were measured by means of transthoracic echocardiography.

The obese patients and the controls were selected from subjects who underwent coronary angiography with a suspicion of coronary artery disease in our hospital. The indication of coronary angiography was either the presence of typical angina or positive non-invasive screening tests for myocardial ischemia in obese and the control groups. All of the obese patients and controls were subjects with angiographically proven normal epicardial coronary arteries. Local ethics committee approved this study, and informed consent was obtained from all participants.

BMI was calculated by dividing the body weight in kilograms by the square of height in meters (nor- mal defined as <25.0 and obesity >30.0). Patients with a history or clinical evidence of bundle-branch block, atrial flutter or fibrillation, hypothalamic or pituitary disease, depression, pregnancy, or hepatic, renal or thyroid diseases were excluded. None of the subjects was taking medication known to affect electrocardiographic intervals, and there were no electrolyte abnormalities.

Blood pressure was measured using a mercury sphygmomanometer in a sitting position after a 10-minute rest period, with the mean of three determinations being recorded; diastolic pressure was measured at the fifth Korotkoff sound. For the obese subjects, an arm cuff of appropriate size was used. Hypertension was defined as systolic blood pressure 140 mmHg, a diastolic blood pressure 90 mmHg, or self-reported use of an antihypertensive drug. Diabetes mellitus was defined as the use of antidiabetic medication and/or a fasting serum glucose level 126 mg/dl. Hypertensive patients did not discontinue antihypertensive treatment before measured P-wave intervals on electrocardiogram (ECG). Information on smoking habits and drug use were recorded for the study population.

Using standard laboratory methods, blood samples were drawn after an overnight 12-hour fasting to determine levels of blood glucose, electrolytes (Na, K, and Ca), and total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipopro- tein (LDL) cholesterol, and triglycerides.

P-Wave Measurements in 12 Lead ECGs

All standard 12-lead ECGs were obtained simultaneously using a recorder set at 50 mm/s paper speed and 2mV/cm standardization in a comfort- able supine position. For standardization, ECG was taken between 10 and 11 a.m. During ECG recordings all patients breathed freely and did not speak. ECGs were numbered and presented to the analyzing investigators without name and date information. P-wave duration was measured manually in all simultaneously recorded 12 leads of the surface ECG. Same observer measured all measurements of P-wave duration blindly in order to exclude interobserver variability. For greater accuracy, measurements were performed with calipers and magnifying lens, as described by previous investigators. Patients with measurable P wave in nine or fewer ECG leads were excluded from the study. The onset of the P wave was defined as point of first visible upward departure from baseline for positive waveform, and as the point of first down-ward departure from baseline for negative wave- forms. The return to the baseline was considered to be end of the P wave. The difference between the Pmax and the Pmin was calculated and defined as Pd.

Echocardiographic analysis

All study subjects underwent standard rest two dimensional echocardiography in the left lateral de-cubitis position. Parasternal long and short-axis, apical two and four-chamber views were obtained with 2.5 MHz transducer interfaced to ATL system (HDI 5000) ultrasound equipment. To determine LA dimension, the maximal dimension was measured between the leading edge of the posterior aortic wall to the leading edge of the posterior wall of the left atrium at end-systole. LV internal diameters at end-diastole and end-systole and wall thicknesses at end-diastole were measured by M-mode echocardiography according to the recommendation of the American Society of Echocardiography.23 In addition, left ventricular mass (LVM) was estimated from M-mode dimensions of septal thickness, posterior wall thickness, and left ventricular internal dimensions at end- diastole. LVM was calculated according to the regression equation: 0 [(1.04 (LVDD IVST LVPWT)3-(LVDD)3] 0.6 gm. All echocardiographic data were analyzed by one of the authors who were blind to subjects' past histories.

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Statistical analysis

All data were expressed as mean SD. The student's t-test was used to determine if significant differences in mean values for specific continuous variables existed between obese patients and normal weight controls. Pearson correlation analysis was used for estimating the relationship between test parameters. A P value <0.05 was considered statistically significant.

Results

Table 1: Clinical and Echocardiographic Characteristics of Obese and the Control Subjects

Variables	Obese N=60	Controls N=35	P Value
Gender Female %	52 (86.66)	28 (80)	0.956
Age in years	54 ±6	51 ± 15	0.860
BMI (kg/m2)	35.5 ± 5.3	24.6 ± 1.6	<0.001
Smoking (%)	13 (21.66)	8 (22.85)	0.785
HT (%)	19 (31.66)	10 (28.57)	0.983
DM (%)	15 (25)	9 (25.71)	0.647
Total cholesterol (mg/dl)	222 ± 28	216 ± 20	0.569
LDL cholesterol (mg/dl)	140 ± 16	138 ± 12	0.450
HDL cholesterol (mg/dl)	34 ±6	37 ±5	0.497
Triglycerides (mg/dl)	248 ± 32	238 ± 32	1.240
LAD (cm)	3.5 ± 1.8	3.3 ± 1.4	<0.001
LVDD (cm)	5.5 ± 1.7	5.1 ± 1.3	<0.001
LVSD (cm)	2.6 ± 0.9	2.4 ± 0.7	1.604
LVEF (%)	53	54	1.704
IVST (cm)	1.0 ± 0.1	0.80 ± 0.09	<0.001
LVPWT (cm)	1.0 ± 0.1	0.75 ± 0.07	<0.001
LVM (gm)	218 ± 28	196 ± 21	<0.001

Clinical and echocardiographic characteristics of 60 obese and 35 normal weight subjects were listed. Mean BMI for the obese and control groups were 35.5 ± 5.3 and 24.6 ± 1.6 ($P < 0.001$). Total cholesterol and LDL cholesterol levels, and triglycerides showed a trend toward higher values in the obese patients, but these values between both groups did not differ statistically. In addition, the obese patients did not differ from the controls with regard to age, gender, and smoking and the percentage of hypertension and diabetes mellitus and HDL cholesterol and electrolytes levels. There were statistically significant differences between the obese and the control groups as regards to left atrial diameter (LAD), and left ventricular diastolic diameters (LVDD), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), and LVM ($P < 0.001$; for all parameters).

Table 2: Comparison of P-Wave Measurements in Obese and the Control Subjects

Variables	Obese N=60	Controls N=35	P Value
Pmax (ms)	109.7 ± 8.4	102.4 ± 8.2	< 0.001
Pmin (ms)	83.7 ± 8.8	83.6 ± 7.3	0.450
Pd (ms)	26.4 ± 5.0	17.0 ± 4.3	< 0.001

Statistically significant differences were found in the values of Pmax and Pd between the obese and the control groups (109.7 ± 8.4 ms vs 102.4 ± 8.2 ms; $P < 0.001$ and 26.4 ± 5.0 ms vs 17.0 ± 4.3 ms; $P < 0.001$). However, Pmin did not show any difference in obese patients compared to the controls (83.7 ±8.8 ms vs 83.6 ± 7.3 ms; $P > 0.05$; respectively).

Discussion

Obesity is associated with the developmental of cardiovascular diseases (CVDs) as an uncontrolled risk factor with a gradually increasing frequency due to the increase of atrial fibrillation (AF) in obese subjects [16]. However, the potential mechanisms that increase AF risks are not precise. Obesity-related conditions like hypertension, left ventricular hypertrophy, sleep apnea and left atrial enlargement are potential risk factors for AF development [17, 18]. Age by itself is associated with increased AF frequency. It has a strong impact on cardiovascular changes which is manifested in electrocardiogram (ECG) [19]. Currently it is a serious public health problem with established cardiovascular co-morbidities and a major cause of sudden death in developed as well as developing countries [20]. According to the National Family Health Survey-4 (NFHS-4) in 2015-16 conducted by Ministry of Health and Family Welfare (MOHFW) in India, the percentage of men and women aged 15-49 years who are obese are 19% and 21% respectively [21]. In a large prospective study 'Framingham Heart study' there is evidence for inclusion of obesity as a major modifiable cardiovascular risk factor by American Heart Association and also sudden cardiac death has been reported 40 times higher in obese men and women [22].

Clinical and echocardiographic characteristics of 60 obese and 35 normal weight subjects were listed.

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Mean BMI for the obese and control groups were 35.5 ± 5.3 and 24.6 ± 1.6 (P < 0.001). Total cholesterol and LDL cholesterol levels, and triglycerides showed a trend toward higher values in the obese patients, but these values between both groups did not differ statistically. In addition, the obese patients did not differ from the controls with regard to age, gender and smoking and the percentage of hypertension and diabetes mellitus and HDL cholesterol and electrolytes levels. There were statistically significant differences between the obese and the control groups as regards to left atrial diameter (LAD), and left ventricular diastolic diameters (LVDD), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), and LVM (P < 0.001; for all parameters). P-wave duration and Pd are the most important non-invasive ECG markers that have been introduced to assess atrial arrhythmias risk of patients. Pd has been proposed as being useful for the prediction of AF.[23-26] It has been known that increased P duration and dispersion is also associated with atrial conduction prolongation, left atrial enlargement, and left atrial hypertension. Additionally, the autonomic tone, which induces changes in the velocity of impulse propagation, affects P intervals.

Statistically significant differences were found in the values of Pmax and Pd between the obese and the control groups (109.7 ± 8.4 ms vs 102.4 ± 8.2 ms; P < 0.001 and 26.4 ± 5.0 ms vs 17.0 ± 4.3 ms; P <0.001). However, Pmin did not show any difference in obese patients compared to the controls (83.7 ±8.8 ms vs 83.6 ± 7.3 ms; P > 0.05; respectively). It is well known that patients with obesity have a higher prevalence for hypertension, which may lead to left ventricular hypertrophy and left atrial enlargement that may play a role in alteration of P-wave measurements. In addition, the autonomic control of the heart is abnormal in obese subjects due to a prevalence of sympathetic over parasympathetic limb of the autonomic balance. Therefore, the autonomic imbalance observed in obese subjects may affect intraatrial and interatrial conduction times, and leave them prone to develop atrial arrhythmias, such as atrial fibrillation. Because of these reasons, obese individuals may have an increased risk for atrial fibrillation. Recently, Seyfeli *et al*. 22 showed that obesity caused important increase in P wave and QTc values and also they may under the risk atrial arrhythmias.

Conclusion

In conclusion, we found that Pd values are elevated in obese patients and these increases in Pd were also correlated positively with BMI, LAD, LVDD, IVST, LVPWT and LVM in obese patients. More importantly, increased Pd values in obese patients are closely associated with all of these parameters such as the clinical and echocardiographic parameters BMI, LAD, IVST, LVPWT and LVM. Accordingly, these results support the hypothesis that obesity is associated with increased risk for atrial fibrillation and that obesity contributes to the development of AF. The limitations of our study, the study included a small number of patients in a selected population. Further studies will be necessary.

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