ORIGINAL ARTICLES

P-Wave Duration and Dispersion in Obese Subjects

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Background: Although previous studies have documented a variety of electrocardiogram (ECG) abnormalities in obesity, P-wave alterations, which represent an increased risk for atrial arrhythmia, have not been studied very well in these patients. 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.

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: There were statistically significant differences between obese and controls as regards to Pmax (maximum P-wave duration) and Pd (P dispersion) ($P < 0.001$ and $P < 0.001$, respectively). Pmin (minimum P wave duration) was similar in both groups. Correlation analysis showed that Pd in the obese patients was related to any the clinical and echocardiographic parameters including BMI, LAD, LVDD, IVST, LVPWT, and LVM.

Conclusion: Our data suggest that obesity affects P-wave dispersion and duration, and changes in P dispersion may be closely related to the clinical and the echocardiographic parameters such as BMI, LAD, IVST, LVPWT, and LVM. **A.N.E. 2008;13(1):3–7**

ECG; P-wave duration; P-wave dispersion; obesity

INTRODUCTION

Obesity is one of independent risk factors for development of cardiovascular diseases, including essential hypertension and myocardial ischemia,[1–3] and is also associated with sleep apnea syndrome[4] and insulin resistance.[5] It is well known that obesity is associated with left atrial enlargement and left ventricular filling abnormalities,[6–8] both known predictor for atrial fibrillation. Several studies suggested that obese subjects are associated with impaired heart rate variability.[9–11] Therefore, obese patients may have high risk for atrial fibrillation. Alterations in QT and RR intervals and T wave occurring in obesity[12–15] and the effect of weight loss on QT intervals[16,17] in this condition have been relatively well defined.

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 Pd and maximum P-wave duration predict the development of AF in patients with various heart diseases.[18–22] However, P-wave alterations occurring in obese subjects have not been documented well in the literature. In the present

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study we evaluated 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.

MATERIALS AND METHODS

Patient Population

This study included 52 obese patients (mean age 53 ± 9 years, mean body mass index (BMI, the weight in kilograms divided by the height in meters squared [kg/m^2]) of 38.1 ± 5.8; range 32 to 45) and thirty normal weight control subjects (mean age 50 ± 12 years, BMI of 22.5 ± 1.6; range 22–24). The obese patients and the controls were selected from subjects who underwent coronary angiography with a suspicion of coronary artery disease in our hospital from February 2005 to September 2006. 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 (normal 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 lipoprotein (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 comfortable 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 downward departure from baseline for negative waveforms. 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 decubitis 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: $0.8 \times [(1.04 \times (LVDD + IVST + LVPWT)^3\text{-}(LVDD)^3] + 0.6$ gm. All echocardiographic data were analyzed by one of the authors who were blind to subjects' past histories.

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

Clinical and echocardiographic characteristics of 52 obese and 30 normal weight subjects are listed in Table 1. Mean BMI for the obese and control groups were 38.1 ± 5.8 and 22.5 ± 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 measures are shown in Table 2. Statistically significant differences were found in the values of Pmax and Pd between the obese and the control groups (110.9 ± 8.7 ms vs 101.2 ± 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 (83.6 ± 8.9 ms vs 83.2 ± 7.8 ms; P > 0.05; respectively).

Pearson's correlation analysis showed that there were positive correlations between Pd, and BMI (r = 0.504, P < 0.001), LAD (r = 0.466, P < 0.001), LVDD (r = 0.419, P = 0.002), IVST (r = 0.344, P = 0.012), LVPWT (r = 0.288, P = 0.038), LVM (r = 0.355, P < 0.01) in obese group (Table 3).

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

Variables	Obese n = 52	Controls n = 30	P value
Age (yrs)	53 ± 9	50 ± 12	NS
Gender (female %)	45 (86.5)	24 (80)	NS
BMI (kg/m^2)	38.1 ± 5.8	22.5 ± 1.6	**<0.001**
Smoking (%)	12 (23)	7(23)	NS
HT (%)	17 (32.6)	9 (30)	NS
DM (%)	13 (25)	7 (23)	NS
Total cholesterol (mg/dl)	224 ± 27	218 ± 21	NS
LDL cholesterol (mg/dl)	142 ± 15	137 ± 12	NS
HDL cholesterol (mg/dl)	35 ± 5	37 ± 5	NS
Triglycerides (mg/dl)	245 ± 34	238 ± 32	NS
LAD (cm)	3.6 ± 1.8	3.3 ± 1.4	**<0.001**
LVDD (cm)	5.4 ± 1.7	5.1 ± 1.3	**<0.001**
LVSD (cm)	2.6 ± 0.9	2.4 ± 0.7	NS
LVEF (%)	52	53	NS
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**

NS = not significant; HT = hypertension; DM = diabetes mellitus; BMI = body mass index; LAD = left atrial diameter; LVDD = left ventricular diastolic diameter; LVSD = left ventricular systolic diameter; LVEF = left ventricular ejection fraction; IVST = interventricular septum thickness; LVPWT = left ventricular posterior wall thickness; and LVM = left ventricular mass.

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

Variables	Obese	Controls	P value
Pmax (ms)	110.9 ± 8.7	101.2 ± 8.2	P < 0.001
Pmin (ms)	83.6 ± 8.9	83.2 ± 7.8	NS
Pd (ms)	26.4 ± 5.0	17.0 ± 4.3	P < 0.001

NS = not significant.

DISCUSSION

It has been previously shown that obese subjects may be associated with electrocardiographic abnormalities even in the absence of clinical symptoms. Studies of obese people have identified T-wave abnormalities, leftward axis deviation, and low QRS voltage on the ECG as the most frequent changes.[12,13] Some studies have also suggested that obesity increases duration of QT interval and QT dispersion,[14,15] and that even prolonged QT intervals in obese patients return to normal range after weight loss.[16,17] Recently, Wang et al.[18] showed that obesity may an important modifiable risk factor for AF. However, little information exists concerning P-wave duration and dispersion in obese subjects. This study is the first comprehensive survey of values of P-wave duration and Pd in obese subjects.

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.[18–21] 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.

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.

In the present study, in order to minimize the effects of other disease or factors which could influence P-wave measurement, patients with metabolic and electrolyte abnormalities, the associated cardiac and pulmonary diseases were excluded from the study. More importantly, cardiovascular risk factors including diabetes mellitus, hypertension, smoking, and hyperlipemia were similar between both the groups. In addition to these considerations, age and gender were homogeneously distributed in two groups. They were also on medical therapy. Because some medications may affect ECG intervals by changing heart rate or alter the characteristics of repolarization.

Our study demonstrated that values of P-wave measures in obese subjects without complications were higher than those of age-matched normal weight controls, which is consistent with the

Table 3. Correlation Between P Dispersion and the Clinical and the Echocardiographic Parameters in Obese Subjects

Variables	BMI	LAD	LVDD	IVST	LVPWT	LVM
Pd	r = 0.504, P < 0.001	r = 0.466, P < 0.001	r = 0.419, P = 0.002	r = 0.344, P = 0.012	r = 0.288, P = 0.038	r = 0.355, P < 0.01

BMI = body mass index; LAD = left atrial diameter; LVDD = left ventricular diastolic diameter; LVSD = left ventricular systolic diameter; LVEF = left ventricular ejection fraction; IVST = interventricular septum thickness; LVPWT = left ventricular posterior wall thickness; and LVM = left ventricular mass.

findings of previous studies.[12,13,18–21,22] Also, there were positive correlations between Pd, and the clinical and echocardiographic variables including BMI, LAD, LVDD, IVST, LVPWT, and LVM in obese subjects. In other words, Pd values were increased in parallel with the clinical and echocardiographic variables stated above. Based on these results, we have suggested that obesity has the important effects on P-wave duration and dispersion, and even on echocardiographic variables.

Previous studies[24–28] demonstrated that P-wave dispersion had predictive value for atrial fibrillation in patients with hypertensive, heart failure, and diabetes mellitus. Similarly, our findings showed that obese patients had high Pd values, increasing the risk of atrial fibrillation. It is therefore possible to say that obesity is an important risk factor for atrial fibrillation.

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 to validate our data.

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