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Original Article

The relationship between left ventricular mass and insulin resistance in obese patients☆

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ABSTRACT

Objective: In this study, we investigated the relationship between left ventricular mass and insulin resistance in obese patients.

Methods: A total of 90 subjects, 66 women, and 24 men, with an age range from 24 to 56 years, were enrolled in the study. Forty-nine patients were in the obesity group whose body mass index (BMI) was >29.9 kg/m^2 and 41 subjects were in the control group with a BMI <25 kg/m^2. All of them were normotensive, nondiabetic, and did not have any cardiovascular disease. They were not taking any medication. Weight, height, and waist circumference were measured and BMI was calculated. Plasma glucose, insulin, serum total, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol, and triglyceride levels were measured, and insulin resistance was calculated via homeostasis model of assessment-estimated insulin resistance (HOMA-IR). Subjects were examined by echocardiography and left ventricular mass (LVM) and index (LVMI) were calculated with Devereux formula.

Results: Insulin levels, HOMA-IR, LVM, and LVMI were significantly higher in obesity group ($p < 0.01$). Fasting glucose, triglyceride, fasting insulin levels, and waist circumference did not correlate with LVMI.

Conclusion: In conclusion, though findings of the present study suggest increased left ventricular hypertrophy (LVH) in obese subjects compared to controls, it appears that the increased LVM or LVH is not linked to BMI and insulin resistance in this study population.

1. Introduction

Cardiovascular disease (CVD) constitutes one of the major causes of deaths and disabilities, globally claiming 17.3 million lives a year. Incidence of CVD is expected to rise to 25 million by 2030.[1] In 2008, 30% of all global deaths is attributed to CVD. Death caused by cardiovascular diseases is also higher in low- and middle-income countries, as over 80% of all global deaths caused by cardiovascular diseases occurred in those countries.[2–4] Changes in the left ventricular structure increase the risk of cardiovascular diseases and death.[5] The risk of acute myocardial infarction, congestive heart failure, sudden death, ventricular ectopy, serious arrhythmias, and other cardiovascular events increases sixfold to eightfold with the occurrence of left ventricular hypertrophy (LVH).[6,7]

☆ This work was carried out at the Okmeydani Training and Research Hospital, Istanbul, Turkey.

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LVH, which leads to increase in left ventricular mass (LVM), poses a serious risk for an increase in cardiovascular diseases and deaths caused by them.[8] LVH can be prevented by eliminating the factors leading to increased LVM, but the nature of the factors that cause an increase in LVM is yet to be completely understood among normotensive and even among hypertensive individuals.[9,10] A prior study reports that no change in the LVM was observed in 25–30% of individuals with high blood pressure. These observations support the views that there are other metabolic and genetic factors effective in cardiac mass increase.[11]

Obese patients have persistent myocardial wall stress because of an increase in circulatory volume and minute volume.[12] There are studies suggesting a relationship between obesity and cardiac mass increase as well.[13,14] Duration of obesity is significantly correlated with LVH.[15] In vivo studies have shown that insulin resistance (IR) and hyperinsulinemia have effects on LVM.[16,17] Additionally, in vitro studies have shown that with the IR-associated hyperinsulinemia impact, an increase is observed in sympathetic stimulation, peripheral vascular resistance, renal sodium retention, cardiac workload, and the anabolic effect on cardiac proteins.[17,18] Some studies have determined an independent relationship between insulin level and cardiac mass.[18,19] However, it has not been completely proven that IR is an independent indicator of the LVM increase.[20]

According to the TEKHARF and the TURDEP I studies conducted in Turkey, prevalence of obesity was 28% and 32%, respectively.[21,22] TURDEP II study reported that obesity in Turkey increased by 40% compared to that reported in 1998, and that overweight and obese patients constitute 2/3 of the Turkish population.[22] Therefore, the aim of our study was to investigate the relationship between obesity, a significant health issue in Turkey, and IR, hyperinsulinemia, blood lipid level disorders, and LVM, which are commonly associated with obesity.

2. Materials and methods

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from each subject following a detailed explanation of the protocol of the study. All study procedures were conducted in accordance with the ethical principles stated in the ''Declaration of Helsinki''.

The study included 49 obese patients whose body mass indices (BMI) were equal to or greater than 30 kg/m^2 and 41 healthy subjects with BMI < 25 kg/m^2. The participants included in the study did not have history of hypertension, diabetes (familial or self), or drug therapy. Patients with blood pressure over 140/90 mmHg[23] and fasting plasma glucose level over 100 mg/dl were excluded from the study. The thyroid function tests, urea, and creatinine levels of the patients were within the normal range. There was no history of smoking in either patient or control groups. Atrial fibrillation, presence of intraventricular electrodes, ischemic heart disease, hypertrophic cardiomyopathy, congenital heart disease, valve prostheses, chemotherapy, pericarditis, intracardiac masses or thrombi, moderate or severe valvular stenosis or regurgitation, cor pulmonale, pulmonary thromboembolism, or heart failure were excluded from study.

Age and gender information of all individuals were recorded. Weight measurements were made with thin clothing and no shoes on the same scale (Arzum Peso Model AR535, China, 2008). Height measurements were made bare foot. BMI was calculated with the weight (kg)/height^2 (m) formula. Waist circumference (WC) was measured with a measuring tape at the level of the umbilicus. Blood pressure of the patients was measured from both arms after a rest of at least 15 min using a sphygmomanometer (Erka).

Glucose, total, very-low-density lipoprotein (VLDL) and high-density lipoprotein (HDL)-cholesterol, triglyceride, and insulin levels of the patients were measured in the venous blood sample drawn after 12-h fasting by a clinical biochemistry otoanalyzer (Olympus AU2700). Low-density lipoprotein (LDL)-cholesterol value was calculated using the Fridewalt formula.[24]

Insulin levels were measured via chemiluminescence method via Beckman Coulter DXİ 800 (Miami, USA 2006) device in the serum sample obtained from venous blood after 10 min of centrifugation at 3000 rpm. In each subject, the degree of IR was estimated at the baseline by homeostasis model of assessment (HOMA) method described by Matthews et al.[25] In particular, a homeostasis model of assessment-estimated insulin resistance (HOMA-IR) score was computed with the formula of ''fasting plasma glucose (mmol/L) $\times$ fasting serum insulin (mU/L) divided by 22.59''.[25]

All of the patient and control group participants received echocardiography (GE Vivid 3 pro, Indiana, USA). In case of 10 or more cycles, left ventricular diastolic diameters and thickness of posterior wall and septum in diastole in 2D parasternal long-axis position were measured by using 2.5 MHerz probes in the left lateral decubitus position.

LVM was calculated via Devereux formula.[26] LVM index (LVMI) was calculated as LVM divided by the body surface area. Males with LVMI >134 g/m^2 and females with LVMI >110 g/m^2 were considered to have LVH.[26]

Statistical analyses were conducted using the NCSS (Number Cruncher Statistical System) 2007 and PASS (Power Analysis and Sample Size) 2008 statistical software packages (UTAH, USA). Unpaired t test and Mann–Whitney U test was used for comparisons based on the distribution pattern of the numerical data. Chi-square test was used to compare categorical data. Associations between the parameters were evaluated with the Pearson and Spearman's correlation analyses where appropriate. The results were evaluated as significant when $p < 0.05$ within a 95% confidence interval.

3. Results

3.1. *Baseline characteristics*

The mean age of the obese group was 36.9 $\pm$ 7.61 years (range 25–56 years) and of the control group was 34.17 $\pm$ 7.77 years (range 24–56 years), with no significant difference between groups ($p = 0.097$). There were 40 (81.6%) females and 9 (18.4%) males in the obese group; and 26 (63.4%) females and 15 (36.6%) males in the control group. The groups did not differ significantly in terms of gender distribution ($p = 0.052$).