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Original Article

Relationship of Left Ventricular Mass with BMI and Insulin Resistance in Normotensive Obese Women

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Abstract

Objective: Obesity is correlated with left ventricular mass (LVM) and insulin resistance (IR) and is an independent predictor of LVM in nondiabetic, nonhypertensive, obese people. Our aim was to investigate the relationship of body mass index (BMI) and IR with left venticular mass index (LVMI) in obese and nonobese, normotensive, nondiabetic women.

Methods: 81 obese, normotensive, nondiabetic women and 36 healthy women of normal weight were included in the study. We compared the demographic features, biochemical values, insulin and HOMA-IR values, heart rate, blood pressure, and echocardiographic parameters of the obese and nonobese subjects.

Results: The mean age was 39.3±11.2 years and the mean BMI was 39.5±5.7 kg/m^2 in the obese group; the mean age was 38.4±9.5 years and the mean BMI was 22.5±1.9 kg/m^2 in the control group. Hyperinsulinemia (19.3±10.4 μU/mL) and IR (HOMA-IR: 4.6±2.9) were correlated with obesity. Insulin levels and IR were associated with LWM, LWMI, aortic diameter (AD), left atrial diameter (LAD), interventricular wall thickness (IVWT), left ventricular end diastolic diameter (IVEDD), and left ventricular posterior wall thickness (LVPWT). Age, insulin value, and BMI were determinants of LVMI ($p<0.001$ for age and BMI, $p=0.003$ for insulin). IR was negatively correlated with left ventricular systolic function. AD, LAD, IVWT, LVEDD, and LVPWT were higher in the obese subjects than in the controls.

Conclusion: Although left ventricular structural abnormalities were found, we did not observe diastolic dysfunction. Although systolic and diastolic functions were within normal limits, both functions were impaired in the obese group compared to normal subjects. This finding may indicate myocardial involvement in obesity. We observed that obesity is associated with LVM independently from hypertension and diabetes mellitus.

Keywords: Obesity, left ventricular mass, insulin resistance

Introduction

The relationship between excess body weight and cardiovascular disease has been well known for years (1-5). Increasing body mass requires increased cardiac debi and vascular volume to supply metabolic demands (3, 6, 7). This may be partly due to the altered metabolic needs induced by increased body weight (8).

Left ventricular hypertrophy (LVH) may cause diastolic and systolic heart failure; it is also an independent risk factor for cardiac arrhythmia, sudden death, myocardial ischemia, and heart failure (9). LVH in obese people is related to the severity of comorbidities, mainly hypertension. Although obesity itself is an independent risk factor for LVH in hypertensive patients, the roles of excess body weight and obesity in LVH in normotensive subjects are inconclusive (5, 10). In this article, we investigated the relationships of obesity and insulin resistance with LVH and diastolic function in nondiabetic, normotensive women who were followed up at our obesity outpatient clinic; the results were compared with those of normal women.

Methods

This study was conducted at our hospital with 81 obese, nondiabetic, normotensive female subjects (study group) and 36 healthy female controls (control group). All participants were informed about the survey and freely signed and dated the consent form. The protocol was approved by the Local Ethics Committee and was conducted in accordance with the Declaration of Helsinki.

Patients were excluded if they had a history of ischemic heart disease, congestive heart failure, cardiac valve disease, liver or kidney disease, hypertension, cancer, adrenal disease (such as Conn's disease, pheochromocytoma, or Cushing's syndrome, which can cause secondary hypertension), diabetes mellitus, thyroid disease, glucose intolerance, malnutrition, or malabsorption.

The age, weight, height, body mass index (BMI), waist circumference (WC), systolic and diastolic blood pressure, and heart rate of the subjects were recorded. Serum fasting blood glucose (FBG),

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total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), and triglyceride (TG) levels were measured enzymatically. High density lipoprotein cholesterol (HDL-C) was measured by a direct enzymatic method. Serum insulin levels were measured by the immunochemiluminescence method (Roche Diagnostics, GmbH, Mannheim, Germany) with an E-170 autoanalyzer.

The subjects' heights were measured without shoes (cm), and their weights were measured with a standard scale (kg). For blood pressure, the values were obtained on the right arm from the mean value of two measurements using a calibrated device after resting for 10 minutes. BMI was calculated as the ratio of weight (kg) to height squared (m^2). Insulin resistance was calculated using the homeostasis model assessment of insulin resistance (HOMA-IR) formula. Subjects with HOMA-IR levels higher than 2.4 were accepted as having insulin resistance. Standard electrocardiographic examinations were performed using a Vingmed Vivid System 3 device (General Electric, Fairfield, Connecticut, USA). A 2.5 MHz probe was used for Doppler measurements. Echocardiographic evaluations were performed by the same cardiologist using the same echocardiographic machine. All measurements represent averages taken from three cardiac cycles. LV dimensions and wall thicknesses were obtained from the parasternal long axis with an M-mode cursor positioned just beyond the mitral leaflet tips and perpendicular to the long axis of the ventricle. The LV end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD), the thickness of the interventricular septum (IVS), and the posterior wall of the left ventricle (PW) were measured. The LV ejection fraction (EF) was calculated according to the Simpson method. Mitral inflow velocities were evaluated by pulse-wave Doppler with the sample volume placed at the tip of the mitral leaflets from an apical 4 chamber view. Using the average of three beats, we measured diastolic peak early (E) and peak late (A) transmitral flow velocities, peak E to peak A velocities (E/A), and the deceleration time of the peak E velocity (EDT). Isovolumic contraction time (ICT), isovolumic relaxation time (IVRT), and ejection time (ET) were also measured.

We used the Devereux and Reichek formula for detecting left ventricular mass index (LVMI), as shown below (11).

LVMI=0.8x1.04([LVEDD+LVPWT+IVWT]3-[LVEDD]3)+0.6
LVMI: Left ventricular mass index
IVWT: Interventricular wall thickness
LVEDD: Left ventricular end diastolic diameter
LVPWT: Left ventricular posterior wall thickness

Statistical analysis
For statistical analysis, Statistical Package for Social Sciences 15.0 for Windows (SPSS Inc.; Chicago, IL, USA) was used. Descriptive statistics for categorical variables were indicated as a number or proportion, and means and standard deviations were used for numeric variables. Comparisons were made using the Student's t-test and the Mann-Whitney U test. Pearson's correlation test, the Spearman correlation test, and linear regression analyses were also used. Significance was accepted as $p<0.05$.

Results

A comparison of the demographic, anthropomorphic, and biochemical findings is shown in Table 1. As expected, BMI and WC were higher in the obese group compared to the age-matched control subjects. The FBG, insulin, and HOMA-IR values were also higher in the obese subjects.

Table 1. Comparison of demographic characteristics and laboratory parameters between the obese and control groups

Variables	Obese Group	Control Group	p
Age (years)	39.3±11.2	38.4±9.5	0.924
Body mass index (kg/m^2)	39.5±5.7	22.5±1.9	<0.001
Waist circumference (cm)	114.3±12.0	71.9±6.4	<0.001
Heart rate (beats/min)	81.2±8.5	73.4±9.0	<0.001
Systolic blood pressure (mmHg)	109.0±11	111.67±7.4	0.193
Diastolic blood pressure (mmHg)	74.0±6.2	66.2±6.7	<0.001
Fasting blood glucose (mg/dL)	95.4±12.9	83.0±9.9	<0.001
Serum insulin level (μU/mL)	19.3±10.4	6.5±3.1	<0.001
HOMA-IR	4.6±2.9	1.4±0.6	<0.001
Urea (mg/dL)	27.7±7.8	22.7±4.5	<0.001
Albumin (g/dL)	4.4±0.3	4.2±0.3	0.623
Triglycerides (mg/dL)	130.2±71.1	106.1±40.5	0.052
Creatinine (mg/dL)	0.7±0.1	1.1±0.1	0.622
Total cholesterol (mg/dL)	193.6±36.0	188.0±10.7	0.444
HDL cholesterol (mg/dL)	35.9±2.4	55.5±4.5	0.054
LDL cholesterol (mg/dL)	116.9±34.2	117.7±28.2	0.915

Echocardiographic findings
The left ventricular diameters, EFs, and aortic and left atrial diameters were different between the two groups (Table 1). Although the EF was within the normal range in both groups, it was significantly lower in the study group (60.7±2.2 vs. 66.3±4.3 percent, respectively, $p<0.001$).

Although LVM and LVMI were within normal limits, they were higher in the study group than in the control group (185.7±41.0 vs. 125.1±17.9 g, respectively, $p<0.001$; 87.4±20.8 vs. 75.4±12.5 g/m^2, respectively, $p<0.001$).

LAD, AD, IVWT, LVEDD, and LVPWT were higher in the obese subjects, as expected. LVWT was significantly higher in the obese group compared to the control group. Except for the E/A ratios (obese: 1.1±0.4 vs. controls: 1.3±0.3; $p=0.033$), all parameters were higher in the obese subjects than in the controls (Table 2). With regard to mitral Doppler flow measurements, mitral E and A were significantly higher in the obese group compared to the controls. Left ventricular diastolic functions, obtained with conventional mitral Doppler parameters, were preserved; however, the E/A ratio, which is a determinant of diastolic dysfunction, was significantly lower in the study group.

Correlation analysis
Correlation analyses of age, BMI, WC, fasting blood glucose, insulin, and HOMA-IR with conventional echocardiographic parameters and left ventricular mass indices are shown in Table 3.

Regression analysis
Regression analysis revealed that LVM and LVMI are predominantly determined by age, serum insulin, WC, and BMI (Table 4).

Discussion

The main findings of our research were the high LVM and LVMI in obese, nondiabetic, non-hypertensive women compared to normal subjects. LVM and LVMI were correlated with BMI and WC. LVMI was also correlated with insulin and HOMA-IR.

Obese people have higher cardiac debi with lower peripheral resistance (3). The increase in cardiac debi and blood volume activates the sympathetic nervous system and results in hypertension via several mechanisms (12, 13). Increased filling pressure and volume induce left ventricular dilatation; myocardial mass then increases and gives rise to eccentric left ventricular hypertrophy. Ongoing hemodynamic stress causes left ventricular diastolic and systolic dysfunction (1, 3, 7, 14, 15). Left ventricular filling is abnormal, peak atrial velocity is higher, and late diastolic flow rate is longer in the obese subjects. Left ventricular diastolic dysfunction and increased LVM may be related to collagen accumulation due to a volume overload in the left ventricle, myocyte hypertrophy, and interstitial fibrosis (16, 17). Altered FGF23 levels may also contribute to cardiac remodeling (18). The association of insulin resistance with obesity, mainly visceral obesity, is well known (19-22). Our research revealed similar correlations, as expected. Hyperinsulinemia in obese people stimulates anabolic effects and increases renal sodium absorption, causing increased blood volume and myocardial hypertrophy. Insulin resistance may also augment atherosclerosis and contribute to heart failure. Hyperinsulinemia causes myocardial hypertrophy and fibrosis, increasing the effects of angiotensin II (23, 24). Obesity also results in triglyceride accumulation in the myocardium (25). Our study population had higher FBG, insulin, and HOMA-IR values compared to the control subjects. Their left ventricular volumes were also significantly increased compared with the control subjects. These findings were in accordance with findings reported in the literature. We excluded hypertensive patients, as hypertension can directly affect LVM. The altered values of LVM and LVMI support the idea that left ventricular hypertrophy is induced by insulin resistance and high insulin levels. This is one of the

Table 2. Comparison of echocardiographic parameters between the obese and control groups

	Obese Mean±SD	Control Group	p
Left ventricular mass (g)	185.7±41.0	125.1±17.9	<0.001
Left ventricular mass index (g/m²)	87.4±20.8	75.4±12.5	<0.001
Mitral A (cm/min)	0.8±0.2	0.6±0.2	<0.001
Mitral E (cm/min)	0.9±0.2	0.8±0.2	0.001
Aortic diameter (mm)	29.4±3.4	25.1±3.2	<0.001
Left atrial diameter (mm)	34.1±3.0	29.4±2.6	<0.001
Left ventricular posterior wall thickness (mm)	10.4±1.4	9.2±0.7	<0.001
Interventricular septum thickness (mm)	10.6±1.3	9.1±1.3	<0.001
Left ventricular end-diastolic diameter (mm)	48.1±3.5	44.1±2.3	<0.001
Ejection fraction (%)	60.7±2.2	66.3±4.3	<0.001
E/A ratio*	1.1±0.4	1.3±0.3	0.033

*E/A ratio: early diastolic peak flow rate/atrial peak flow rate
SD: standard deviation

Tablo 3. Correlation analysis between age, body mass index, biochemical parameters, and echocardiographic parameters

		Age (years)	BMI (kg/m²)	Waist circum (cm)	Fasting blood glucose	Insulin levels	HOMA-IR
Left ventricular mass (g)	R	0.404	0.512	0.556	0.305	0.539	0.527
	p	<0.001	<0.001	<0.001	0.001	<0.001	<0.001
Left ventricular mass index (g/m²)	R	0.456	0.407	0.128	0.147	0.229	0.231
	p	<0.001	<0.001	0.172	0.115	0.013	0.012
Mitral A (cm/min)	R	0.439	0.427	0.461	0.342	0.390	0.418
	P	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mitral E (cm/min)	R	0.024	0.238	0.207	0.031	0.223	0.223
	p	0.801	0.010	0.026	0.738	0.016	0.016
Aortic diameter (mm)	R	0.315	0.447	0.525	0.241	0409	0.407
	p	0.001	<0.001	<0.001	0.009	<0.001	<0.001
Left atrial diameter (mm)	R	0.299	0.600	0.639	0.285	0.416	0.402
	p	0.001	<0.001	<0.001	0.002	<0.001	<0.001
Left ventricular posterior wall thickness (mm)	R	0.401	0.283	0.290	0.212	0.389	0.389
	p	<0.001	0.002	0.002	0.022	<0.001	<0.001
Interventricular septum thickness (mm)	R	0.432	0.348	0.346	0.203	0.372	0.375
	p	<0.001	<0.001	<0.001	0.029	<0.001	<0.001
Left ventricular end diastolic diameter (mm)	R	0.188	0.466	0.533	0.203	0.410	0.379
	p	0.044	<0.001	<0.001	0.029	<0.001	<0.001
Ejection fraction (%)	R	-0.306	-0.497	-0.504	-0.253	-0.448	-0.447
	p	0.001	<0.001	<0.001	0.006	<0.001	<0.001
E/A ratio	R	-0.384	-0.157	-0.209	-0.222	-0.142	-0.142
	p	<0.001	0.092	0.025	0.017	0.128	0.129

Tablo 4. Regression analyses of parameters determining LVM and LVMI

	LVM			LVMI		
	B	Beta	p	B	Beta	p
Constant	33.231			48.465		
Age (years)	1.084	0.262	<0.001	0.746	0.420	<0.001
Serum insulin levels (μU/mL)	1.374	0.323	<0.001	0.455	0.249	0.003
Waist circumference (cm)	0.708	0.350	<0.001			
BMI (kg/m^2)	0.734	0.387	<0.001	0.654	0.345	<0.001

LVM: left ventricular mass; LVMI: left ventricular mass index

main outcomes of our study, which suggests that obesity may cause LVH independently, possibly before the development of hypertension. We did not follow up with our patients to determine if they had hypertension in the long term. This may be a limitation of this study. The association of HOMA-IR and insulin levels with LVM and LVMI also suggests that high insulin levels and insulin resistance are mainly responsible for increased LVM. Regression analysis also revealed that age, serum insulin levels, and BMI were factors determining LVMI. If our study population contained more subjects, we may have found that BMI was statistically significant.

The results of some studies conflict with our results. Galvan et al. (26) reported that insulin sensitivity was not a risk factor for LVM in nondiabetic subjects. Kibar et al. (27) investigated normotensive obese children echocardiographically and found a significant relationship between HOMA-IR and both LAD and LVEDD, although no correlation was found between BMI and LVMI.

A close association has been observed between LVH and left ventricular diastolic functions. While the existing knowledge about the effects of obesity on left ventricular diastolic functions is inconclusive, limitations in left ventricular filling may be a factor. Licata et al. (28) reported that left ventricular hypertrophy and diastolic dysfunction may be early determinants of cardiovascular disease. Chakko et al. (29) found an increase in the A wave velocity and a decrease in the E/A ratio; meanwhile, no difference was observed in the E wave velocity in obese subjects. Stoddart et al. (30) observed increases in both the A and E wave velocities; thus, the E/A ratio was unchanged. In our study, the mitral E and E/A ratio were lower in obese subjects, although the mitral A wave was significantly higher. We also found a correlation between insulin and HOMA-IR with the E and A wave velocities. This association suggested to us that insulin resistance may be a marker of left ventricular diastolic dysfunction.

There are many limitations to our study. First, it was a small observational study. Our results reflect only nondiabetic, normotensive obese women and cannot be generalized to all obese people. Our parameters may have been influenced by many factors, including preload, age, and heart rate. Obesity may affect echogenicity and impair image quality. The duration of obesity, lifestyle characteristics, and biochemical markers of endothelial damage that could influence LVMI were not examined. Lastly, left ventricular diastolic functions were only evaluated by traditional echocardiography instead of by tissue Doppler echocardiography.

Conclusion

Our study has shown that obesity is related to increased left ventricular mass independently of diabetes mellitus and hypertension. We also observed that age, BMI, WC, and serum insulin levels were the major determinants of left ventricular mass alterations in obese subjects. Routine followup examinations and initiating therapy before an increase in left ventricular wall thickness and before cardiac dysfunction may help to improve cardiac parameters and prevent cardiovascular complications.

Ethics Committee Approval: Ethics committee approval was received for this study from local ethics committee.

Informed Consent: Informed consent was obtained from patients who participated in this study.

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