PAPER

Left ventricular mass and function in young obese women†

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OBJECTIVE: Obesity is associated with a high mortality rate due to cardiovascular disease. Left ventricular (LV) hypertrophy has been described in relation to obesity. The aim of this study was to evaluate echocardiographically the LV mass and function in young obese women as compared to lean women with similar characteristics.

DESIGN: Prospective study.

SUBJECTS: Eighty-two young women (≤ 40 y), with obesity degree varying from I to III (BMI from 30 to 50 kg/m^2) were compared to eighty young lean women. All of them were normotensive, none had cardiovascular complaints or any previous history of pulmonary disease, and none were taking any medication. The LV mass was calculated by the Devereux and Reichek formula.

RESULTS: The LV mass was strongly increased in all obese groups ($P < 0.00003$ to 0.000005) compared to lean subjects. LV mass adjusted indexes for height, BMI or volume were also increased compared to lean subjects and when adjusted for weight it was decreased. However when comparing LV mass/body surface area index this difference was not statistically significant. The linear regression analysis showed a strong association between the degree of obesity and LV mass, ($r = 0.52$, $P < 0.001$). Systolic and diastolic function in obese patients were similar to lean subjects, except for a lower E/A ratio in the obese group ($P = 0.005$).

CONCLUSION: In asymptomatic young obese women, there are some echocardiographic findings suggesting early cardiac involvement that seems to be related to the degree of obesity.

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Introduction

Recognition of the adverse cardiovascular complications of obesity and the increasing frequency of obesity has stimulated considerable research into the relationship between obesity and the cardiovascular system.[1,2]

It has been observed that in obesity there is an expansion of blood volume and increased cardiac output. Increased left ventricular (LV) filling in obesity leads to an increase of LV cavity dimension and LV wall stress. This increase in wall stress induces ventricular hypertrophy and enlargement of ventricular mass that can be measured by echocardiography or angiography.[3,4] LV hypertrophy, detected through echocardiography, identifies patients at high risk of future morbid events, irrespective of age, blood pressure and ventricular function in relaxation. It has been proven to be an independent predictive risk factor of cardiovascular morbidity and mortality.[5–6]

Several authors have described an association between obesity and LV hypertrophy.[7–6] Some of these patients had associated cardiac failure or hypertension, conditions that can independently induce LV mass increase.[11,16] Some reports have included also old obese subjects.[10,16] Others have studied only morbidly obese.[9,14]

Systolic dysfunction and alteration of ventricular relaxation have also been described in obese subjects as compared to lean individuals,[9,14–26] but a variety of associated conditions such as diabetes mellitus, respiratory disease and hypertension could also affect LV function.

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†Many reports have described LV mass and function in obese patients. Is the LV mass increased in young and normotensive obese subjects? If so, is it associated with degree of obesity?

In the present study we evaluated LV mass and function in young, obese, normotensive women, without cardiovascular or pulmonary disease, as compared to a similar group of lean women to clarify the influence of the degree of obesity on cardiac performance.

Subjects and methods

One hundred and seventy (170) women were studied and according to the body mass index (BMI = weight/height^2) and World Health Organization they were divided into: grade I, 31 obese subjects (BMI $\geq$ 30 kg/m^2 and < 35 kg/m^2); grade II, 38 obese (BMI $\geq$ 35 kg/m^2 and < 40 kg/m^2); grade III, 16 obese (BMI $\geq$ 40 kg/m^2); and 85 lean subjects (BMI < 25 kg/m^2). All of them were volunteers for the study; the obese group had been sent for evaluation from an obesity outpatient clinic. All women were young, normotensive (systolic blood pressure $\leq$ 140 mmHg and diastolic blood pressure $\leq$ 90 mmHg), and had normal cholesterol and glucose levels. None of them had cardiovascular complaints, pulmonary disease or other associated systemic illness, and none were taking any medication.

All the patients had normal clinical examination and normal 12 lead resting electrocardiogram. Table 1 shows the clinical and echocardographic characteristics of the studied groups. Patients with inadequate echocardiographic windows were previously excluded.

Echocardiography

Echocardiography and cardiac Doppler studies were performed with a Siemens Sonoline CF image system (Siemens AG, Germany) using a 2.5 MHz transducer. Two-dimensional directed M-mode LV dimension measurement in accordance with American Society of Echocardiography recommendations was used to measure the diameter and thickness of the heart, in transversal paraexternal axis, at the level of the tendinous cords of the mitral valve.[27] The LV mass was calculated using the Devereux and Reichek formula (1.04 diastolic LV diameter + septal thickness + LV wall thickness)^3 − (diastolic LV diameter)^3 − 13.6), with Penn convention.[28] The adjusted indexes were obtained: LV mass/height, LV mass/weight, LV mass/BMI, LV mass/volume, and relative

thickness of the wall, which is equal to (LV wall thickness + septal thickness)/diastolic LV diameter. To evaluate the LV systolic function, the ejection fraction of the LV and the shortening fraction were obtained. The LV volume was obtained by using the revolution ellipsoid method.[29] The LV diastolic function was evaluated through the ratio between peak E and A wave velocity, isovolumetric relaxation time (IVRT) of the LV and halftime E wave deceleration.[29,30] LV hypertrophy was defined as LV mass and LV mass adjusted indexes equal to two standard deviations above lean healthy women mean values.

All echocardiographic recordings were obtained and interpreted by the same cardiologist (LLC). Eighteen percent of the patients, following entry into the study group, had echocardiographic studies performed by three observers working independently who were informed of the recommendations for the measurement of the diameter and the thickness of the LV wall. The level of agreement between the observers was estimated by means of Spearman's correlation coefficient, which varied from 0.75 to 0.96 with $P < 0.01$.

Statistical analysis

For the data analysis, the software Epi-info, version 6.0 (CDC, USA; OMS, Switzerland) and Statistical Analysis Software were used. The ANOVA or Kruskal–Wallis one way variance analysis was applied to compare the differences among the groups under study; the normality of the samples was tested by means of the Shapiro–Wilk test. The Student's t- or Mann–Whitney test was performed to compare difference between two groups. Linear regression analysis (Spearman's correlation coefficient) was applied to test the association between LV mass and the degree of obesity was performed. A P-value < 0.05 was considered necessary for statistical significance.

Results

Table 1 shows the LV mass and adjusted indexes for height, weight, BMI and volume in the obese and lean groups. Thirty-one of the individuals, both in the obese and lean group were white and the others, mulatto or black.

Table 1 Clinical data, LV mass and adjusted indexes in obese and lean women. Means ± standard deviation

Degree of obesity	n	BMI (kg/m^2)	Age (y)	LV mass (g)	LV mass/ height index (g/m)	LV mass/ BMI index (g/(kg/m^2))	LV mass/ weight index (g/kg)	LV mass/ BSA index (g/m^2)	LV mass/ volume index (g/mm^3)	RWT
Grade I	30	32.9 ± 1.3	28 ± 7	141 ± 30	88 ± 19	4.3 ± 0.9	1.7 ± 0.4	75 + 14	0.90 ± 0.18	0.28 ± 0.04
Grade II	37	37.2 ± 1.4	30 ± 5	146 ± 33	88 ± 24	3.9 ± 0.9	1.6 ± 0.3	74 + 18	0.87 ± 0.20	0.29 ± 0.06
Grade III	15	43.2 ± 3.0	30 ± 6	161 ± 39***†	101 ± 26***†	3.7 ± 0.8***†	1.5 ± 0.3***†	76 + 16	0.83 ± 0.19*/**	0.30 ± 0.06
Lean	80	21.3 ± 2.0	29 ± 6	115 ± 23	70 ± 18	5.4 ± 1.1	4.1 ± 12.4	80 + 17	0.79 ± 0.19	0.30 ± 0.05
P			NS	0.0000001	0.00002	0.0000001	0.0000001	NS	0.018	0.03

BMI = body mass index; BSA = body surface area; RWT = relative wall thickness. NS = not significant. *$P < 0.002$ grade I vs lean; **$P < 0.05$, grade II vs lean and grade III; ***$P < 0.00001$, grade I, II or III vs lean. †$P < 0.00001$, grade I vs grade III.

The LV mass and LV mass adjusted indexes (LV mass/height, LV mass/BMI, LV mass/volume) were increased in obese groups as compared to the lean group while the LV mass/weight was decreased. In contrast, LV mass/body surface index in the groups was similar.

The comparison of the LV mass indexes between the grade of obesity I, II or III group *vs* lean group have shown strong differences ($P < 0.00001$), except for the LV mass/volume index (Grade I *vs* lean, $P < 0.002$; grade II *vs* lean, $P < 0.03$; grade III *vs* leans, not significant) and for the LV mass/body surface area index (all not significant).

The comparison of LV mass indexes between the grade of obesity I *vs* grade III, were highly significant ($P < 0.00001$), except for the LV mass/body surface area index and LV mass/volume index. Patients with grade II obesity had LV mass indexes similar to grade III, except for a smaller LV mass/height index ($P < 0.04$).

Figure 1 shows the LV mass distribution in the groups. The frequency of LV hypertrophy was 33.7% in obese groups and 1.2% in lean group.

The relative wall thickness was also increased in obese groups compared with lean subjects.

There was no significant difference in the parameters of systolic and diastolic function of the LV of the obese and lean women, except for a lower E/A ratio in the obese group ($P < 0.03$). Table 2 shows the values for the variables of the LV systolic and diastolic functions.

The Spearman's correlation coefficient between LV mass and BMI was significant ($r = 0.52$, $P < 0.001$), Figure 2. The linear regression of LV mass and LV mass indexes showed better correlation with LV mass/BMI and LV mass/height indexes ($r = 0.59$ and $r = 0.52$, respectively, $P < 0.001$).

Discussion

Echocardiographic LV hypertrophy has been described in obese subjects, whether hypertensive or not.[8–23] Our data showed that the LV mass increased with the degree of obesity, and the LV mass adjusted indexes for height or BMI were higher, even when adjusted for weight. The relative wall thickness was lower in the groups of obese women than in the lean group.

Lauer *et al*[11] have shown increased LV mass and LV mass/height in healthy subjects from the Framingham Heart Study. They observed a positive correlation with BMI, age and blood pressure, but in their group white race was predominant. Hypertension and obesity each had significant independent associations with LV mass and wall thickness.
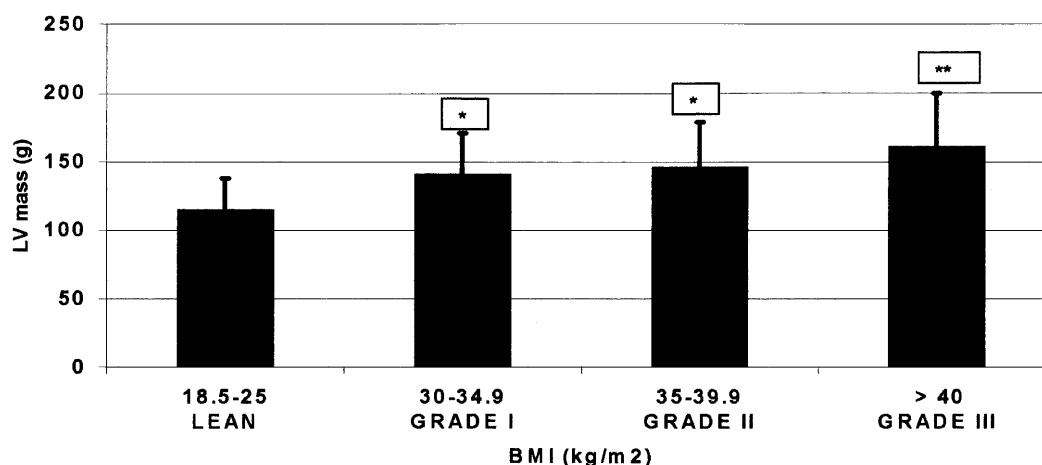

Figure 1 Mean and standard deviation of the LV mass in the groups of obese and lean women. *$P < 0.00003$; **$P < 0.000005$.

Table 2 Systolic and diastolic function of the left ventricular in obese and lean women. Means ± standard deviation

Degree of obesity	n	Systolic function		Diastolic function		
		EF (%)	(%D)	E/A	IVRT(msec)	EWD (msec)
Grade I	30	73 ± 6	37 ± 4	1.47 ± 0.33	70 ± 14	141 ± 43
Grade II	37	74 ± 6	37 ± 4	1.43 ± 0.33	71 ± 14	165 ± 52
Grade III	15	74 ± 4	38 ± 5	1.39 ± 0.28*	70 ± 14	162 ± 51
Lean	80	75 ± 5	37 ± 4	1.65 ± 0.52	88 ± 122	143 ± 38
P		NS	NS	0.03	NS	NS

EF = ejection fraction; %D = shortening fraction; E/A = ratio between the E and A waves; IVRT = isovolumetric relaxation time of the left ventricle; EWD = E wave deacceleration half-time. *$P < 0.03$, grade II *vs* grade III.
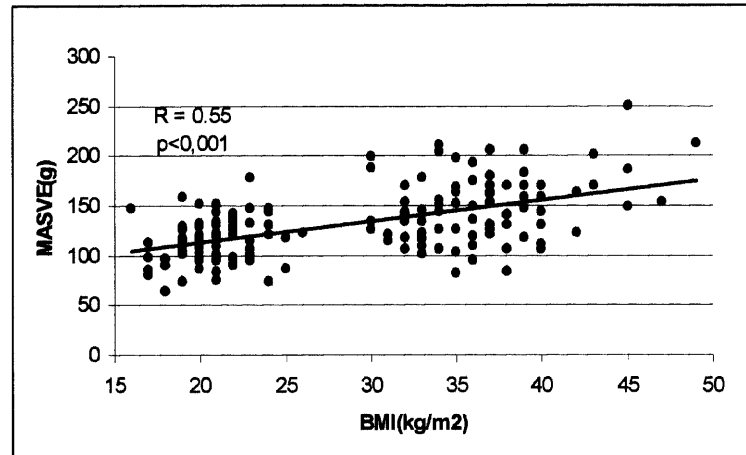
Figure 2 Spearman's coefficient correlation between LV mass and the obesity degree.

They observed LV hypertrophy in 8.4% of the women studied. The observed increase in the LV mass was due to the higher LV cavity dimension and wall thickness. De la Maza et al[15] have studied normotensive and hypertensive Chilean obese subjects (45 women and 5 men) aged 45 ± 11.6 y and 54 ± 8.8 y, respectively, and they observed a positive correlation of LV mass with BMI, blood pressure and tricipital thickness. They have found LV hypertrophy in 28% of normotensive (similar to our result of 33.7%) and 58% of hypertensive subjects. The body surface area did not have a significant correlation with LV mass but height did, similar to our data. They suggested that mentioned LV mass/height is a more sensitive index of LV hypertrophy than the other indexes in obese subjects and could be related to the smaller height of their population. Zarich et al[18] have corrected LV mass for body surface area and it was significantly increased in obese subjects. Although we have performed all these indexes and LV mass/-body surface area index did not show any difference among the groups, in contrast with the other adjusted indexes. The best correlations of LV mass and LV mass indexes were with LV mass/BMI and LV mass/height indexes.

In our study, the mean values observed for LV mass and LV mass/height, in the group of obese women, were greater than those observed by some authors[14,15] but lower than the values observed by others.[13,20] It is possible that these differences are due to the demographic characteristics which distinguish the population under study, such as age, sex, race, height, degree, type of distribution and duration of obesity or even the definition criteria for echocardiographic LV hypertrophy.

In relation to race, our study showed no difference in the LV mass between the two groups. These results are similar to Gottdiemer et al[16] who studied only male individuals but found a tendency, though not significant, of a greater prevalence of LV mass among black subjects as compared to white (31% vs 10%). In addition they found that black race was associated with greater relative wall thickness than non-black subjects.

The LV mass increase is also related to duration of obesity. Nakajima et al[10] have shown that the LV enlargement and wall thickness in obese patients who had been obese for more than 15 y was greater than in those who had been obese for less than 15 y.

The LV mass is also related to the distribution of obesity. Rasooly et al[12] have found that LV hypertrophy is more related to the waist or waist/hip ratio than BMI. In contrast, Bella et al[24] have shown that LV mass is more strongly related to free fat mass by bioelectric impedance than other indexes such as BMI, height or waist/hip ratio. Sasson et al[13] have described that LV mass was strongly associated with the degree of insulin resistance assessed using an index derived from intravenous glucose tolerance test, independent of BMI and blood pressure.

The parameters of LV hypertrophy observed in our study suggest that it is of eccentric, non-dilated type, since there was no increase of LV mass weight, and both relative wall thickness and LV mass/volume in the obese women were normal and even smaller than in the lean group. Therefore, LV hypertrophy was caused essentially by an increase of the diastolic volume of the LV in relation to the thickness of the wall. Our data are in accordance with previous studies.[25,31]

Several studies have shown that LV diastolic filling becomes progressively impaired as LV mass increases.[14,17–23] Zarich et al[18] studied 16 asymptomatic morbidly obese patients of both sexes, with age < 50 y and they found that 50% of them had LV diastolic filling abnormalities. Stoddard et al[19] studied 24 asymptomatic obese volunteers compared to lean control subjects and they found a prolonged of isovolumetric relaxation time in the obese group. They suggested that this index would be useful in the early detection of LV dysfunction in obesity. Similar results were published by Mureddu et al.[23]

In the present study, the systolic and diastolic function were similar in obese and lean women except for a lower E/A ratio in obese subjects. Similar results were observed by Zarich et al[18] who have studied morbidly obese patient. The highly positive correlation between systolic blood pressure and LV mass increase suggests that blood pressure may play a disproportionate role in the development of LV hypertrophy even in normotensive morbidly obese patients.

Karason et al[25] have shown that structural heart abnormalities occurring in conjunction with obesity decrease after weight loss and this regression was better predicted by weight loss than the reduction of blood pressure. Alpert et al[9,14,22,26] have, in morbidly obese individuals submitted to gastric restriction observed that weight reduction is also associated with decrease of LV mass and improvements in LV diastolic filling with a favorable effect on LV ejection fraction.

Our study showed that cardiac manifestations could happen in young, normotensive, symptomless, obese women due to the association of obesity with LV mass. The study also demonstrated a higher chance of LV hypertrophy in obese as compared to lean women. It is possible that sex, age, the duration of obesity, and the presence of arterial hypertension may play a significant role in the association between obesity and LV mass, and thus modify the evolutionary course of LV hypertrophy and consequently the prognosis of obese patients. Therefore it would be necessary to extend this study to individuals of different age levels, of both sexes and from various racial groups.

Based on the data of the present study, we suggest that the LV mass is increased in obese women and that echocardiographic study should be included in the evaluation of obese women to detect precocious LV hypertrophy. Preventive measures should be taken in the control of obesity, thus contributing to decrease the morbidity and mortality by cardiovascular diseases.

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