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# Left Ventricular Structural Changes in Uncomplicated Obesity

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## SUMMARY

*To determine the structural changes in left ventricle in moderate to severe uncomplicated obesity, 46 normotensive, healthy obese subjects (16 men and 30 women Mean±SE age 36.87±1.31 years; body mass index 36.46±0.57 kg/m$^2$) and 41 normotensive, normal weight volunteers, 16 men and 25 women, matched for age and sex distribution were studied by 2-dimensional and M-mode echocardiography. Mean arterial pressure (MAP) was higher normal in obese than in normal weight control subjects (Mean±SE 90.05±0.89 mmHg vs 94.65±0.82 mmHg, p<0.001). It was directly correlated with LV mass/Height index in obese subjects (p<0.02). Sum of posterior wall thickness (PWT) and interventricular septal thickness (IVST) known as wall thickness (SWT) was significantly increased in obese than in control subjects (p<0.001). LV internal diastolic diameter/height index (LVIDD/height index) was significantly increased in obese than in control subjects (p<0.001) and was directly correlated with LVmass/height index (<0.001). But the ratio of SWT to LV internal diastolic diameter/Height index (SWT divided by LVIDD/height index) known as relative wall thickness or RWT did not show difference between the two groups (p=0.512). LV mass/Height index was higher in obese than in normal weight subjects (p<0.001, highly significant). BMI was directly correlated with LV mass/Height index (r=0.724, p-<0.001), showing positive relationship between LVmass index and body mass index. LVmass increases as obesity becomes more severe and the mean arterial pressure (MAP) tends to increase with increasing LVmass/height index.*

## INTRODUCTION

Obesity is a condition of increased adipose tissue mass.[1] Excess adipose tissue requires a higher cardiac output to provide for higher metabolic demands. Because of this, left ventricular (LV) stroke volume is augmented in obesity, and LV end-diastolic pressure may become elevated.[2] This hemodynamic alteration, when sustained, is capable of producing LV structural abnormalities that may predispose to congestive cardiac failure even in the absence of systemic hypertension or underlying heart disease.[3] Heart weight increases as the body weight does but without a linear relationship. This increased weight is due to an increase in LV weight resulting from eccentric wall hypertrophy.[4]

Body mass index (BMI) is calculated as weight in kilograms divided by the square of the height in metres[5]. Normal weight is defined by BMI ≤27 kg/m$^2$, overweight as 27-30 kg/m$^2$ and obese as BMI ≥30 kg/m$^2$.[6]

Echocardiography has emerged as a sensitive, noninvasive technique for evaluating left ventricular dimensions, left ventricular mass and thus detecting left ventricular hypertrophy[7]. Studies on morbidly obese patients whose BMI was more than 40kg/m$^2$ reported increased LV wall thickness, cavity, size and mass compared with non-obese subjects.[8]

This study was designed to examine moderate to severe obese (BMI 30-40 kg/m$^2$) otherwise healthy, normotensive subjects for LV structural changes. We sought to examine the association of obesity with LV wall thickness, LV chamber size, LV relative wall thickness and LV mass index.

## SUBJECTS AND METHODS

We analysed a group of 46 asymptomatic moderate to severe obese normotensive subjects for LV structural abnormalities using the two-dimensional and M-mode echocardiographic techniques. Control group consisted of age, sex and height matched lean normotensive subjects.

**Inclusion criteria**

- Both sexes
- Age between 22-50 years
- Blood pressure $\leq$ 140/90 mmHg[9]
- BMI 30-40 Kg/m$^2$
- Premenopausal females

**Exclusion criteria**

- Pregnancy.
- Patients on anti-hypertensive medication.
- Patients with systemic hypertension, diabetes mellitus, ischaemic heart disease, arrythmias, heart blocks, asthma, chronic obstructive pulmonary diseases or valvular heart disease.
- Poor quality of echocardiogram.

**Study protocol**

Informed consent was obtained from each subject. Height and weight were measured and BMI calculated. Three blood pressure measurements were obtained in the sitting or supine and standing position with a mercury sphygmomanometer and a 14cm cuff, long enough to fit the most obese arm.[10] Normotension was diagnosed when the average measurement of blood pressure was $\leq$ 140/90 mmHg.[11]

Fasting blood sugar level, a resting electrocardiogram, chest X-ray and an echocardiogram (2 dimensional and M-mode) were obtained.

**Echocardiography and analysis**

Echocardiogram was performed using a power vision SA-380 A sys model No. $V_2$-31 ER001 and a probe of PSB 2.5MHz or PSB-2.75 MHz. These tests were done in the Department of Cardiology, Mayo Hospital.

Echocardiographic measurements and their normal ranges included the LV internal systolic (LVIDS) and diastolic (LVIDD dimensions (38-56 mm respectively) the LV posterior wall thickness (PWT) in systole and diastole (6-11 mm) and interventricular septal thickness (IVST) in systole and diastole (6-11 mm).[12] LVmass was calculated using the formula of Devereux and Reichek.

$$LVM = 1.04\ ([LVIDD+PWT+IVST]3-[LVIDD]^3)-13.6$$

(Mureddu et al)[13]

LVmass index was calculated by dividing LVmass with height in metres.[1]

LV hypertrophy was defined as LVmass index $>$136g/m in men and $>$112 g/m in women.[14]

LV wall thickness was determined by adding up thickness of IVST and PWT. Relative wall thickness was calculated by dividing wall thickness with LVIDD/height[15].

**Statistical analysis**

All results were given as mean $\pm$ SE. Differences between groups were assessed by unpaired student t-test. Correlation between clinical and echocardiographic parameters was found with linear regression analysis by the least square method. A p-value $\leq$0.05 were considered significant for all analyses.

## RESULTS

The study group consisted of 46 normotensive, healthy obese subjects. There were 16 male and 30 female subjects. They were matched with 41 normotensive, normal weight control subject, 16 male and 25 female.

**Comparison of clinical parameters**

The control and obese groups were comparable in age (37.15$\pm$1.44 years vs 36.87$\pm$1.31 years, p=NS), height (159.41$\pm$1.18 vs 158.48$\pm$1.12 cms, p=NS) and sex (male/female ratio 16/25 vs 16/30, p=NS for both groups). Body weight was significantly greater in obese group as compared with control group (60.12$\pm$1.25 vs 97.20$\pm$1.90 kg, p$<$0.001) (Table-1).

Body mass index was significantly greater in obese as compared to control group (23.48$\pm$2.53 vs 36.46$\pm$0.57 kg/m$^2$, p$<$0.001).

The normotensive obese subjects had higher normal systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) as compared to their age, sex and height matched counterparts in the control group (P$<$0.01) (Table 2). The significant positive correlation

**Table 1: Demographic characteristics of study group.**

| *Variables* | *Normal Weight (n=41)* | *Obese (n=46)* | *p-value* |
|---|---|---|---|
| Age (years) | 37.15±1.44 | 36.87±1.31 | 0.887 |
| Gender (M/F) | 16/25 | 16/30 | 0.652 |
| Body weight (kg) | 60.12±1.25 | 97.20±1.90 | <0.001 |
| Body Height (cms) | 159.41±1.18 | 158.48±1.12 | 0.567 |
| Body Mass Index kg/m2 | 23.48±0.2538 | 36.46±0.5702 | <0.001 |
| Heart Rate Beats/min | 81.51±0.84 | 78.78±1.24 | 0.078 |
| Systolic Blood Pressure (SBP) mmHg | 119.51±1.09 | 127.28±1.24 | <0.001 |
| Diastolic Blood Pressure (DBP) mmHg | 74.78±0.97 | 78.30±0.75 | 0.004 |
| Mean Arterial Pressure (MAP) mmHg | 90.05±0.89 | 94.65±0.82 | <0.001 |

**Table 2: Obesity and LV structure, M-mode echocardiographic features**

| *Variables* | *Normal Weight (n=41)* | *Obese (n=46)* | *p-value* |
|---|---|---|---|
| LVIDD (mm) | 43.39±0.56 | 49.56±0.70 | <0.001 |
| LVIDD/HT (mm/ht) | 27.28±0.417 | 31.354±0.5116 | <0.001 |
| LVIDs (mm) | 28.29±0.40 | 32.8±0.65 | <0.001 |
| PWT (mm) | 7.663±0.115 | 9.222±0.156 | <0.001 |
| IVST (mm) or VST | 7.88±0.117 | 9.148±0.174 | <0.001 |

**Table 3: Obesity and LV wall thickness, relative wall thickness, LV massing and LV mass/Ht index**

| *Variables* | *Normal Weight (n=41)* | *Obese (n=46)* | *p-value* |
|---|---|---|---|
| Wall thickness (mm) SWT | 15.54±0.18 | 18.37±0.31 | <0.001 |
| Relative wall thickness SWT/LVIDD ratio | 0.36±0.04 | 0.37±0.04 | 0.512 |
| LV mass (g) | 115.707±3.347 | 187.37±8.4 | <0.001 |
| LV mass/Ht index g/m | 72.7024±2.131 | 118.03±5.085 | <0.001 |

between LVmass/Ht index and mean arterial pressure is shown in Fig.1 ($r=0.376$, $p<0.02$).

Figure 2 shows a positive correlation between BMI and LV mass/Height index ($r=0.724$, $p<0.001$).

Figure 3 shows a positive relationship between LV mass/Ht index and LVIDD/Ht index ($r=0.753$, $p<0.001$).

Table 3 shows that obese subjects had left ventricles with larger cavity size and thicker walls. The left ventricular internal diastolic (LVIDD) dimension was significantly more in obese as compared to control group (43.39±0.56 vs 49.56±0.70 mm, $p<0.001$) even when normalized for height (adjusted values 27.28±0.41 vs 31.35±0.511 mm/metre, $p<0.001$). Left ventricular

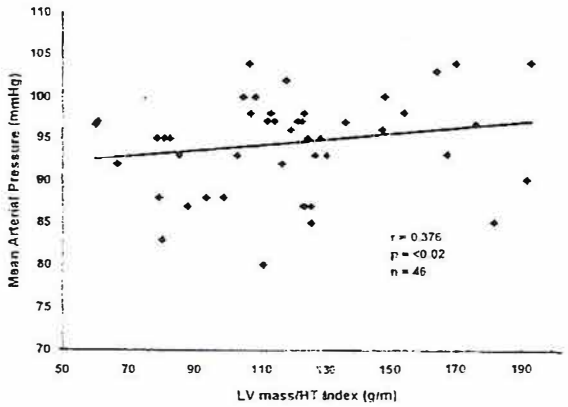


**Fig. 1: Relationship between LVmass/HT Index and Mean Arterial Pressure (MAP).**

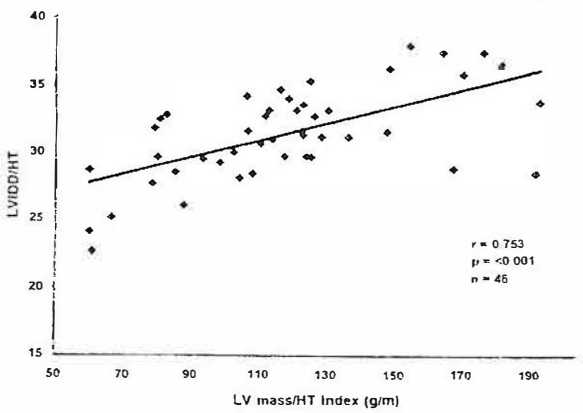


**Fig. 3: Relationship between LVmass/HT Index and LVIDD/HT.**

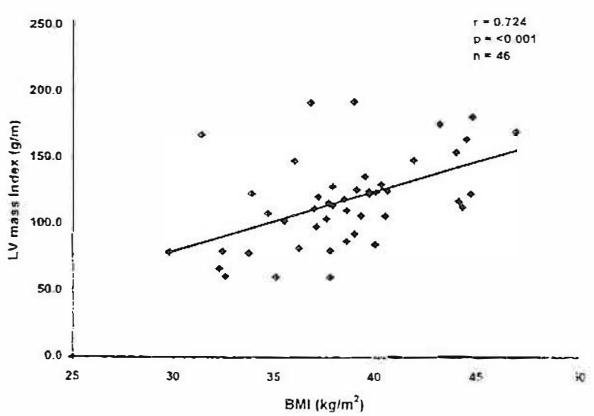


**Fig. 2: Relationship between BMI ($Kg/m^2$) and LVmass/Height indexs) (Grams/meter).**

internal systolic dimension (LVIDS) was more in obese as compared to the control group ($28.29\pm0.40$ vs $32.8\pm0.65$ mm, $p<0.001$). Left ventricular posterior wall (PWT) thickness was more in obese than in control group ($7.663\pm0.115$ vs $9.222\pm0.156$ mm, $p<0.001$). The interventricular septal thickness (IVST) was also increased in obese group as compared to control ($7.88\pm0.117$ vs $9.148\pm0.174$ mm, $p<0.001$).

However absolute increase in LVIDD which is >5.6cms was found in 7 obese subjects out of 46, yielding 15.2% prevalence rate of eccentric hypertrophy.

Absolute increase in PWT i.e., >1.1cm was seen in only 3 out of 46 obese subjects making 6.52% of the study population. Absolute increase in IVST i.e., >1.1cms was noted in 5 obese subjects out of 46, yielding 10.8% of the study population. Absolute increase in LVIDD, PWT or IVST was not found in the control group.

Table 4 shows that though obese subjects had more wall thickness (SWT) than control subjects ($15.54\pm0.18$ vs $18.37\pm0.31$ mm, $p<0.001$), relative wall thickness ratio (RWT) was not statistically different between normal weight and obese subjects ($0.36\pm0.04$ vs $0.37\pm0.04$, $p=0.512$). LV mass was higher in obese than in control subjects, both in absolute values ($115.7\pm3.3$ vs $187.3\pm8.4$ grams, $p<0.001$) and after normalization for height, LV mass/Ht index ($70.70\pm2.1$ vs $118.03\pm5$ g/metre, $p<0.001$). LV mass/Ht index criteria for left ventricular hypertrophy was fulfilled in 43% of obese subjects.

## DISCUSSION

Results of present study demonstrate that moderate to severe obesity is characterized by

pecific changes in LV structure. A significant ositive correlation between BMI and V mass/height index was found in obese subjects. elative wall thickness which is the ratio of wall ckness to LV internal diastolic dimension SWT/LVIDD) in moderate to severe obesity was milar in both groups. Another significant finding as that obese normotensive subjects had higher ormal systolic blood pressure (119.51±1.09 vs 27.28±1.24 mmHg, p<0.001) and mean arterial ressure (90.05±0.89 mmHg vs 94.65±0.82 mHg, p<0.001) as compared to the lean ormotensive control group.

Previous echocardiographic studies on ormotensive and asymptomatic morbidly obese ubjects have reported prevalence rate of LV ypertrophy ranging from 64-87%. LV hypertrophy these studies was defined by LVmass/height ndex>135g/m in men and >112 g/m in omen[8,14]. Our study showed a positive correlation etween BMI and LVmass/height index. LV ypertrophy diagnosed on the basis of same V mass/height index criteria was found in 43.4% the study population. The prevalence of LV ypertrophy in our study is less because our ubjects were moderate to severe obese (BMI 30-40 g/m$^2$) while previous studies were done on orbidly obese (BMI>40 kg/m$^2$) subjects.

Messerli et al[16] studied 17 normotensive lean nd obese patients and reported left ventricular ypertrophy (LVH) (as defined by a posterior wall hickness (PWT) of greater than 11mm) in more han 50% of all obese patients and in less than 20% f the lean patients who had same arterial pressure. our study the mean posterior wall thickness of bese was more than the control group (9±1 vs ±0.7 mm, p=<0.001) but absolute increase in WT which is >11 mm was found only in 5 ubjects (10.6%). This striking difference could be e to a number of reasons. Firstly, the number of ubjects recruited for both studies is different. revious study was done only on 17 obese ormotensive healthy subjects while present study xamined 46 such subjects. Secondly, the important spect of the duration of obesity was not addressed both of these studies. Other criteria of weight, MI, blood pressure and age of subjects were the ame in two studies. Thirdly, the most important ause could be the regional difference between USA nd Pakistan.

In 1995b Alpert et al[3] identified LVIDD as indirect index of LV preload and systolic blood pressure and relative wall thickness (an indirect index of LV wall stress) as indirect indexes of after load that influence the LVmass. They noticed positive relationship between each of these indexes and LVmass/height index[3].

Nakajima et al[17] performed a noninvasive study on 35 obese patients who weighed 57 to 118 kg (85±16) aged 16-60 years (34.5±12). They showed significant increase in LVIDD (P<0.001), LVIDS (P<0.001) thickness of interventricular septum (P<0.001) and posterior wall (PWT) (P<0.001) compared with non-obese subjects[17]. Our results are therefore concordant with those of Alpert et al.[3] and Nakajima et al[17].

## CONCLUSIONS

Normotensive obese subjects had higher normal systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) as compared to their age, sex and height matched control group members. Obesity is associated with preclinical structural abnormalities of left ventricule. There is increased left ventricular wall thickness, increased left ventricular internal diastolic and systolic dimensions and increased LVmass/height index. However in moderate obesity the relative wall thickness which is the ratio of LV wall thickness to LVIDD remains similar to the lean normotensive control subjects. This study demonstrates the potential importance of controlling for weight in studies detecting structural abnormalities of left ventricle in patients of different body mass indexes. Further studies are required to find out the left ventricular pump function and diastolic filling in uncomplicated moderate obesity.

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