

The Relationship between Body Mass Index and Left Ventricular Structure and Function in Healthy Adults

Amjad Fawzi*
Enaas S. Aziz**

MBCbB, PhD
MBCbB ,MSc

Summary:

Background: There is considerable evidence which associate cardiovascular morbidity and mortality with obesity, however, a direct effect of uncomplicated obesity on cardiac function is not well established.

Objective: To evaluate the relationship between body mass index (BMI) and left ventricular structure and function.

Methods: This cross-sectional study was carried out on (146) apparently healthy adults from both sexes (85 male and 61 female) aged 20-59 years (36.49±9.92). Subjects were grouped according to BMI into 1. normal weight group included 47 subjects (BMI=18.5-24.9Kg/m²); 2. Overweight group included 43 subjects (BMI=25-29.5 Kg/m²) and 3. obese group included (56) subjects (BMI≥30). Echocardiographic indices of left ventricular structure and function were obtained. This study was performed between October 2009 till April 2010 at the Echocardiography Unit in Ibn Sina Teaching Hospital in Mosul.

Results: There was a significant increase in LVM(left ventricular mass), LVMI(left ventricular mass index), LVIDD(left ventricular internal dimension in diastole) and PWT(posterior wall thickness) among over weight and obese subjects (P< 0.001), however, both contractility indices [LVEF%(left ventricular ejection fraction percent) and LVFS %(left ventricular fractional shortening percent)] seemed unchanged significantly. The left ventricular diastolic function [E/A ratio(ratio of passive to active velocity inflow)] has significantly decreased among the obese group (P< 0.001). This observed decrease in E/A ratio with increasing BMI coincided with a statistically significant increase (P< 0.001) in LAD (left atrial dimensions).

Conclusions: Increased body weight is positively correlated with LVM but not with systolic dysfunction. All subjects with isolated obesity have subclinical left ventricular diastolic dysfunction; this correlates with BMI.

Key words: Obesity, Body Mass Index, Left Ventricular Function.

Fac Med Baghdad
2011; Vol. 53, No. 3
Received Mar. 2011
Accepted May. 2011

Introduction:

The prevalence of obesity is increasing in both the developed and developing countries¹. It is associated with increased mortality and morbidity. Increasing studies on the relation of body weight to left ventricular structure and function have revealed a spectrum of more minor cardiovascular changes, ranging from hyperdynamic circulation to subclinical cardiac structural changes in obesity². However, the scarcity of information added to the existing controversy was the motivation behind conducting the present study on healthy subjects whether normal weight, overweight or obese.

Subjects and methods:

One hundred forty six healthy volunteers of 85 males and 61 females were involved in the present study.

*.Dept. of physiology, Ninevah College of Medicine, University of Mosul

**Echocardiography unit- Ibn-Sina Teaching Hospital.

Their ages ranged from 20 to 59 years (mean 36.5 ± 9.92). The study was performed between October 2009 till April 2010 at the Echocardiography Unit in Ibn Sina Teaching Hospital in Mosul. The study group included medical staff, patient relatives, and hospital workers. Personal and health information was taken after obtaining an informed consent from all participants. An independent scientific committee revised and approved the study protocol and information to be provided to the subjects.

The present study was performed in the following sequence:

History and Clinical Examination:-Based on history and clinical examination, the following criteria for subject exclusion were considered: (a) The presence of any history or findings of cardiopulmonary diseases. (b) Hypertension, Diabetes mellitus, malignancy, impaired renal or liver functions, and pregnancy.

Physical Measurements: The standing height and body weight were measured to the nearest 0.5 cm and 0.5 Kg respectively.

BMI = Wt (Kg) / Ht (m)²(3). Based on BMI, study participants were classified according to WHO classification (2004) into the following groups: (a) Normal Body Weight Group: BMI between 18.5-24.9 kg/m². (b) Overweight Group: BMI between 25–29.9 kg/m². (c) Obese Group: BMI ≥ 30 kg/m².

Electrocardiography (ECG): ECG recording (GE Medical system, India, model 2005) was performed as part of subjects' checking in addition to history and clinical examination to exclude the existence of any significant cardiovascular disease.

Echo-Doppler Examination: Two-dimensionally guided M-mode echocardiograph (Philips envisor, Italy, model 2004) was used to evaluate left ventricular function in the studied group. M-mode recordings of the left ventricle were obtained with the subject in the supine and left lateral decubitus position. All measurements were obtained according to the recommendations of the American Society of Echocardiography⁴.

1) The LV structure were assessed by left ventricular mass (LVM), left ventricular mass index(LVMI), posterior wall thickness (PWT), and relative wall thickness (RWT).

2) Left Ventricular Systolic Function was assessed by: (a) Ejection fraction (EF %): This is the fraction of the end diastolic blood volume that is ejected by the left ventricle in one beat. Usually equal to about 65% of the EDV(3). (b) Fractional endocardial shortening (FS %): Fractional shortening is a rough measurement of left ventricular systolic function, with the normal range being 25% - 45%.

FS = (LVIDd - LVIDs) / LVIDd * 100%..... (5).

3) Left Ventricular Diastolic Function was assessed by: (a) Maximum velocity of passive transmitral inflow (E). (b) Maximum velocity of active transmitral inflow (A). (c) Ratio of passive to active velocity (E/A) ratio.

Data analysis: All data were analyzed using the Statistical Package for the Social Science (SPSS, version 17). The students' t-test was used to compare means of the different variables measured. Analysis of Variance in order to determine the changes in the means of the parameters. Duncan Multiple Range Tests were used to compare the means after a significant analysis of variance (ANOVA) test.

Results

Table1 compares between different measured left ventricular echocardiographic data when subjects were grouped according to body mass index (BMI) into normal weight, over weight and obese.

There was a significant increase (P< 0.001) in LVM, LVMI, and PWT among over weight and obese subjects(table1, figure1). On the other hand, both contractility indices (LVEF% and LVFS %) seemed unchanged significantly with increasing body weight. The left ventricular diastolic function, as presented by E/A ratio, showed a clear consistent and statistically significant decrease with increasing BMI especially among the obese group (P< 0.001). This observed significant decrease in E/A ratio with increasing BMI coincided with a statistically significant increase in LAD (P< 0.001) as shown in figure2.

Table1: Comparison of the Left Ventricular Echocardiographic Parameter between Different BMI Groups. Mean (Standard Deviation)

Parameter	Normal weight(n=47)	Over weight(n=43)	Obese(n=56)	P value
LVM(g)	144.72 (45.95) a	173.05(45.40) b	182.58 (46.95) b	<0.001
LVMI (g/m ^{2.7})	35.55(8.42) a	43.20(10.33) b	48.42 (10.96) c	<0.001
PWT(cm)	0.91(0.22) a	1.00(0.15) b	1.01(0.18) b	<0.001
RWT	0.42 (0.08)	0.44 (0.08)	0.42 (0.08)	NS
LVEF (%)	68.89 (7.82)	66.60 (8.78)	66.51 (6.87)	NS
LVFS (%)	39.74 (0.06)	36.07 (0.06)	34.44 (0.08)	NS
E/A Ratio	1.54(0.41) b	1.4(0.28) b	1.26 (0.29) a	<0.001
LAD(cm)	2.66(0.43) a	2.87(0.44) b	3.11 (0.41) c	<0.001

The Means that have at least one similar letter mean that there is no significant different between them according to Duncan test at the subset or alpha = 0.05.

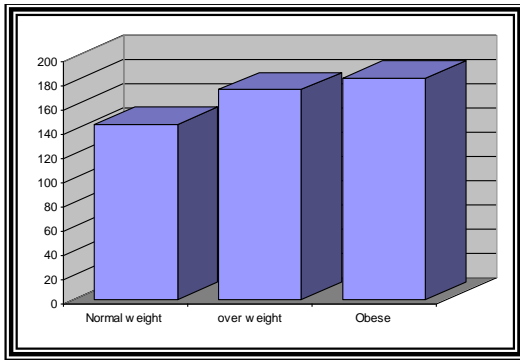


Figure 1: Comparison of Mean LVM between Different Body Weight Groups (P<0.001)

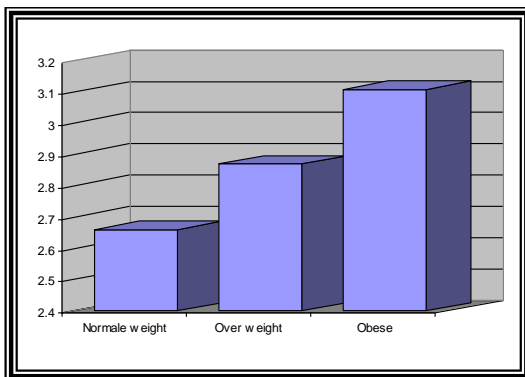


Figure (2): Comparison of Mean LAD between Different Body Weight Groups (P<0.001)

Table 2 demonstrates the relationship between all measured LV echocardiographic parameters and body weight represented by the BMI (r and p values). There was a significant positive correlation between LVM and body weight among the total study group (P<0.001). This relationship was especially significant among the obese group (P<0.001) as compared to the normal and over weight groups (NS). The same was true for PWT. On the other hand, the LVEF% was negatively but not significantly correlated with body weight among the total study group, unlike the LVFS% which showed significant negative correlation with the body weight (p<0.001) especially among obese group (P<0.001). The E/A ratio also revealed significant negative correlation with body weight only when considering the study group as a whole (p<0.001). Similarly, LAD revealed significant positive correlation with body weight (p<0.001).

Table2: The Relationship between All Measured LV Echocardiographic Parameters and Body Weight (r and p values)

Parameter	Total	Normal weight(n=47)	Over weight(n=43)	Obese(n=56)
LVM(g)	0.27 <0.001	0.01 NS	0.22 NS	0.29 <0.001
LVMi(g/m ²)	0.46** <0.001	0.04NS	0.13 NS	0.25 NS
PWT(cm)	0.23** <0.001	0.01NS	0.18 NS	0.28* <0.001
RWT	0.05 NS	-0.005NS	0.01 NS	0.19 NS
LVEF%	-0.15 NS	0.08NS	-0.08 NS	-0.18 NS
LVFS%	-0.17* <0.001	0.18 NS	-0.10 NS	-0.26* <0.001
E/A ratio	-0.35* <0.001	-0.07 NS	-0.10 NS	-0.16 NS
LAD (cm)	0.34* <0.001	0.01 NS	0.06 NS	0.25 NS

** Correlation is significant at the level 0.01 (2 tailed)

* Correlation is significant at the level 0.05 (2 tailed)

Discussion:

Impairment of cardiac function has been reported to correlate with degree of obesity i.e. body mass index and duration of obesity6-7. Obese subjects who have no other clinically appreciable cause of heart disease may exhibit abnormalities in LV structure. In our study, LVM increased consistently with BMI (figure1) and the relationship was significantly positive reinforcing the view that LVM may increase in accordance with body size in truly uncomplicated obesity which is in accordance with Peterson et al., 8 and Valocik et al. 9. The likely causes of the increased LV mass in obese persons include increase in the total blood volume as a result of an increase in the size of the vascular bed in the excess adipose tissue, cardiac output, and resultant increased afterload8 as each of these changes may occur in obesity, they may induce cardiac hypertrophy and play a part in LV morphologic alterations.

This study also demonstrated that BMI correlated positively with PWT (table 2) which is in complete agreement with Peterson et al.8, Valocik et al. 9 and Wang et al.10. In addition to LV morphologic

alterations in obese subjects found in our study, functional changes of the heart have been noticed as well. Various authors have reported depressed LVEF11-12, normal EF13-14, and supernormal EF15-16 in obese subjects. In our study, however, no significant relationship was found between BMI and LVEF% and this agrees with Valocik et al.9 and Dorbala et al.17 but disagrees with Chadha et al.18 who reported a positive relationship between BMI and LVEF% while LVFS% correlated negatively with the body weight especially in the obese subjects and this disagrees with Chadha et al.18 who reported positive correlation between LVFS% and BMI. The ejection fraction is a reliable index but is relatively insensitive to left ventricular contractile function, so its value may be maintained within normal limits even when there is substantial compensatory modification of the contractile state. However, the normality of the ejection fraction was in accordance with the normal RWT of our study group, which indicates that systolic function was preserved, however. Most echocardiographic studies using measurements of the ejection phases to evaluate systolic function in obese subjects have shown normal results19. Regarding the left ventricular diastolic function, several studies have also assessed LV diastolic filling and diastolic function in obesity. As with the findings in systolic function, results are variable and somewhat conflicting. Some studies have found reduced early diastolic (E-wave) velocities 17 while others have found them to be unchanged20-21-22. The late diastolic (A-wave) velocities have been reported to be increased16-20 or unchanged8-22. In contrast to systolic function, our results demonstrated that diastolic function (E/A ratio) correlated negatively with BMI (table 2); not only in the obese personnel but also in overweight subjects and this agrees with Valocik et al. (10) and Chadha et al.18. This point is vital because a greater awareness of weight control can induce beneficial changes in cardiac function, since alterations that occur in obesity can be reversed easily and quickly by weight loss23. Moreover, our results showed that LAD increases significantly with BMI (table1,2, figure 2) and this agrees with Iacobellis et al.16 Peterson et al.8 and Chadha et al.18. The mechanisms of increasing left atrial size (LA) appear to be similar to those causing LVH: increasing BMI, volume overload, and possibly LV diastolic filling abnormalities. Interestingly, obese subjects were found to have an increased risk of developing atrial fibrillation, and this risk can be entirely explained by the increase in LA size.

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