

ORIGINAL ARTICLE

IMPACT OF BODY MASS INDEX ON LEFT VENTRICULAR MASS

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Background: Obesity is an independent factor implicated in left ventricular (LV) hypertrophy and increased left ventricular mass. Early detection of increased LV mass in obese patients has prognostic and therapeutic implications. **Methods:** This case series was carried out in Choudhry Parvaiz Ellahi Institute of Cardiology, Multan from March to August 2011. A total of 100 consecutive patients including male and female referred for transthoracic echocardiography were included in this study. Patient's demographics were obtained using a questionnaire. Anthropometry (BMI) and two-dimensional echocardiography to calculate LV mass. **Results:** Echocardiograms of 100 patients showing mean age 42.42 ± 6.04 years, with BMI of 28.42 ± 5.52 and LV Mass of 136.05 ± 29.117 shown linear correlation between BMI and LV mass. **Conclusion:** It is concluded from the study that LV mass increases with increasing BMI.

Keyword: Echocardiography, Body mass index, LV mass

J Ayub Med Coll Abbottabad 2014;26(2):167-9

INTRODUCTION

Obese and overweight persons are at increased risk of heart diseases and constitute an important public health problem because of associated increased risk of cardiovascular (CV) morbidity and mortality.¹ A world health organization (WHO) consultation described obesity as a chronic disease that is prevalent in developed and developing countries.² Body mass index (BMI) is recognized and widely used for identifying overweight or obesity. The cut-off points of BMI for Asians were: in overweight BMI >25.0 kg/m² and in obesity BMI >30.0 kg/m². BMI was calculated by the formula: $BMI = Wt. (kg)/Ht (m^2)$.³

Obesity also influences blood pressure (BP) and left ventricular hypertrophy (LVH). The degree of increased myocardial muscle mass (LVH) is also a strong and independent risk factor for cardiac morbidity and mortality.⁴ Left ventricular hypertrophy is defined as an increase in the mass of the left ventricle, which can be secondary to an increase in wall thickness, an increase in cavity size, or both. A significant increase in the number and/or size of sarcomeres within each myocardial cell is the pathologic mechanism. The normal LV mass in men is 135 g and 99 g in women.⁵ LVH is usually defined as two standard deviations above normal. In clinical practice, however, the presence of LVH is more commonly defined by wall thickness values obtained from M-mode or 2D images from the parasternal views. For inclusion, echocardiographic images had to consist of a two-dimensionally guided M-mode evaluation of the left ventricle using the recommendations of the American Society of Echocardiography.⁶ From these end-diastolic M-mode measurements of septum (SD), posterior wall (PWD) and left ventricle (LVD), LVM was

calculated using the Devereux formula: $LVM (in grams) = 0.84 * 1.04 [(VSTd + LVIDd + PWTd)^3 - (LVIDd)^3] + 0.6$.^{7,8}

MATERIAL AND METHODS

This descriptive case series was carried out in Ch. Parvaiz Ellahi Institute of Cardiology, Multan from March to August 2011. A total of 100 consecutive normal subjects including males and females ≤ 50 years of age were included in the study. Subjects were referred from OPD with non-specific complaints as shortness of breath, palpitation, preoperative cardiac risk assessment for non cardiac surgery, and screening for cardiovascular structural abnormalities.

Patients with current coronary artery disease, current/prior angina, or myocardial infarction, current/prior history of arrhythmia, cardiovascular co-morbidity (prior cerebrovascular accidents and/or peripheral vascular disease), current therapy with vasoactive drugs, statins or fibrates, active smoking, creatinine >2 mg/dl, presence of neoplasia and/or systemic disease, and suboptimal echocardiographic window were excluded from the study.

Participants provided information on age, family history, personal habits (alcohol intake, tobacco consumption, type and level of physical exercise, drugs ingestion). Routine measurements of blood pressure, height in centimetres (cm), and weight in kilograms (kg) were recorded. An echocardiogram was obtained on all participants (sonos 5500, Hewlett-packard, equipped with mediview). Echocardiograms included cross sectional, M mode, and Doppler studies. The following indices of cardiac functions were evaluated: left ventricular systolic function, left ventricular end diastolic dimension (EDD) and

fractional shortening (FS) were obtained in the parasternal long axis views using M Mode; left ventricular end diastolic (EDV) and end systolic volumes (ESV), stroke volume (SV) and ejection fraction (EF) were measured from apical four chamber view. The relative wall thickness (RWT) was calculated from the posterior wall thickness (PWT) and the EDD, as $(2 \times \text{PWT}) / \text{EDD}$. Left ventricular mass (LVM) was calculated by the Devereux formula: $\text{LVM (in grams)} = 0.84 * 1.04 [(\text{VSTd} + \text{LVIDd} + \text{PWTd})^3 - (\text{LVIDd})^3] + 0.6$.^{7,8}

Data were analyzed using SPSS-10. Categorical variables were described as frequencies and percentages. Quantitative variables were described as minimum, maximum, mean and standard deviation (SD). The relationship between two quantitative variables was assessed using Pearson's correlation coefficient. Level of statistical significance was set at $p < 0.05$.

RESULTS

Echocardiograms of 100 patients to assess left ventricular mass was obtained. Mean age of the patients was 42.42 ± 6.04 years [range 24–50], while BMI was in the range of 19–45 with mean of 28.42 ± 5.52 . LV Mass was in the range of 76.197 with mean of 136.05 ± 29.117 . There were 59 (59%) male patients and 41 (41%) female patients. The absolute values of the left ventricular mass were correlated to BMI (Figure-1). * Linear correlation coefficient r was used to examine the relation between LVM measurements and BMI. The value of $r = 0.6$. Our data showed significant correlation between LVM and BMI with p value of ($p = 0.00$).

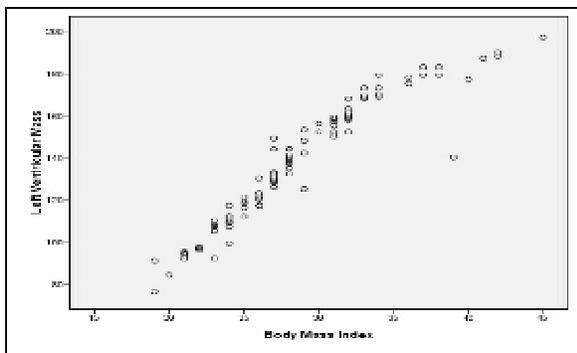


Figure-1: Linear positive correlation between left ventricular mass and body mass index $r = 0.6$

DISCUSSION

Overweight and obesity influences left ventricular muscle thickness, structure and function. WHO Western Pacific Region in 2000 recommended lower cut-off for overweight (Body Mass Index-BMI ≥ 23.0) and obesity (Body Mass Index-BMI ≥ 25.0) in

Asians.¹⁰ However, studies considering the new recommendations of BMI are lacking.

The finding that BMI is the driving factor behind increased LV mass was not totally unexpected but has never been specifically tested before, especially in the context of non-diabetic and normotensive patients and in patients without comorbidities.

Left ventricular mass plays an essential role in determination of left ventricular hypertrophy (LVH). The degree of hypertrophy indicates the severity of volume overload of the systemic circulation.^{11–13} Electrocardiogram is relatively non-sensitive and infrequently predicts LVH¹⁴, especially in obese persons due to decrease in surface voltage. M-mode echocardiography provides an accurate assessment of LVM that is more sensitive and specific than the electrocardiogram for detecting LVH¹⁵ which is an important prognostic finding to evaluate the high risk of subsequent cardiovascular morbidity and mortality^{14–16}. The invasive biplane angiographic method of Rackley *et al* is the most accurate method for assessment of LVM.¹⁶ This method is invasive and cannot be applied on a larger population. However, LVM can be accurately calculated by echocardiography and it may provide a better indicator of the extent of the cardiac involvement and evaluation in normal and disease state.

In our study patients were found with almost 1:1 male to female and with positive correlation between BMI and LV mass. These results are comparable with the findings of Rajesh showing that increasing body mass increases LV mass.¹⁷

In another study by Rodrigues it was concluded that BMI in general and particularly increased abdominal adiposity (waist circumference WC as surrogate) seems to account for most of LV mass increase in normotensive individuals, mainly in women.¹⁸ The same conclusion was made in a study by Sivanandam.¹⁹

In a cohort on healthy females by Rider, it was observed that subjects with a wide range of BMIs, ventricular hypertrophy occurs without associated cavity dilatation in overweight individuals; while in manifest obesity, both cavity dilatation and ventricular hypertrophy occur.¹⁹

As discussed above that the increasing BMI is associated with increasing LV mass of ventricles which leads to increased resistance to ventricular diastolic filling and leading to diastolic dysfunction as shown from increased atrial filling velocity and left atrial enlargement and predisposing to atrial and ventricular arrhythmias and results in increasing morbidity and mortality. Thus strict control of body weight is absolutely necessary in the maintenance of

cardiovascular health and functional aspect of myocardium.

The study is not designed to show the cause and effect relationship between BMI and LVM, however in study design the confounding variables as hypertension, diabetes mellitus, extreme age, congenital and valvular heart disease are controlled. The study only shows that significant positive correlation exists between body BMI and LVM.

CONCLUSION

BMI is significantly correlated with left ventricular mass, even after controlling for confounding variables as diabetes mellitus, renal failure, blood pressure and other co-morbidities mentioned earlier. The increase in left ventricular mass associated with increasing BMI reflects increase in both left ventricular wall thickness and left ventricular internal dimension.

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