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RESEARCH ARTICLE

Effect of obesity on cardiac function in healthy individuals without any other cardiac comorbidities – A study based on echocardiography

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ABSTRACT

Background: Obesity constitutes a major health issue in the modern world because of its association with morbidity, mortality, and cardiovascular diseases (CVD). The excess in body fat determines a rise in each preload and afterload leading to a hyperdynamic circulation, chronic volume overload and increase in peripheral resistance. Impairment of cardiac function has been reported to correlate with body mass index (BMI) and duration of obesity, with most studies reporting abnormal diastolic function. **Aims and Objectives:** The aim of the study was to assess the effect of obesity on the left and right ventricular (RV) function by conventional two-dimensional (2D) echocardiography in non-hypertensive, nondiabetic healthy obese individuals. **Materials and Methods:** A total of 60 healthy subjects aged between 18 and 30 years, without any comorbid conditions for CVD such as hypertension, diabetes mellitus, and chest pain were enrolled in the study. Demographic data including height, weight, and blood pressure were collected from all the subjects through a detailed medical history and physical examination. 2D echocardiography was performed using Philips iE33 (Andover, MA, USA) echo machine by a trained sonographer who is blinded to the study. **Results:** Obesity has an impact on left ventricular (LV) function as there is a significant increase in Tei index for LV ($P < 0.001$) and LVEDV ($P = 0.046$). Obesity has no effect on RV function as there is no change in tricuspid annular plane systolic excursion ($P = 0.628$) and Tei index for RV ($P = 0.682$). Obesity has no effect on LV ejection fraction ($P = 1.00$). LV mass left atrial (LA) diameter, septal wall diameter (IVSd), and posterior wall diameter are significantly increased with an increased BMI. **Conclusion:** The present study concluded that obese individuals with BMI ≥27.5 kg/m^2 have increased LV wall thickness and LV mass, significantly higher risk of LV diastolic dysfunction. Although LA enlargement is associated with diastolic dysfunction which is an evidence of subclinical LV systolic dysfunction, we found no change in LVEF in all the groups which indicates that obesity does not have any effect on LV systolic function.

KEY WORDS: Obesity; Body Mass Index; Cardiac Function; Echocardiography

INTRODUCTION

Obesity constitutes a major health issue in the modern world because of its association with morbidity, mortality, and cardiovascular diseases (CVD).[1,2] As per the world health organization (WHO) estimates, the global prevalence of obesity has been increasing and projecting more than 1.9 billion people were overweight from adults aged eighteen years and older. The worldwide prevalence of obesity between 1980 and 2014 is more than doubled. Obesity has been associated with heart failure,[3,4] with a significantly higher risk of diastolic dysfunction.[5] The excess in body fat determines a rise in each preload and afterload leading to a hyperdynamic circulation, chronic volume overload, and increase in peripheral resistance.[6,7] Impairment of cardiac function has been reported to correlate with body mass index

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(BMI) and duration of obesity,[8,9] with most studies reporting abnormal diastolic function.

Hemodynamically, in obesity, an increase in total blood volume is seen along with increased cardiac output, which in turn is fueled by increased metabolic demand due to increased body weight. Due to incremental increase in the left ventricular (LV) filling pressure and volume in obesity, a shift to left is seen in the "Frank–Starling curve." This overtime leads to dilatation of the chamber which causes increased wall stress, subsequently leading to ventricular hypertrophy of eccentric type. As a result, LV dilation and increased LV mass are frequent findings in individuals with obesity,[10-12] with both eccentric and concentric LV geometric patterns described in these conditions. LV diastolic dysfunction might represent one of the pathophysiological links between an increase in body weight and an increased incidence of heart failure.

The role of obesity as an independent predictor for LV hypertrophy (LVH), left atrial (LA) enlargement, and subclinical impairment of LV systolic and diastolic function as well as microvascular cardiac changes is not fully established. Hence, the present study sought to determine a link between echocardiographically demonstrated subclinical cardiac impairment and increasing BMI in non-hypertensive, nondiabetic healthy individuals without any other cardiac comorbidities.

MATERIALS AND METHODS

Study Design

This is a prospective, cross-sectional study conducted at our institute in the department of physiology from August 2018 to September 2018.

This study was approved by the Institutional Ethics Committee of Santhiram Medical College and General Hospital (IEC/2018/003). Written informed consent was obtained from all the study participants before enrolling into the study.

Inclusion Criteria

Healthy subjects aged between 18 and 30 years, without any comorbid conditions for CVD such as hypertension, diabetes mellitus, and chest pain were enrolled in the study.

Exclusion Criteria

Subjects aged below 18 years and above 30 years, subjects with other comorbid conditions such as diabetes mellitus and hypertension, history of CVD were excluded from the study.

Study Population

The study participants were divided into three weight groups according to the criteria suggested by the WHO expert consultation: Normal weight (BMI <23.0 kg/m^2), overweight (BMI: 23.0–27.4 kg/m^2), and obese (BMI ≥ 27.5 kg/m^2).[8] BMI was calculated as weight (kg) divided by height squared (m^2). Twenty subjects were recruited in each group (n = 60).

Assessment

Demographic data including height, weight, and blood pressure were collected from all the subjects through a detailed medical history and physical examination.

Echocardiographic Analysis

Two-dimensional echocardiography was performed using Philips iE33 (Andover, MA, USA) echo machine by a trained sonographer who is blinded to the study. All the echo tests were performed by a single operator to avoid intraobserver variability. LV diameter was measured in the parasternal short-axis view as recommended by the American Society of Echocardiography.[9] The LV mass was calculated using simple geometric cube formula[10] and indexed by both for body surface area and height. The LV ejection fraction was calculated by the biplane modified Simpson's method.

LV diastolic function was evaluated using mitral inflow velocity and mitral annular velocity. Peak E and A velocity of the mitral inflow were measured from an apical four-chamber view, and then E/A ratio was calculated. The mean value of E' velocities measured by tissue Doppler imaging from septal and lateral annulus was calculated, and E/E' ratio was used as an indicator of LV filling pressure.

Statistical Analysis

Data was collected on Microsoft Excel spreadsheets. Data was expressed as Mean±SD and frequencies with percentages for continuous variables and categorical variables, respectively. Differences observed were tested for significance with analysis of variance for comparison of means and Chi-square test for comparison of proportions. $P \leq 0.05$ was considered as statistically significant. Statistical analysis was performed using SPSS version 20.0 (IBM, Armonk, NY, USA).

RESULTS

A total of 60 healthy male medical students were studied in the present study. Female subjects were not included in the study as we do not have female echocardiographer. They were divided into three groups – normal weight group, over-weight group, and obese group (20 subjects per group) according to their BMI. Demographic details including age, height, weight, BMI, systolic blood pressure (SBP), and diastolic blood pressure (DBP) are tabulated in Table 1. All the three groups are similar (P = not significant) with respect to age, sex, SBP, and DBP, whereas there is a significant difference in weight and BMI among all the three groups ($P < 0.0001$).

Echocardiographic parameters comparing the three groups revealed correlation between increasing BMI and increased LV mass ($P < 0.001$), increased LA diameter ($P < 0.01$), increased septal wall diameter ($P < 0.001$), and increased posterior wall diameter (PWd) ($P = 0.03$). However, there was no significant change in LV ejection fraction ($P = 1.00$), LV end diastolic dimension (LVEDD) ($P = 0.079$), and LV end systolic dimension (LVESD) ($P = 0.083$) [Table 2].

We found no change in LV filling pressures – E velocity ($P = 0.61$), A velocity ($P = 0.60$), E/A ratio ($P = 0.98$), E' ($P = 0.67$), E/E' ($P = 0.15$), and deceleration time ($P = 0.16$) [Table 3 and Figure 1].

Higher BMI was associated with higher Tei index for LV ($P < 0.001$) and LVEDV ($P = 0.046$). No significant changes were found in Tei index for RV ($P = 0.682$) and LVESV

Table 1: Demographic details among the three groups

Variable	Normal weight (*n*=20)	Overweight (*n*=20)	Obese (*n*=20)	*P*-value
Age (in years)	20.3±0.7	21.2±0.5	20.8.3±0.9	0.64
Male (%)	100	100	100	NS
Height (in meters)	1.76±0.03	1.76±0.03	1.75±0.01	0.77
Weight (in kg)	68.86±5.33	78.22±5.29	98.10±12.90	<0.001*^
BMI (kg/m^2)	22.15±1.20	25.18±1.11	31.81±4.18	<0.001*^
Systolic BP (mm of Hg)	121.95±2.30	122.54±1.84	119.20±1.5	0.79
Diastolic BP (mm of Hg)	82.30±1.21	81.48±0.98	81.24±1.17	0.81

BP: Blood pressure. *Indicates significant *P* value between normal weight and obese. ^Indicates significant *P* value between overweight and obese. BMI: Body mass index

Table 2: Comparison of echocardiographic parameters

Variable	Normal Weight (*n*=20)	Overweight (*n*=20)	Obese (*n*=20)	*P*-value
LV mass (g)	159.85±24.55	188.75±31.50	196.40±27.18	<0.001*^
LA diameter (mm)	31.83±2.19	33.89±2.10	34.97±2.38	<0.01*^
IVSd (mm)	10.28±1.38	11.45±1.10	11.72±1.21	<0.001*^
PWd (mm)	10.52±1.23	10.89±1.04	11.56±1.51	0.03^
LVEDD (cm)	43.16±3.98	45.76±3.72	45.69±3.04	0.079
LVESD (cm)	26.78±6.20	29.82±2.71	29.79±2.89	0.083
LV ejection fraction (%)	64.03±4.03	64.41±3.40	64.66±3.66	1.00

LV: Left ventricle, LA: Left atrium, IVSd: Septal wall diameter, PWd: Posterior wall diameter, LVEDD: Left ventricular end-diastolic dimension, LVESD: Left ventricular end-systolic dimension. *Indicates significant *P* value between normal weight and obese. ^Indicates significant *P* value between overweight and obese

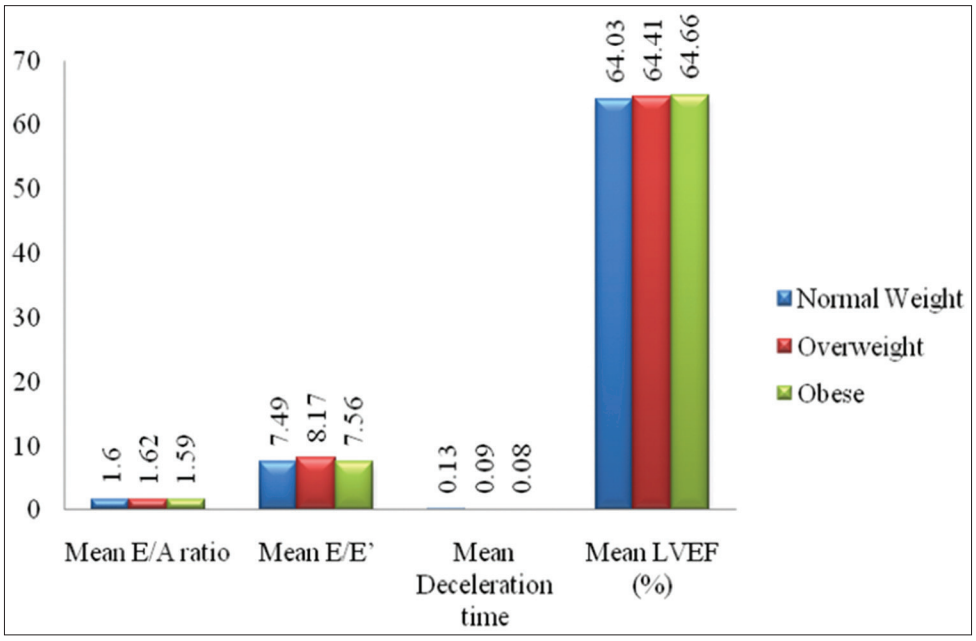

Figure 1: Comparison of echocardiographic parameters. LVEF: Left ventricular ejection fraction

($P = 0.696$). There is no significant change in tricuspid annular plane systolic excursion (TAPSE) ($P = 0.628$) [Figure 2].

LV and LA Morphology

Increasing BMI showed increase in septal wall thickness ($P < 0.001$), posterior wall thickness ($P = 0.03$), LV mass ($P < 0.01$), and LA diameter ($P = 0.0274$) among the obese group compared to the normal and overweight weight groups. There were no significant changes in LVEDD ($P = 0.079$) and LVESD ($P = 0.083$) [Figure 3].

LV Systolic Function

There was no significant difference in ejection fraction ($P = 0.6769$) across the three groups [Figure 4].

LV Diastolic Function

Our findings revealed that there is a significant rise in Tei index for LV ($P < 0.001$) and LVEDV ($P = 0.046$) with increased BMI, whereas we found no significant change in LV filling pressures – E velocity ($P = 0.61$), A velocity ($P = 0.60$), E/A ratio ($P = 0.98$), E' ($P = 0.67$), E/E' ($P = 0.15$), and deceleration time ($P = 0.16$) [Table 3 and Figure 1].

RV Function

There was no significant change in TAPSE ($P = 0.628$) and Tei index for RV ($P = 0.682$).

DISCUSSION

The present study results demonstrate subclinical changes in the LV structure and as well LV function in obese healthy subjects whose BMI ≥27.5 kg/m^2 and who do not have any other comorbid conditions or risk factors for cardiac disease. Echocardiographic observations showed an increase in septal wall thickness, posterior wall thickness, and LV mass in obese group as compared to the normal weight group which indicates a significant increase in LV size and LVH. Cardiac output will get increased in obese subjects to meet their raised metabolic requirements. The increase in blood volume will increase venous return to both right and left ventricles, ultimately producing dilation of these cardiac chambers, increasing wall tension. This ends up in LVH that is in the course of decrease in diastolic chamber compliance, finally resulting in a rise in LV filling pressure and LV enlargement. As long as LVH adapts to LV chamber enlargement, systolic function is preserved. When LVH fails to keep pace with progressive LV dilation, wall tension increases even more and

Table 3: Comparison of left ventricular filling pressures

Variable	Normal Weight (*n*=20)	Overweight (*n*=20)	Obese (*n*=20)	*P*-value
E velocity (cm/s)	99.37±17.25	103.40±14.54	98.46±18.07	0.61
A velocity (cm/s)	62.75±10.40	65.47±12.60	67.03±16.86	0.60
E/A ratio	1.60±0.25	1.62±0.29	1.59±0.62	0.98
E' (cm/s)	13.36±1.76	12.77±2.01	13.21±2.14	0.67
E/E'	7.49±1.29	8.17±0.90	7.56±1.33	0.15
Deceleration time	0.13±0.15	0.09±0.02	0.08±0.02	0.16

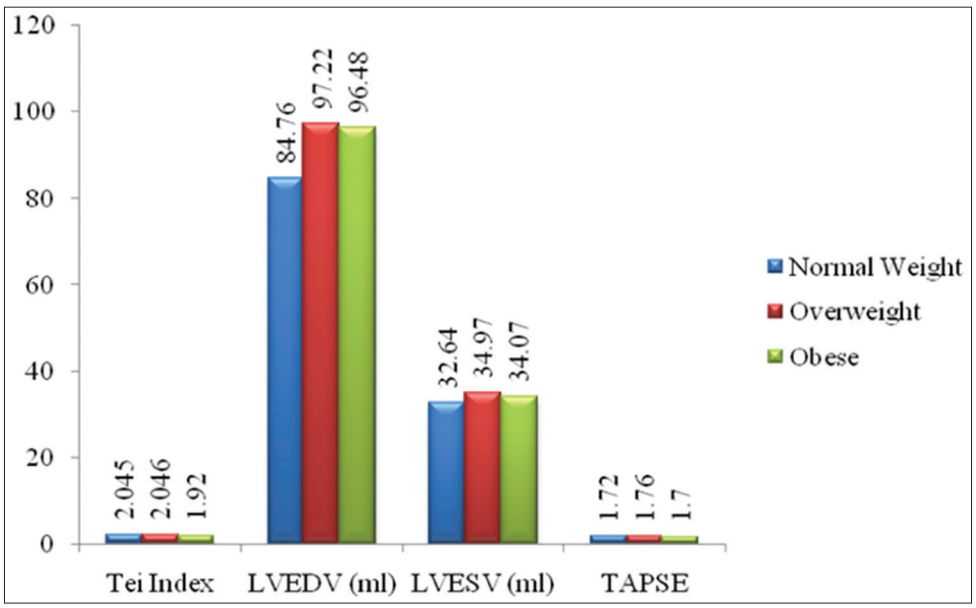

Figure 2: Comparison of parameters related to right ventricle function. LVEDV: LV end-diastolic volume; LVESV: LV end-systolic volume; TAPSE: Tricuspid annular plane systolic excursion

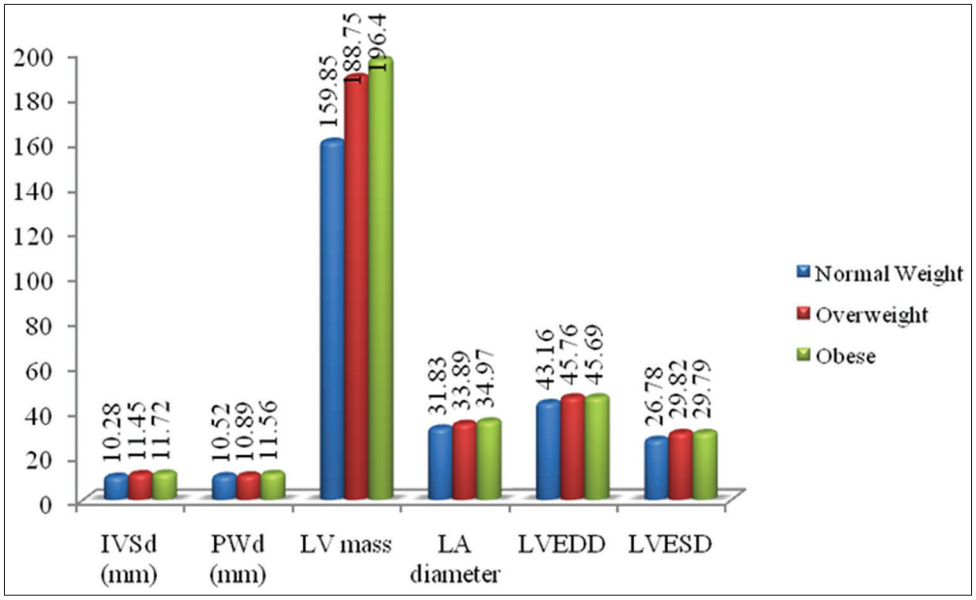

Figure 3: Left ventricular and left atrial morphology. IVSd: Septal wall diameter; PWd: posterior wall diameter; LV: Left ventricle; LA: Left atrium; LVEDD: Left ventricle end-diastolic diameter; LVESD: Left ventricle end-systolic diameter

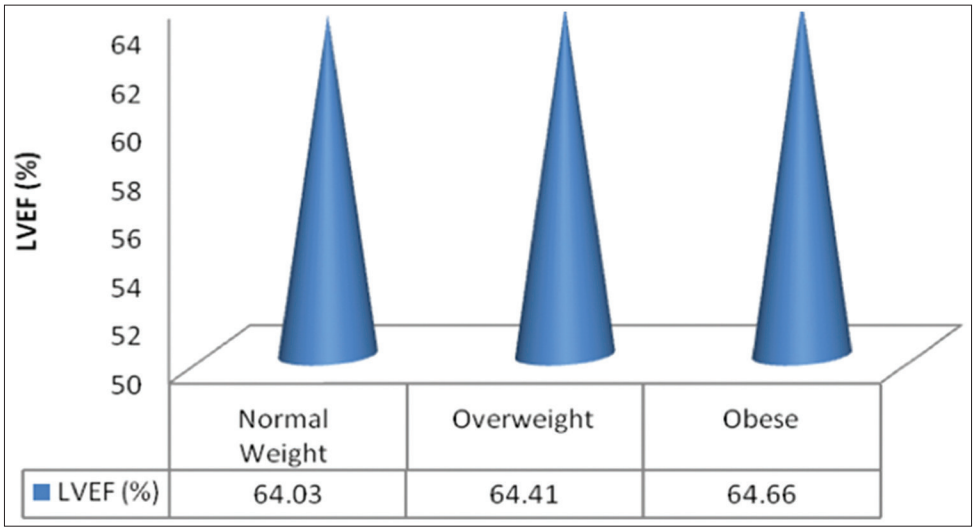

Figure 4: Comparison of LVEF. LVEF: Left ventricular ejection fraction

systolic dysfunction may ensue.[7] The present study results show no significant changes in the indices for LV ejection fraction in the obese group as compared to the normal and overweight groups, which indicates preservation of systolic function.[13]

The limitation with conventional methods such as LVEF and fractional shortening, which are relatively less sensitive and thus not able to pick up early preclinical changes, hence, the more sensitive, newer echo techniques and strain imaging are recommended to demonstrate the presence of subclinical LV changes in young obese subjects. We found a statistically significant increase in Tei index for LV and LVEDV which are indicative for LV diastolic dysfunction with increased BMI. This indicates obesity is associated with LV diastolic dysfunction. We also found a significant increase in LA diameter in obese subjects as compared to normal-weight individuals. The association of LA enlargement with decreased diastolic function is more consistent with current understanding of the left atrium as a biomarker of filling pressure.[14] Right ventricular (RV) dysfunction in relation with obesity has been studied and found that obesity has no effect on RV dysfunction as there is no change in TAPSE and Tei index for RV.

In a study conducted by Russo *et al*.,[5] 250 subjects grouped according to increasing BMI found that BMI was independently associated with higher E, A, and E/E'. Overweight and obese subjects had lower E' (both $P < 0.01$) and higher E/E' ($P < 0.01$) when compared to normal-weight subjects. Compared to normal-weight subjects E/A was lower in obese ($P < 0.01$). However, in our study, we found statistically no significant change in LV filling pressures which includes E velocity, A velocity, E', and E/E' ratio. In

another study, Kossaify and Nicolas[15] evaluated LV diastolic function in 99 patients sub-grouped as per their BMI revealed that values of LV mass, LV mass index, and septal wall thickness (IVSd) were considerably superior in overweight/obese groups compared to normal weight group. TDI showed a significantly lower E′ in overweight/obese groups compared to the E′ in the normal BMI group.

Obesity has impact on LV function as there is a significant increase in Tei index for LV ($P < 0.001$) and LVEDV ($P = 0.046$). Obesity has no effect on RV function as there is no change in TAPSE ($P = 0.628$) and Tei index for RV ($P = 0.682$). Obesity has no effect on LV ejection fraction ($P = 1.00$). LV mass, LA diameter, septal wall diameter (IVSd), and PWd are significantly increasing with an increased BMI. There is no change in LV filling pressures – E Velocity ($P = 0.61$), A velocity ($P = 0.60$), E/A ratio ($P = 0.98$), E' ($P = 0.67$), E/E' ($P = 0.15$), and deceleration time ($P = 0.16$).

Limitations of the Study

This is a single center study and sample size is relatively small. Hence, we may not generalize these findings.

CONCLUSION

The present study concluded that obese individuals with BMI ≥27.5 kg/m^2 have increased LV wall thickness and LV mass, significantly higher risk of LV diastolic dysfunction. Although LA enlargement is associated with diastolic dysfunction which is an evidence of subclinical LV systolic dysfunction, we found no change in LVEF in all the groups which indicates that obesity does not have any effect on LV systolic function. We did not find RV dysfunction in obese group as there is no change in TAPSE.

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