ASSESSMENT OF MYOCARDIAL MECHANICS IN OVERWEIGHT AND OBESE INDIAN SUBJECTS

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Abstract : Obesity is associated with increased cardiovascular morbidity and mortality. A direct effect of isolated obesity on cardiac function is not well established. The study was designed to determine the direct effect of various grades of isolated obesity on Echocardiographic indices of systolic and diastolic left ventricular function. Forty five overweight & obese and 30 normal weight, serving personnel without any other pathological condition were studied. Group I (n=23) consisted of subjects with normal weight and body mass index (BMI 18.5-22.9 kg/m^2), Group II (n=28) of overweight subjects (BMI 23-24.9 kg/m^2) and Group III (n=24) of obese subjects (BMI ≥25 kg/m^2). Echocardiographic indices of systolic and diastolic function were obtained and dysfunction was assumed when at least two values differed by ≥2 SD from the normal weight group. Ejection fraction was increased (p=0.001) in group II and III however fractional shortening was increased significantly in group III (<0.001). Left ventricular dimensions (EDD & ESD) were increased (P<0.001, 0.002) but relative wall thickness was unchanged in group II & III. Systolic dysfunction was not observed in any of the obese patients. The deceleration time was increased (P<0.01) in overweight and obese subjects compared to normal group individuals. No difference was found between obesity subgroups. Subclinical diastolic dysfunction in the form of reduced E/A ratio and increased deceleration time was more prevalent among obese subjects. BMI correlated significantly with indices of left ventricular systolic and diastolic function. Subclinical left ventricular diastolic dysfunction was noted in all grades of obesity which correlates with BMI.

Key words : obesity   systolic function
                      diastolic function   echocardiography

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INTRODUCTION

The dramatic increase in the prevalence of obesity and its strong association with cardiovascular disease has resulted in unprecedented interest in understanding the effects of obesity on the cardiovascular system. Many studies have evaluated left ventricular (LV) systolic function in obesity. The findings of these studies are quite variable. Various authors have reported depressed LV ejection fraction (EF) (1), normal EF (2, 3, 4, 5), and supernormal EF (6, 7) in obese subjects. The prevalence of obesity is increasing in the developing world. Obesity is associated with cardiomyopathy, resulting in heart failure in severe obesity cases (8, 9). This has been attributed to chronic volume overload characterized by left ventricular (LV) dilation, increased left ventricular wall stress and compensatory (eccentric) left ventricular hypertrophy (9). Impairment of cardiac function has been reported to correlate with degree of obesity i.e. body mass index (BMI) and duration of obesity (10, 11). Abnormal diastolic function is the most important component of the impaired cardiac function (12), while systolic dysfunction is not so common (9, 10, 11, 12, 13).

Obesity has also been linked to a spectrum of minor reversible cardiovascular changes, ranging from a hyper dynamic circulation to subclinical cardiac morphological changes in the form of greater aortic root and left atrial enlargement (7, 8). Abnormal cardiac functions are noted in individuals even with slight or mild obesity (14). The abnormal cardiac function in association with obesity may reflect the role of co-morbidities like hypertension, diabetes, coronary artery disease and obstructive sleep apnea. However individuals with isolated obesity have altered loading of the ventricles due to increased stroke volume and cardiac output leading to cardiac dysfunction (7, 8, 9, 10, 11).

Echocardiography has consistently been the most accurate non-invasive method of assessing the left ventricular function (15). The relation between obesity (assessed by BMI) and alterations in cardiac function, as well as the impact of different grades of obesity on cardiac structure and function is less well documented.

The aim of this study was to determine the direct effect of different grades of isolated obesity on echocardiographic indices of systolic and diastolic left ventricular function. The relation between obesity (assessed by BMI) and alterations in cardiac function, as well as the impact of different grades of obesity on cardiac structure and function is less well documented. The aim of this study was to determine the direct effect of overweight and obesity on Echocardiographic and Doppler indices of left ventricular systolic and diastolic function.

METHODS

The present study was conducted at Department of Physiology/Cardiology, GSL Medical College & General Hospital, Rajahmundry. 100 healthy non-diabetic, normotensive male individuals reporting for medical examination under regular health check-up scheme from rural and urban coastal Andhra were included in the study population. Written informed consent was taken from the entire participant. Prior

institutional ethical approval for the study was taken.

Participants were instructed to empty their bladder prior to anthropomorphic measurements. A trained person was deputed from the physiology department to take the anthropometric measurements of participants while they stand in light clothing without shoes, using a standard protocol. Height was measured to the nearest 0.1 cm using a free-standing stadiometer. Weight was measured to the nearest 100 g using digital scales. Body mass index (BMI) was calculated from weight (Kg) divided by the square of height in meter (m^2). A D-loop non stretch fiberglass tape was used for the waist circumference measures. Waist circumference was measured at the smallest circumference between the costal margin and the iliac crest to the nearest 0.1 cm while the participant in standing with the abdomen relaxed, at the end of a normal expiration. Where there was no natural waistline, the measurement was taken at the level of the umbilicus. Hip circumference was measured at the maximum circumference between the iliac crest and the crotch while the participant in standing and recorded to the nearest 0.1 cm. Waist-hip ratio was then calculated from the waist and hip measures (waist circumference/hip circumference).

A 12-lead electrocardiogram (ECG) was obtained. Hematological and biochemical variables including glucose, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, urea and uric acid were determined from fasting blood samples. Revised BMI cut-off for Asians as recommended by WHO was considered to classify overweight and obesity (34). The participants

in the study were classified into three groups based on the BMI: Group I (n=23) consisted of subjects with normal weight and body mass index (BMI 18.5-22.9 kg/m^2), Group II (n=28) of overweight subjects (BMI 23-24.9 kg/m^2) and Group III (n=24) of obese subjects (BMI $\geq$25 kg/m^2). Patients suffering from hypertension, diabetes mellitus, coronary artery disease or dyslipidemia were excluded from the study. Participants with normal ECG without any acute or chronic disease, medication that could affect heart were included.

A cross sectional echocardiogram was performed in all participants (Sonos 5500, Hewlett-Packard, Palo Alto, California, USA). Echocardiograms included cross sectional, M mode, 2D and Doppler studies (9, 10). The following indices of cardiac function was evaluated: Left ventricular systolic function: Left ventricular end diastolic diameter (EDD), end systolic diameter (ESD) and fractional shortening (FS) was obtained in parasternal long axis views using M mode. The relative wall thickness (RWT) was calculated from the posterior wall thickness (PWT) and the EDD, as ($2 \times$ PWT)/EDD. Left ventricular diastolic function: Doppler measurements were obtained in the apical four chamber view. The following variables were calculated: maximum velocity of passive mitral filling (E), maximum velocity of active mitral filling (A), ratio of passive to active velocity (E/A), deceleration time (DT) and isovolumic relaxation time (IVRT). Sub-clinical dysfunction was assumed when two or more indices of altered diastolic or systolic functions were present.

Statistical methods

Descriptive statistics were done on each

of the variables to obtain the frequency distributions. Quantitative variables were presented as mean±SD. Comparison of data between the overweight & obese groups was performed by '*t*' tests. Analysis of variance (one-way ANOVA) was used to compare the data between obese subgroups. Correlations between clinical variables and left ventricular function were determined by Pearson correlation analysis. A probability value of P<0.05 was considered significant.

RESULTS

We studied 52 overweight (n=28) and obese (n=24) men with mean age of 38.9±10.4 years, 36.6±9.8 (range 25–52 years) respectively and 23 normal weight controls men with mean age of 36.3±7.6 years (range 25–52 years). The characteristics of the samples studied are presented in Table I. Only weight and BMI were significantly different within the obese subgroups, with respect to the normal weight group. The measured indices of left ventricular systolic function are presented in Table II.

Left ventricular dimensions (EDD, ESD) were significantly increased in overweight & obese personnel compared to normal weight (P<0.001 & P=0.002), though relative wall thickness was almost similar to control group. The contractility index (ejection fraction) was significantly higher (P<0.001) in the overweight & obese personnel than in the controls. The changes in percentage fractional shortening (FS %) was observed only in obese groups and it was significantly higher (P<0.001) in obese group (44.9±6.3) than control (40.8±8.1) and overweight individuals (38.6±7.7). In the prevalence analysis, no obese patient met the criteria for systolic dysfunction. Overall there was improved mechanical performance in the overweight and early obese group individuals. The measured indices of left ventricular diastolic function are presented in Table II.

In obese subjects deceleration time was significantly increased (202.2±25.3) compared to normal (173.6±13.9) and overweight individuals (191.0±19.4) (P<0.01). Subgroup

TABLE I : Baseline characteristics of different study populations.

Parameters	Group I Normal (n=23)	Group II Overweight (n=28)	Group III Obese (n=24)	P value
Age (years)	36.3±7.6	38.9±10.4	36.6±9.8	NS
Weight (kg)	65.8±6.6	75.1±9.1	90.5±11.4	< 0.001
Height (m)	1.66±0.11	1.65±0.09	1.62±0.05	NS
BMI (kg/m2)	21.2±2.1	24.1±1.8	29.3±5.9	< 0.005
HR(beats/minute)	77±10	79±9	76±9	NS
SBP (mmHg)	116±7.7	120±4.9	122±11.8	NS
DBP (mmHg)	68.5±9.8	78.6±8.8	84.2±6.5	NS
RPP	9098±456	9318±663	9340±878	0.0473

Data presented as mean±SD. P<0.005 = significant, P<0.001 = highly significant. BMI = body mass index, HR = Heart rate, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, RPR = Rate Pressure Product (SBP x HR).

Group I = Subjects with normal weight and BMI 18.5-22.9 kg/m^2,
Group II = Overweight subjects with BMI 23-24.9 kg/m^2 and
Group III = Obese subjects with BMI ≥25 kg/m^2.

TABLE II : Left ventricular function based on body mass index.

Parameters	Group I Normal (n=23)	Group II Overweight (n=28)	Group III Obese (n=24)	P value
EDD(mm)	41.4±5.61	45.9±6.5#	50.9±5.5$^{\alpha,\beta}$	<0.001
ESD(mm)	24.2±5.1	28.6±3.7#	29.1±3.8$^{\alpha}$	0.002
EF%	66.4±10.3	77.4±8.1#	75.6±11.8$^{\alpha}$	0.001
FS%	40.8±8.1	38.6±7.7	44.9±6.3$^{\alpha,\beta}$	<0.001
RWT	0.38±0.05	0.39±0.04	0.40±0.08	0.60
E (cm/sec)	77.7±13.9	78.1±15.3	79.0±16.1	0.92
A (cm/sec)	52.6±9.93	56.4±10.2	58.0±12.5	0.58
E/A	1.51±0.3	1.43±0.4	1.15±0.4$^{\alpha,\beta}$	0.015
DT (ms)	173.6±13.9	191.0±19.4#	202.2±25.3$^{\alpha,\beta}$	<0.01

Data presented as mean±SD. #: Group I vs Group II; α : Group I vs Group III; β : Group II vs Group III, *P<0.05; for differences between subgroups. E: maximum velocity of passive mitral filling; A : maximum velocity of active mitral filling; DT : Deceleration time; EDD: end diastolic diameter; ESD : end systolic diameter; EF : ejection fraction; FS: fractional shortening; RWT: relative wall thickness.

Group I = Subjects with normal weight and BMI 18.5-22.9 kg/m^2,
Group II = Overweight subjects with BMI 23-24.9 kg/m^2 and
Group III = Obese subjects with BMI ≥ 25 kg/m^2.

TABLE III : Correlations between the echocardiographic variables and left ventricular structure and function.

Variables	BMI (kg/m^2)		Age (years)	
	r	P	r	P
Variables of LV Structure and Systolic function				
EDD	0.19	0.056	0.14	0.069
ESD	0.14	0.069	0.17	0.062
EF%	0.283*	0.041	−0.05	0.78
FS%	−0.067	0.166	0.001	1.68
RWT	0.19	0.059	0.07	1.067
Variables of LV Diastolic Function				
E (cm/s)	−0.62	1.08	−0.38*	0.0231
A (cm/s)	0.02	1.096	0.20	0.051
E/A	−0.61	0.0014	−0.48**	0.00016
DT (ms)	0.52**	0.0012	0.14	0.069
IVRT (ms)	−0.19	0.65	0.13	0.078

Data presented as mean±SD. *P<05 = significant, **P<0.001 = highly significant.

EDD: end diastolic diameter; ESD: end systolic diameter; EF: ejection fraction; FS: fractional shortening; RWT: relative wall thickness.

E: maximum velocity of passive mitral filling; A: maximum velocity of active mitral filling;

DT: Deceleration time; IVRT: Interventricular relaxation time.

analysis showed significant differences among the overweight and obese subgroups for these variables. The value of early transmitral filling (E) was almost similar in all the three subgroups but late filling (A) was augmented in obese & overweight individuals resulting in significant decrease in E/A ratio (P=0.015) in obese group compared to normal and overweight groups. In the prevalence assessment, sub-clinical diastolic dysfunction was significantly more prevalent among obese patients than in the control group. The correlations between echocardiographic variables and left ventricular structure and function are shown in Table III. Among the indices of systolic function, EF% correlated positively BMI and negatively with age however FS% correlated negatively with BMI and positively with age but is not significant. Among the indices of diastolic function, BMI correlated negatively with early transmitral filling velocity (E), E/A ratio, IVRT & positively with late filling velocity (A), deceleration time (DT). Similarly, age correlated positively with A, IVRT & DT and negatively with E and E/A ratio.

DISCUSSION

Overweight and obesity are the most common nutritional disorders and this has heightened our concern given the strong association between obesity and cardiovascular morbidity (8). In the present study we have made an attempt to assess the effect of overweight and obesity on the left ventricular myocardial mechanics. No subject with isolated obesity presented with sub-clinical systolic dysfunction. On the contrary, there was a significant increase in the ejection fraction and in the percentage of

fractional shortening, but this only reached significance in obesity groups compared with the controls. 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 (27).

However, the normality of the ejection fraction was in accordance with the normal relative wall thickness in our obese groups, which indicates that systolic function was preserved. These findings imply that in the overweight groups and lesser degrees of obesity there is a compensatory increase in systolic function, which has not yet reached the stage of cardiac deterioration. Most echocardiographic studies using measurements of the ejection phases to evaluate systolic function in obese subjects have shown normal results (2, 3, 5, 17, 24) or supernormal results (6, 9, 23) that is well matched with our results of current study. Tanalp AC et al (23) reported that ejection phase indices were increased in mildly and moderately obese subjects but diastolic function was shown to be impaired in the moderately and significantly obese subjects when measured by tissue Doppler imaging. These observations (6, 9, 23) are very much in accordance with our findings. However some studies observed reduced mechanical performance with increased duration of obesity (25, 29, 30) and loss of weight leads to an improved functional status, reduction of left ventricular remodeling and an increase of the ejection fraction (32).

Obert P et al (29) observed that isolated severe obesity in adolescents, at a preclinical

stage, is associated with changes in myocardial deformation and torsional mechanics that could be in part related to alterations in relaxation and contractility properties of subendocardial fibers. Alpert et al (26) and Peterson LR et al (30) have argued that duration of obesity is the factor that determines the likelihood of developing

systolic dysfunction and heart failure and observed that these early abnormalities in LV structure and function may have important implications for explaining the myocardial dysfunction that is associated with increased cardiovascular morbidity and mortality caused by obesity. Left ventricular systolic function is affected late in the course of obesity and more so in patients with considerable degree of obesity (8, 11). However Wong CY et al (31) reported that overweight subjects without overt heart disease have subclinical changes of LV structure and function even after adjustment for mean arterial pressure, age, gender, and LV mass. This is not in accordance with our study but duration of overweight may be responsible for reduced LV structure and performance as explained by other investigators (25, 29, 30).

In our study alterations in the left ventricular diastolic function were more frequent with increasing obesity. Similar finding has been noted in other studies and suggests an abnormal relaxation of the left ventricle and there is increased dependency on left atrial contraction for normal filling (18, 19, 20, 33). Similarly, the deceleration time was significantly prolonged in the obese subjects and correlated inversely with BMI. IVRT was similar in obese subjects and controls, and did correlate with BMI, this is in contrast to previously published data in which this variable has been found to be prolonged in both moderate and gross obesity (5, 17). The E, A and E/A values did not differ across the spectrum of obesity. The associations of these indices with obesity reported in previous studies have been variable. Some studies have reported a decrease in the maximum velocity of passive mitral filling (E) in obese individuals (17), while others have found no significant change in it (19, 20). However in both these studies E/A ratio was decreased, in the former due to decrease in the E velocity and in later study due to increase in the active mitral filling (A) velocity with unchanged E velocity. Our findings matched almost completely with the observations of Di Bello V et al (19) and Chadha DS et al (28).

Doppler method is a good way of assessing diastolic function but when volume overload is present, as seen in obesity, normal values may result, as the increase in left atrial pressure caused by intravascular volume can mask the alterations observed in the early phases of abnormal diastolic relaxation (9, 10, 16). In contrast to systolic function, we found that alterations in diastolic function were common, not only in the severely obese personnel but also in overweight subjects. These alterations in diastolic function correlated strongly with BMI. In obesity, cardiac adaptation to chronic volume overload is associated with eccentric hypertrophy and abnormalities of diastolic function from the initial stages, indicating

that structural changes and an obesity cardiomyopathy are present in all obese individuals (9, 11, 14, 28). In the present study LV mass and LV geometry was not assessed hence it is difficult to say whether the alteration of the diastolic function is due to morphological change or represents a mere functional change.

A greater awareness of weight control is required to induce beneficial changes in cardiac function. The alterations that occur in obesity can be reversed easily and quickly by weight loss, to the long term benefit of the patient (21, 22). To conclude, all patients with overweight and obesity have sub-clinical left ventricular diastolic dysfunction, which correlates with BMI however they have an increased contractile performance in the early stages of obesity.

Limitations of the study

The study sample size was small. Obesity was measured using only BMI, and no measurements of body fat distribution were made. In view of higher prevalence of abdominal obesity in our country correlating the echocardiographic variables with anthropometric markers of abdominal obesity could have provided additional information. Conventional measures of LV function assessment (mitral inflow velocities, isovolumetric ventricular relaxation times and ejection fraction) used in the present study are load dependent and hence may show inconsistent changes. Newer echocardiography technique such as tissue doppler which is less load dependent may be better tool for assessing LV function in obese individuals.

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