

Impact of Body Mass Index on Left Ventricular Function

Wg Cdr DS Chadha*, Wg Cdr A Swamy⁺, Col SK Malani[#], Air Vice Mshl (Retd) RK Ganjoo AVSM, VSM**,
Lt Gen OP Mathew, AVSM, SM⁺⁺

Abstract

Background: 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.

Methods: Fifty one obese and 25 normal weight, serving personnel without any other pathological condition were studied. Group I (n=25) consisted of subjects with normal weight and body mass index (BMI <25kg/m²), Group II (n=34) of overweight subjects (BMI 25-29.9 kg/m²) and Group III (n=17) of obese subjects (BMI >30 kg/m²). 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. **Result:** Ejection fraction, fractional shortening were increased (p<0.05) in Group II and III. Left ventricular dimensions were increased (p< 0.001) but relative wall thickness was unchanged. Systolic dysfunction was not observed in any of the obese patients. The mitral valve pressure half time (p< 0.01), left atrial diameter (p < 0.01) and the deceleration time were increased (p< 0.01) in obese subjects, while other diastolic variables were unchanged. No difference were found between obesity subgroups. Subclinical diastolic dysfunction was more prevalent among obese subjects. BMI correlated significantly with indices of left ventricular systolic and diastolic function.

Conclusion: Subclinical left ventricular diastolic dysfunction was noted in all grades of obesity which correlates with BMI.

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Key Words : Obesity; Systolic function; Diastolic function; Echocardiography

Introduction

The prevalence of obesity is increasing in the developing world. Obesity is associated with cardiomyopathy resulting in heart failure in severe obesity cases [1,2]. 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 [3]. Impairment of cardiac function has been reported to correlate with degree of obesity i.e. body mass index (BMI) and duration of obesity [4,5]. Abnormal diastolic function is the most important component of the impaired cardiac function [6], while systolic dysfunction is not so common [3-7]. 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 [1,2].

Abnormal cardiac functions are noted in individuals even with slight or mild obesity [8]. 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 [1-5]. Echocardiography has consistently been the most accurate non-invasive method of assessing the left ventricular function [9,10]. 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.

Material and Methods

The present study was carried out at the Air Force Central Medical Establishment New Delhi. Healthy non-diabetic, normotensive male service personnel reporting for medical examination constituted the study population. Informed consent was obtained from all the volunteers. After taking medical history, a detailed physical examination was conducted for all participants which included recording of height and weight. BMI was calculated by dividing the bodyweight (in kilograms) by the square of height (in meters).

*,⁺ Classified Specialist (Cardiology), Command Hospital (AF), Bangalore. [#]Senior Advisor, (Cardiology), Command Hospital (EC), Kolkata. ^{**}Ex-Commandant, Command Hospital (AF), Bangalore. ⁺⁺Commandant, Army Hosp (R&R), Delhi Cantt.

A 12-lead electrocardiogram (ECG) was obtained. Hematological and biochemical variables were determined from fasting blood samples. This included glucose, total cholesterol, triglycerides, high density lipoprotein cholesterol, low density lipoprotein cholesterol, urea, uric acid and full blood count.

Obesity was defined as a BMI of $\geq 30\text{kg/m}^2$, with clear evidence on physical examination of excessive subcutaneous adipose tissue. The participants in the study were classified into three groups based on the BMI: a normal weight (control) group had a BMI of $< 25\text{ kg/m}^2$, an overweight group was classified as a BMI between $25\text{--}29.9\text{ kg/m}^2$, and an obese group was classified as BMI $\geq 30\text{ kg/m}^2$. The number of participants in each group was 25, 34, and 17 respectively.

Patients suffering from hypertension, diabetes mellitus, coronary artery disease or dyslipidemia were excluded. Patients with normal ECG without any chronic or acute disease, not on medication that could affect heart and those not involved in competitive sports were included. A cross sectional echocardiogram was obtained on all participants (Sonos 5500, Hewlett-Packard, Palo Alto, California, USA). Echocardiograms included cross sectional, M mode and Doppler studies [9,10].

The following indices of cardiac function were evaluated:

Left ventricular systolic function : Left ventricular end diastolic (EDD), end systolic diameter (ESD) and fractional shortening (FS) were obtained in the parasternal long axis views using M mode. Pre ejection period (PEP) and ejection period (EP) was also recorded in all patients. 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 diastolic function : Pulsed doppler measurements were obtained in the apical four chamber view. The doppler beam was aligned perpendicular to the plane of the mitral annulus and a 5 mm pulsed wave doppler sample volume was placed between the tips of the mitral leaflets during diastole. 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), mitral valve pressure half time (MVPHT), deceleration time (DT) and isovolumic relaxation time (IVRT). The left atrial diameter was measured using M mode in the parasternal long axis view.

Table 1
Baseline characteristic of study population

	Group I Normal (n=25)	Group II Over weight (n=34)	Group III Obese (n=17)	p value (overall)
Age (years)	35.3 \pm 10.0	35.3 \pm 10.5	35.28 \pm 9.5	NS
Weight (kg)	69.3 \pm 7.3	78.1 \pm 10.1 ^a	93.5 \pm 13.5 ^{b,c}	< 0.001
Height (m)	1.62 \pm 0.07	1.64 \pm 0.07	1.62 \pm 0.05	NS
BMI (kg/m ²)	22.9 \pm 1.2	27.1 \pm 1.2 ^a	32.2 \pm 2.6 ^{b,c}	< 0.001
BMI (range)	16.7 - 24.7	25.8 - 29.9	30.1 - 35.9	-
HR (beats/minute)	78 \pm 10	75 \pm 9	80 \pm 9	NS

a: Group I vs Group II, b: Group I vs Group III, c: Group II vs Group III, *p < 0.05; for differences between subgroups BMI: body mass index, HR: heart rate, NS: non significant

A difference of more than 2 SD from the mean values of the normal weight group, was used to estimate the prevalence of cardiac functional abnormalities. Subclinical dysfunction was assumed when two or more indices of altered diastolic or systolic function were present.

Descriptive statistics were done on each of the variables to obtain the frequency distributions. Quantitative variables were described as mean. Comparisons between the obese group and the normal weight group were analyzed by *t* tests. Analysis of variance (one way ANOVA) was used to compare obese subgroups. Correlations between clinical variables and left ventricular function were determined by linear regression analysis. Probability values of $p < 0.05$ were considered significant.

Results

We studied 51 over weight and obese men with mean age of 35.3 ± 10.5 years, (range 26–52 years) and 25 normal weight controls with mean age of 35.3 ± 10.0 years (range 25–52 years). The characteristics of the patients studied are presented in Table 1. 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 2. A left ventricular diameter was significantly increased in obese personnel, though relative wall thickness was similar to control group. The contractility indices (ejection fraction and fractional shortening) were significantly higher in the obese personnel than in the controls. In the prevalence analysis, no obese patient met the criteria for systolic dysfunction.

The measured indices of left ventricular diastolic function are presented in Table 3. In obese subjects, MVPHT, left atrial diameter and deceleration time were significantly increased. Subgroup analysis showed significant differences among the over weight and obese subgroups for these variables. The values of E, A, and E/A ratio, were similar in all the three subgroups.

In the prevalence assessment, subclinical diastolic dysfunction was significantly more prevalent among obese patients ($p = 0.002$) than in the control group. It was found in seven (20.5%) individuals in the over weight group and eight (47%) individuals in the obese group.

The correlations between clinical variables and left ventricular function are shown in Table 4. Among the indices

Table 2**Indices of left ventricular systolic function**

	Group I Normal (n=25)	Group II Over weight (n=34)	Group III Obese (n=17)	p value (overall)
EDD (cm)	43.4 ± 5.61	45.7 ± 4.5 ^a	51.1 ± 3.5 ^{b,c}	< 0.001
ESD (cm)	25.2 ± 5.1	27.9 ± 3.7 ^a	28.1 ± 3.8 ^b	0.002
FS (%)	40.8 ± 18.1	38.6 ± 7.7	44.9 ± 6.3 ^{b,c}	< 0.001
EF (%)	65.7 ± 12.8	73.4 ± 9.1	74.6 ± 9.8 ^b	0.001
PEP/EP	0.31 ± 0.07	0.35 ± 0.07	0.38 ± 0.08	0.58
RWT	0.38 ± 0.05	0.39 ± 0.04	0.40 ± 0.08	0.60

a: Group I vs Group II, b: Group I vs Group III, c: Group II vs Group III, *p < 0.05; for differences between subgroups BMI: body mass index, EDD: end diastolic diameter; ESD: end systolic diameter; FS: fractional shortening; EF: ejection fraction; EP: ejection period; PEP: pre-ejection period; RWT: relative wall thickness.

Table 3**Indices of left ventricular diastolic function**

	Normal (n=25)	Over weight (n=34)	Obese (n=17)	p value (overall)
E (cm/s)	77.6 ± 13.9	78.0 ± 15.3	79 ± 16.1	0.92
A (cm/s)	52.6 ± 9.93	56.4 ± 10.2	58.0 ± 12.5	0.58
E/A	1.51 ± 0.3	1.43 ± 0.4	1.35 ± 0.3	0.15
DT (ms)	173.6 ± 13.9	191.0 ± 19.4 ^a	202.2 ± 25.3 ^{b,c}	< 0.01
MVPHT (ms)	63.7 ± 7.8	63.2 ± 11.0	70.0 ± 14.8 ^{b,c}	< 0.01
LAD (cm)	2.9 ± 0.4	3.3 ± 0.4 ^a	3.6 ± 0.42 ^{b,c}	< 0.01

a: Group I vs Group II, b: Group I vs Group III, c: 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; MVPHT: mitral valve pressure half-time; LAD: left atrial diameter

of systolic function, BMI correlated positively with %FS and PEP/EP while EP correlated positively with age.

Among the indices of diastolic function, BMI correlated positively with MVPHT, left atrial diameter and deceleration time. Similarly, age correlated positively with left atrial diameter and negatively with E and E/A.

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 [2]. In the present study we have made an attempt to assess the effect of different grades of obesity on the left ventricular function.

No patient with isolated obesity presented with subclinical 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. 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 groups with lesser degrees of obesity there is a compensatory

Table 4**Correlations between the clinical variables and left ventricular function**

Variable	BMI	Age
LV systolic function		
EF	0.23	- 0.05
FS	0.26*	0.00
PEP	0.25	0.00
EP	- 0.14	0.25*
PEP/EP	0.30*	- 0.14
LV diastolic function		
E	0.06	- 0.28*
A	0.02	0.20
E/A	0.01	- 0.38**
DT	0.32**	0.14
MVPHT	0.27**	- 0.13
IVRT	- 0.19	0.13
LAD	0.42**	0.34*

*p < 0.05; **p < 0.01; ***p < 0.001, EF: ejection fraction; FS: fractional shortening; PEP: pre-ejection period; EP: ejection period; E: maximum velocity of passive mitral filling; A: maximum velocity of active mitral filling; DT: deceleration time; MVPHT: mitral valve pressure half-time; IVRT: isovolumetric relaxation time; LAD: left atrial diameter.

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 [11,12]. Left ventricular systolic function is affected late in the course of obesity and more so in patients with considerable

degree of obesity [2,5]. Alterations in the left ventricular diastolic function were more frequent with increasing obesity. The MVPHT was found to be significantly prolonged in the obese personnel and correlated directly with the grade of 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 [13-15]. Similarly, the deceleration time was significantly prolonged in the obese subjects and correlated inversely with BMI.

IVRT was similar in obese subjects and in 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 [11,12]. 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 [11], while others have found no significant change in it [14]. 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.

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 [3,4].

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 [3,5,8]. 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 [16,17].

There are a few limitations of our study, the study sample included only service personnel and this precludes extrapolation of our results to the general population. 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 [18].

To conclude, all patients with isolated obesity have subclinical left ventricular diastolic dysfunction, which correlates with BMI and is associated with an increased in systolic function in the early stages of obesity.

Conflicts of Interest

None identified

Intellectual Contribution of Authors

Study Concept : Wg Cdr DS Chadha

Drafting & Manuscript Revision : Wg Cdr DS Chadha,
Wg Cdr A Swamy, Col SK Malani

Statistical Analysis : Wg Cdr DS Chadha

Study Supervision : Air Vice Mshl (Retd) RK Ganjoo, AVSM, VSM,
Lt Gen OP Mathew, AVSM, SM

References

1. Kenchaiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. *N Engl J Med* 2002; 347: 305-13.
2. Haque AK, Gadre S, Taylor J, Haque SA, Freeman D, Duarte A. Pulmonary and cardiovascular complications of obesity: an autopsy study of 76 obese subjects. *Arch Pathol Lab Med* 2008;132:1397-1404.
3. Pascual M, Pascual DA, Soria F, et al. Effects of isolated obesity on systolic and diastolic left ventricular function. *Heart* 2003; 89: 1152-6.
4. Tumuklu MM, Etikan I, Kisacik B, Kaykicioglu M. Effect of obesity on left ventricular structure and myocardial systolic function: assessment by tissue Doppler imaging and strain/strain rate imaging. *Echocardiography* 2007; 24:802-9.
5. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. *Am J Med Sci* 2001; 321: 225-36.
6. Abhayaratna WP, Marwick TH, Smith WT, Becker NG. Characteristics of left ventricular diastolic dysfunction in the community: an echocardiographic survey. *Heart* 2006; 92 : 1259-64.
7. Powell BD, Redfield MM, Bybee KA, Freeman WK, Rihal CS. Association of obesity with left ventricular remodeling and diastolic dysfunction in patients without coronary artery disease. *Am J Cardiol* 2006;98:116-20.
8. Ammar KA, Redfield MM, Mahoney DW, Johnson M, Jacobsen SJ, Rodeheffer RJ. Central obesity: association with left ventricular dysfunction and mortality in the community. *Am Heart J* 2008; 156: 975-81.
9. Khouri SJ, Maly GT, Suh DD, Walsh TE. A practical approach

- to the echocardiographic evaluation of diastolic function. *J Am Soc Echocardiogr* 2004; 17: 290-7.
10. Pirat B, Zoghbi WA. Echocardiographic assessment of left ventricular diastolic function. *Anadolu Kardiyol Derg* 2007; 7 : 310-5.
 11. Otto ME, Belohlavek M, Khandheria B, Gilman G, Svatikova A, Somers V. Comparison of right and left ventricular function in obese and nonobese men. *Am J Cardiol* 2004; 93: 1569-72.
 12. Iacobellis G. True uncomplicated obesity is not related to increased left ventricular mass and systolic dysfunction. *J Am Coll Cardiol* 2004; 44: 2257.
 13. Stante BD, Galandauer I, Aronow WS, McClung LA, Alas L, Salabay C, et al. Prevalence of Left Ventricular Diastolic Dysfunction in Obese Persons with and without Diabetes Mellitus *Am J Cardiol* 2005;95:1527-8.
 14. Di Bello V, Santini F, Di Cori A, Pucci A, Palagi C, Delle Donne MG, et al. Obesity cardiomyopathy: is it a reality? An ultrasonic tissue characterization study. *J Am Soc Echocardiogr* 2006; 8: 1063-71.
 15. Van Putte-Katier N, Rooman RP, Haas L, Verhulst SL, Desager KN, Ramet J, et al. Early cardiac abnormalities in obese children: importance of obesity per se versus associated cardiovascular risk factors. *Pediatr Res* 2008; 64 : 205-9.
 16. Kalantar-Zadeh K, Kopple JD, Kilpatrick RD, McAllister CJ, Shinaberger CS, Gjertson DW, et al. Association of morbid obesity and weight change over time with cardiovascular survival in hemodialysis population. *Am J Kidney Dis* 2005; 46 : 489-500.
 17. Iacobellis G, Sharma AM. Obesity and the heart: redefinition of the relationship. *Obes Rev* 2007; 8: 35-9.
 18. Tanalp AC, Bitigen A, Cevik C, Demir D, Ozveren O, Tigen K, et al. The role of tissue Doppler study in the assessment of left ventricular dysfunction in obesity. *Acta Cardiol* 2008; 63 : 541-6.

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