Assessment of Left Ventricular Diastolic function in Obesity

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ABSTRACT

Background :

Obesity has been linked to a spectrum of cardiovascular changes, ranging from hyper dynamic circulation to subclinical cardiac structural changes like LVH, impaired compliance & finally overt heart failure.

Aims &Objectives:

Early detection of subclinical cardiac dysfunction & it's correlation with grades of obesity.

Methods:

In this case control study 60 isolated obese & over weight subjects included.60 non obese age & gender matched healthy controls were recruited. Cases & controls were assessed for LV functions(diastolic & systolic) by 2D echo colour Doppler study.

Results:

In the present study mean age of obese subjects was **36.4 ± 6.6 years** & **35.7± 6.2yrs** in controls, with a male, female ratio of **1.3 : 1.** The base line characteristics like weight, BMI, WC, waist hip ratio & systolic BP were significantly different in cases than controls. L.V.diastolic dysfunction was more prevalent in obese individuals ie **23 (38.33%.)** while **05(8.33%)** cases showed both systolic & diastolic dysfunction. Among the indices of diastolic function E, A, E/A ratio & DT were significantly decreased while IVRT & MV1/2T were significantly increased in cases than controls..In subgroup analysis E velocity & E/A ratio remained unaltered in over weight subjects while A velocity & DT were not significantly changed in both sub groups than controls**.**

Left atrial diameter(**29.8±3.8mmVs 24.6±3.7mm p<0.0001**)and LV mass (**201.8±60.1gm% Vs152.2±51.1gm% p<0.0001)** was significantly more in cases than controls. The parameters of L.V. diastolic dysfunction like E, A & IVRT further worsened with increasing grades of obesity**.)**

These parameters had a significant negative correlation with BMI & WHR**.**

Conclusion:

Hence we conclude that obesity affects systolic and diastolic left ventricular functions, diastolic more than systolic. Cardiac dysfunction is directly proportional to grade of obesity.

Introduction:

Obesity has turned out to be the most prevalent form of malnutrition in recent years. Its prevalence is 20-40% of adults and 10-20% of children in developed countries[1] and 10-15% in India [2,3,4.] Impairment of cardiac function has been reported to correlate with BMI and duration of obesity.[5] Obesity has been linked to a spectrum of cardiovascular changes ranging from hyperdynamic circulation,[2] to subclinical cardiac structural changes like LVH, impaired L.V. compliance and finally overt heart failure.

South-East Asians and Indians differ phenotypically from the western population. “Indian phenotypic

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obesity" is more of an abdominal obesity with or without increase in BMI[3] There is paucity of literature describing effects of such abdominal obesity on cardiac functions. Further unresolved issues in the relationship between obesity and cardiovascular disease include the form of L.V. dysfunction (systolic or diastolic) most associated with obesity. A better understanding of the link between obesity and ventricular dysfunction may help elucidate mechanisms through which obesity contributes to risk of cardiovascular disease and mortality.

Early detection of subclinical cardiac dysfunction and its correlation with grades of obesity is the purpose of the study.

Material and Methods:

In this case control study, 60 isolated obese subjects and 60 non-overweight, non-obese age and gender matched controls were enrolled from the wards and out-patient department of Indira Gandhi Government Medical College, Nagpur. Study was approved by the institutional ethic committee.

Subjects meeting the eligibility criteria of age between 15 to 45 years and having BMI>25 kg/m2 were included in the study where as those with Diabetes mellitus, hypertension, structural heart disease, anemia [Hb<10 gm], respiratory illness like COAD and pulmonary hypertension, smokers, chronic alcoholics, persons on drug modifying cardiac function like B-blocker, vasodilators, diuretics etc., persons with endocrine disorders like hypothyroidism, cushing's syndrome and pregnant females were excluded from the study. Controls were age ($\pm$ 5 years) and gender matched healthy individuals with normal BMI (18.5 to 25 kg/m2). Exclusion criteria were the same for the controls.

Anthropometric measurements in the form of height, weight,waist circumference (W.C), Waist hip ratio(W.H.R),Hip circumference (H.C.) and BMI were calculated.[6,7]

Evaluation of L.V. function was done at rest using the ESAOTE Megas GP 2-D Echo Colour Doppler MAP model machine. Parasternal long axis view was used to measure left ventricular (L.V.) indices like end diastolic diameter(EDD),end systolic diameter (ESD),relative wall thickness(RWT),Fractional shortening(FS),left atrial diameter (LAD) and Ejection fraction(EF) were calculated by modified Simpson's rule and L.V. mass was calculated by Penn Convention.[8]

Systolic intervals like pre ejection period (PEP) and ejection period(EP) were determined by using combination of M mode at aortic valve and ECG.

Pulsed Doppler was obtained in apical four chamber view to measure L.V. mass early peak velocity [E], late peak velocity [A], E/A ratio, Acceleration time(AT), Deceleration time (DT) and Isovolumetric relaxation time(IVRT)[8]

The parameters considered for L.V. diastolic dysfunction were E, A, E/A, IVRT, DT, AT, Mitral valve pressure half time (MV 1/2T).[8]

The parameters considered for L.V. systolic dysfunction were EDD, ESD, EF, FS, RWT, PEP, EP, PEP/EP.[8]

In the present study a difference of more than 2 SD from mean values of the controls was used to estimate prevalence of cardiac dysfunction. Subclinical dysfunction was assumed when two or more indices of altered systolic or diastolic functions were present.

Statistical Analysis

Descriptive statistics were done on each of the variables to obtain the frequency distributions. Quantitative variables were described as mean ± S.D. Clinical data was analyzed by using **"XPSS version 15"**software.comparison between the obese group and normal weight group were analyzed by "t" test. Analysis of variance [ANOVA] was used to compare obese subgroups. Post–hoc testing was undertaken using the Bonnferroni multiple comparison test. Correlation between clinical variables and L.V. functions were determined by linear regression analysis. Probability value of p<0.05 was considered significant while P<0.01 was taken as highly significant.

RESULTS

In the present study mean age of obese subjects was **36.4 ± 6.6 years** & **35.7± 6.2yrs** in controls,with a male, female ratio of **1.3 : 1.**

The base line characteristics like weight, BMI, WC, waist hip ratio & systolic BP were significantly different in cases than controls**. (see Table 1)**

L.V.diastolic dysfunction was more prevalent in obese individuals ie **23 (38.33%.)**while **05(8.33%)** cases showed both systolic & diastolic dysfunction.Not a single case showed isolated systolic dysfunction. Among the indices of diastolic function E, A, E/A ratio & DT were significantly decreased while IVRT & MV1/2T were significantly increased in cases than controls.(**See Table 2**).In subgroup analysis E velocity & E/A ratio remained unaltered in over weight subjects while A velocity & DT were not significantly changed in both sub groups than controls**.(See Table 3).**

Indices of systolic dysfunction like EDD and PEP were altered in obese subjects. EF was increased in overweight subjects but not in obese subgroups.(**See table 4).**

Left atrial diameter(**29.8±3.8mmVs 24.6±3.7mm p<0.0001**)and LV mass (**201.8±60.1gm% Vs152.2±51.1gm% p<0.0001)** was significantly more in cases than controls. The parameters of L.V. diastolic dysfunction like E, A & IVRT further worsened with increasing grades of obesity**.(SeeTable 5)**These parameters had a significant negative correlation with BMI & WHR**. (See Table 6)**

Discussion :

In the present study L.V. diastolic dysfunction was more prevalent in obese subjects (38.33%) and increased with increasing grades of obesity. Various authors have previously emphasized association of LV diastolic dysfunction & impaired LV systolic function with moderate to severe obesity [9,10,11,12] Significant decrease in peak early mitral velocity (E) in obese subjects than controls is reported in one of the previous study[9] Abnormal relaxation of LV in early diastole is the explaination given by the author.Similar finding was observed in the present study in obese subjects but not in overweight subgroup. However, Stoddard et al[13] and Wong et al[14] have observed an increase in E in obesity. This is perhaps due to increase in L.A. pressure which is a major determinant of peak filling velocities. The above hypothesis also explains the increase in A velocity reported by other studies.[11,15,16] But the present study does not correlate with this finding.

Varying observations regarding E/A ratio have been made in the previous studies [9,11,15,16] In the present study there was significant decrease in the E/A ratio in obese subjects. This disparity among different studies in simple flow measures may reflect the sensitivity of transmitral flow indices to loading conditions as well as influence of increase L.V. mass in obesity. [9,15,16.]

In the present study, we observed an increase in IVRT nd MV1/2T in obese subjects. Similar observations were made by various authors [(68) (96)] but some differed [11.] The increase in IVRT is explained on the basis of cardiomyopathy of obesity which leads to delay in L.V. relaxation and hence late opening of mitral valve. No previous study has considered AT as a parameter of L.V. diastolic dysfunction in obesity. In the present study, we tried to correlate AT with L.V. diastolic function and found it to be significantly decreased in obese subjects than in controls. AT represents one of the visco-elastic forces of L.V. and it is load dependant. Hence, it is reasonable to expect AT to decrease in obese subjects who have an increased preload and decreased after load.

When systolic functions were evaluated, no subject had isolated L.V. systolic dysfunction. In the 5 (8.33%) subjects, who had systolic dysfunction, diastolic dysfunction also co-existed. There was an increase in EF in the overweight subjects but not in obese subgroups. This could be explained on the basis that in lesser degrees of obesity, there is compensatory increase in systolic function which has yet not reached the stage of cardiac deterioration. Also, EF is relatively insensitive to L.V. contractile function so its value may remain unchanged or seen increased when there is substantial compensatory modification of the contractile state [11,12]

However, in our study increase in EF was in accordance with normal RWT in obese subjects, which indicates that systolic function was largely preserved except an increase in EDD and PEP. Similar findings were observed by Pascual et al.[17] Some studies differ from the present one with respect to increase in RWT, indicating increase in L.V. stress in obese subjects.[12] In the present study, EDD was increased in obese subjects but remained unchanged in overweight subjects. Systolic intervals on echocardiography reflect inotropic state of myocardium and changes in preload and afterload. Usually cardiac failure prolongs PEP, shortens EP and hence increases PEP/EP ratio. Present study showed significant increase in PEP across all grades of obesity. However, EP and PEP/EP ratio remained unchanged. Stoddard et al [13] noted significant increase in PEP and PEP/EP while EP remained unchanged.

Most echocardiographic studies using measurements of ejection phases to evaluate systolic functions in obese subjects have shown normal results.[14,17,18] One of Studies reported reduced LV ejection fraction in severe grades of obesity.[19] In the present study the number of subjects contributed from the severe obese subgroup were only 11.6%, hence indices of systolic dysfunction remained largely unaffected.

Associarion of LA enlargement with reduced diastolic function is more consistent with correct understanding of filling pressure than the previous findings of isolated LA enlargement without diastolic changes.[11] Significant increase in LA size in obese has been reported by various authors.[14,17,20,21] Observation of the present study about increase in LA size was not different than others.LV diastolic function worsens with increasing grades of obesity is reported by Pascual etal [17.] We also observed the same.

There are a plethora of studies demonstrating increase in L.V. mass with increasing grades of obesity.[9,14,18,21.] The present study also supports the hypothesis. L.V. mass also showed significant positive correlation with age, BMI and WHR.

Hence we conclude that obesity affects systolic and diastolic left ventricular functions, diastolic more than systolic . Cardiac dysfunction is directly proportional to grade of obesity. Increase in EDD and PEP may be considered earliest changes in LV. Systolic dysfunction in obese subjects. IVRT, AT and LAD are the earliest and most commonly affected parameters of L.V. diastolic dysfunction.

By using a simple, non-invasive investigation like 2-D with doppler Echocardiography, it is possible to detect subclinical cardiac dysfunction in obesity. Early therapy can then be initiated to stop its further progress and perhaps even reverse it.

Table-1

Baseline characteristics of the cases and controls

Character (mean±SD)	Cases (n=60)	Controls (n=60)	P value
Age (years)	36.4 ± 6.6	35.7 ± 6.2	0.243
Weight (kg)	81.7 ± 13.7	64.34 ± 7.3	0.02*
Height (cm)	51.5 ± 10.6	156.88 ± 11.5	0.321
BMI (kg/m2)	32.56 ± 4.8	22.56 ± 0.94	0.0001**
Waist Circumference (cm)	102.4 ± 11	75.06 ± 3.4	0.0001**
Waist Hip Ratio	0.961 ± 0.06	0.821 ± 0.06	0.001**
Systolic BP (mmHg)	134.4 ± 7.3	125.4 ± 6.9	0.01*

*=significant value, **=highly significant value

Table-2

Comparison of Indices of LV diastolic function in cases and controls

Diastolic Indices (mean±**SD)**	Cases (n=60)	Controls (n=60)	P value
E (m/s)	1.05 ± 0.44	1.29 ± 0.3	0.002**
A (m/s)	0.86 ± 0.35	0.95 ± 0.3	0.163
E/A	1.24 ± 0.3	1.41 ± 0.25	0.002**
IVRT (msec)	118 ± 37.8	89.5 ± 23.5	0.0001**
MV1/2 T (msec)	54.5 ± 21.7	44.6 ± 11.7	0.002**
DT (msec)	154.9 ± 57.7	163.1 ± 50.8	0.411
AT (msec)	89.6 ± 24.6	106.7 ± 22.2	0.001**

**=highly significant value

Table-3

Comparison of LV diastolic function indices cases in subgroups

Diastolic Indices (mean±SD)	**Controls (n=60)**	**Overweight (n=20)**	**P value**	**All obese (n=40)**	**P value**
E (m/s)	1.29 ± 0.3	1.11 ± 0.4	0.091	1.02 ± 0.4	0.001**
A (m/s)	0.95 ± 0.3	0.85 ± 0.3	0.269	0.87 ± 0.36	0.236
E/A	1.41 ± 0.25	1.31 ± 0.2	0.16	1.2 ± 0.36	0.0008**
IVRT (msec)	89.5 ± 23.5	128.5 ± 41.2	0.0001**	113.7 ± 35.5	0.0001**
MV1/2 T (msec)	44.6 ± 11.7	58.7 ± 22.4	0.0005**	52.4 ± 21.3	0.02*
DT (msec)	163.1 ± 50.8	161.4 ± 64.1	0.693	151.2 ± 54.5	0.267
AT (msec)	106.7 ± 22.2	91.1 ± 28.3	0.013*	88.8 ± 23.3	0.0002**

*=significant value, **=highly significant value

Table4

Comparison of LV systolic function indices in case and controls

Systolic Indices	Cases (n=60)	Controls (n=60)	P value
EDD (cm)	4.59 ± 0.65	4.43 ± 0.48	0.008**
ESD (cm)	3.13 ± 0.6	3.02 ± 0.51	0.317
EF (%)	62 ± 8.5	60.6 ± 7.6	0.351
FS (%)	32.02 ± 8.02	30.8 ± 7.6	0.317
RWT (cm)	0.435 ± 0.1	0.414 ± 0.1	0.277
PEP (msec)	66.5 ± 22.6	52.01 ± 15.8	0.0001**
EP (msec)	244.8 ± 33	246 ± 26	0.351
PEP/EP	0.27 ± 0.82	0.213 ± 0.07	0.183

*=significant value, **=highly significant value

Table-5				
LV dysfunction in subgroups analysis as per grades of obesity				
LV Dysf.unction	Overweight (n=20)	Mild Obese (n=24)	Moderate Obese (n=09)	Severe Obese (n=07)
Systolic	00	02 (8.33%)	01 (11%)	02 (28.57%)
Diastolic	05	07	05	06

Table-6

Correlation between clinical variable and LV diastolic function indices

Diastolic Indices	BMI	P value	AGE	P value	Waist Hip ratio	P value
E (m/s)	-0.112	0.394	-0.15	0.237	0.028	0.087
A (m/s)	0.008	0.948	0.06	0.598	0.135	0.067
E/A	-0.08	0.511	-0.32	0.01*	-0.16	0.132
IVRT (msec)	-0.181	0.165	0.089	0.498	-0.03	0.05*
MV1/2 T (msec)	-0.09	0.493	0.142	0.278	-0.005	0.53
DT (msec)	-0.12	0.337	-0.065	0.701	-0.06	0.549
AT (msec)	-0.005	0.967	-0.04	0.726	-0.32*	0.01*
LV mass (gm%)	0.43	0.009**	0.32	0.0001**	0.47	0.0001**

*=significant value, **=highly significant value

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