Effect of obesity on Left ventricular diastolic function in young normotensive obese individuals.

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Abstract

Background: The prevalence of obesity is increasing at an alarming rate even in the developing country like India. Increased adiposity is the risk factor for the development of hypertension, diabetes and cardiovascular disease. The excess fat mass associated with obesity is known to cause haemodynamic changes like increase in preload and after load and also peripheral resistance. The study aims to evaluate the association between the obesity and Diastolic function using echocardiography in uncomplicated young obese individuals.

Materials and Methods: The study was conducted in age matched 24 young male obese individuals and 26 male controls. Anthropometric parameters like height (cms), weight (kgs) were measured. BMI was calculated. Controls with BMI of 18.50 – 24.99 kg/m², Obese individuals with BMI \geq 30 kg/m². Echocardiograms were performed using GE Vivid T 8 by standard techniques. The early diastolic (E) and atrial (A) velocities were measured and the E/A ratio was calculated. The deceleration time (in ms) was also assessed. Statistical analysis was done using 't' test

Results: The mean values of the early diastolic (E) velocity and E/A ratio were lesser in the group of obese individuals when compared with the controls and was statistically significant. Deceleration time and left ventricular end diastolic volume were greater in obese individuals.

Conclusion: The present study suggests a significant decrease in the diastolic function in obese individuals when other risk factors are excluded. So obesity is found to have detriment on diastolic function and may lead to cardiac failure. Echocardiographic study should be included in the evaluation of obesity for early detection diastolic dysfunction. Preventive measures should be taken to decrease the morbidity and mortality associated with obesity.

Keywords: Obesity, Body mass Index, Echocardiography

Introduction

Obesity is rapidly becoming the number one health problem worldwide. As the current younger generation ages the percentage of persons affected by obesity is expected to increases enormously. In developing country like India, the prevalence of obesity is increasing at an alarming rate. Easy availability of high energy foods, decreased requirement for physical activity in modern society and genetic predisposition are the causes for the epidemic of obesity globally with the sequel of diabetes, cardiovascular disease and some forms of cancer.One of independent risk factor for the incident of heart failure is obesity. One of the important adaptation that occurs in response to overweight and obesity are changes in the structural and morphological configuration of the heart along with haemodynamic changes like increase in preload and after load and also peripheral resistance. Studies have revealed left ventricular hypertrophy, some showed eccentric hypertrophy others concentric hypertrophy. Left ventricular mass, LV mass index were increased in uncomplicated obese individuals^[1,2]. In mid 20th century, studies found out an association between severe obesity and left ventricular dysfunction. Studies also showed a diastolic dysfunction rather a systolic dysfunction in obese individuals. Left ventricular (LV) diastolic dysfunction is nothing but a condition that reflects an impaired filling of the LV which may leads to development of heart failure and also serves as an

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Associate Professor, Department of Physiology, J. J. M. Medical College, Davangere - 577004 Karnataka, India. Email: renukusagur@gmail.com risk predictor in population settings^[3,4,5]. The probable pathophysiology for the occurrence of heart failure in increased body weight might therefore be the LV diastolic dysfunction that starts early in the obese individual. In this study, the association between the obesity and Diastolic dysfunction was evaluated using echocardiography, in uncomplicated young obese individuals with absent other known risk factor such as diabetes, Hypertension and Ischemic heart disease.

Materials and Methods

After obtaining Institutional ethical clearance this study was done.

Using prevalence of cardiac dysfunction among obese individuals to be 80% [p] and taking relative precision [d] as 20%, sample size was calculated using the formula

n=4pq/d*d, where

n=sample size

p= prevalence of cardiac dysfunction among obese individuals to be 80% [reference needed or we can take as assumption]

q=1-p

d= relative precision= 20% of p

n=24

All the subjects were informed about the procedure and informed consent was obtained. A brief history was taken followed by cardiovascular and respiratory system examination. A structured proforma is used to collect the relevant data. The present study group consists of 24 young male obese individuals and 26 male controls. All subjects were matched for the age. Anthropometric parameters like height (cms) weight (kgs) were measured. BMI was calculated using Quetelet's index i.e. Body weight (kilograms) divided

Results

elet's Index I.e. Body weight (kilograms) divided 16 (Chicago) software

Table 1: Clinical and demographic characteristics of the subjects

by the square of the height (meters). Non- obese individuals with BMI of 18.50-24.99 kg/m2 and Obese individuals with BMI \geq 30 kg/m2 were considered. Heart rate and Blood pressure measurements were done. Healthy males, in the age group of 25-40 yrs, Non smokers, Non alcoholic, while Adults above the age of 40 years and below 25, Systemic illness including hypertension, diabetic mellitus, renal or endocrinal diseases and Subjects on any medications, subjects of acute or chronic illness were excluded from the study

Echocardiographic studies

All the subjects underwent a transthoracic two-(2-D)-quided dimensional M-mode Doppler echocardiogram. Echocardiograms were performed using GE Vivid T 8 by standard techniques with subjects in the left lateral decubitus position. Echocardiograms were recorded on videotape. 2D-echo measurements included the LV enddiastolic volume. All measurements were performed according to the guidelines of American Society of Echocardiography. Pulsed-wave Doppler derived transmitral inflow velocities were obtained with the transducer in the apical four-chamber view and sampling volume at the mitral valve leaflet tips. The early diastolic (E) and atrial (A) velocities were measured and the E/A ratio was calculated. The deceleration time (in ms) was also assessed. Left ventricular end diastolic volume was also found out. All measurements were performed according to the guidelines of American Society of Echocardiography

Statistical Analysis:

Data are presented as means \pm standard deviation. Comparison between obese and controls were done using students t test. A p value of 0.05 or less was considered for statistical significance. SPSS version 16 (Chicago) software was used for all the analysis.

Characteristics	Controls (26) BMI 18.5 - 24.9 kg /m2	Obese (24) BMI ≥ 30 kg/m2	t value	P value
Age, (years)	35.2 ± 1.81	37.9 ± 2.77	0.187	0.98 NS
Weight (kg)	75.2 ± 2.25	95.50 ± 4.81	2.101	0.04 S
Height (cms)	156.4 ± 4.50	158.6 ±3.63	0.176	0.859 NS
BMI (kg/m2)	24.08 ± 1.18	37.97 ± 1.64	7.560	<0.001 HS
Heart Rate (beats/min)	70.6 ± 2.27	85.90±11.80	0.003	0.997 NS
Systolic blood Pressure(mmHg)	117.2±4.34	128±6.32	0.001	0.999 NS
Diastolic Blood Pressure(mmHq)	80.4±2.80	81±3.16	0.671	0.51 NS

Table 1 summarizes the clinical and demographic characteristics of the subjects. 26 controls and 24 obese individuals were taken for the study. No significant difference was found out regarding Age and Height between the controls and obese. But statistically significant increase in the weight and BMI were found out between them. Even though the heart rate, Systolic blood Pressure, Diastolic Blood Pressure were more in obese individuals when compared to the controls but the increase was not statistically significant.

Parameters	Controls BMI 18.5 - 24.9 kg /m2	Obese BMI ≥ 30 kg/m2	t value	P value
LVED ml	71.8 ± 5.14	90.80 ±13.03	9.96	<0.001 HS
E wave m/s	1.007 ± 0.06	0.69± 0.10	3.97	0.0008 HS
E/A ratio	1.38 ± 0.30	1.16 ± 0.21	4.8	<0.001 HS
Deceleration Time ms	197. 6 ± 8.81	209.80 ± 18.81	2.37	0.03 S

LVED – Left Ventricular End Diastolic

E Wave - left ventricular relaxation in early diastole

E/A Ratio - peak velocity blood flow from left ventricular relaxation in early diastole (the E wave) to peak velocity flow in late diastole caused by atrial contraction (the A wave).

Table 2 summarizes the Echocardiographic diastolic function in Controls and obese individuals. The mean values of the early diastolic (E) velocity and E/A ratio were lesser in the group of obese individuals when compared with the controls and was statistically significant. Deceleration time and left ventricular end diastolic volume were greater in obese individuals when compared with the controls and was statistically significant.

Discussion

The excess fat mass associated with obesity is known to increase metabolic demand and, thus, both cardiac output and total blood volume are elevated in obesity^[6]. These circulatory changes cause left ventricular geometric remodelling in the form of cavity dilatation, a structural change commonly seen in obesity, which is then thought to lead to a compensatory left ventricular hypertrophic response in response to increased wall stress^[1,7,8].

A community based study revealed that increased BMI was associated with worse LV diastolic function which was independent of LV mass and associated risk factors. This increased risk of LV diastolic dysfunction in both overweight and obese individuals were partially accounted for the increased risk of heart failure associated with both conditions by the researchers ^[4].

One of the studies revealed that there is a relationship between cardiac manifestations and young, normotensive obese women due to the association of obesity with LV mass ^[7]. The same study also demonstrated a higher chance of LV hypertrophy in obese individuals as compared to lean women^[7].

An array of studies involving humans and in animal model hyperinsulinemia, have been linked to ventricular hypertrophy ^[6,9]. While other set of researchers have proved that LV wall thickness, diameters, volumes, and LV mass indexed to height increases with increasing BMI ^[8]. They also found significant differences in mild and severely obese individual groups as compared with the controls when seen in the morphological measures, except for left ventricular end diastolic diameter. Both the indexed LV mass and wall thickness were correlated with insulin levels^[8].

Research including healthy individuals found that there was age related changes in diastolic dysfunction and these changes were gender specific. Regardless of this in our study we didn't find any age and gender related correlation to diastolic dysfunction as we excluded the age of >40 years^[10].

In addition to this, advances in the understanding of hormonal changes in obesity have highlighted several alternative mechanisms. Increased visceral and subcutaneous adiposity is known to cause higher levels of serum leptin, the hallmark of human obesity, and hyperinsulinaemia, both of which have been linked to ventricular hypertrophy in humans and in animal model^[2,9,11].

Limitations of the study

In our study, we were able to demonstrate that obesity is significantly related to the development of left ventricular diastolic dysfunction. We could have also included other factors like serum cholesterol, serum cholesterol/HDL ratio, FBS, PPBS, GTT levels and any other investigations which have an impact on the health and also provide a bigger picture on the burden of the disease.

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