

AN ECHOCARDIOGRAPHIC INVESTIGATION OF THE IMPACT OF OBESITY ON THE LEFT VENTRICLE'S SYSTOLIC AND DIASTOLIC FUNCTIONS IN HYPERTENSIVE ADULTS

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Abstract

Background: Increased cardiovascular morbidity and mortality are linked to obesity. It is unclear if isolated obesity has a direct impact on heart function. In order to aid in the earliest possible diagnosis of any dysfunction in asymptomatic individuals by non-invasive echocardiography, the current study was undertaken to investigate the impact of obesity on the systolic and diastolic functions of the LV in asymptomatic hypertensive adults. **Materials and Methods:** For six months, a cross-sectional study was carried out at the Department of Physiology Tertiary Care Institute in India. Following the measurement of their blood pressure and BMI, individuals were separated into obese and non-obese hypertensives. The results of an echocardiographic examination were recorded, together with characteristics suggesting LV shape and function. **Result:** When compared to non-obese hypertensives, the echocardiographic characteristics of LV diastolic function were significantly different in obese hypertensives (P 0.05). Function of the Left Ventricular Systole Between the obese and non-obese groups, there was no discernible change in the ejection percentage (p>0.05) or fractional shortening (p>0.05). The values for Tricuspid annular plane systolic excursion (p0.05) and Tei Index for RV (p0.05) did not vary significantly. **Conclusion:** In hypertensives, obesity has a considerable impact on the LV shape and diastolic functioning. Obesity and hypertension together dramatically increase the likelihood of having LVDD. Even in people with maintained systolic function, these changes can be seen on an echocardiogram.

INTRODUCTION

Due to its link to morbidity, mortality, and cardiovascular illnesses, obesity is a significant public health concern in today's society.^[1,2] According to WHO estimates, more than 1.9 billion adults aged eighteen and over are overweight, with obesity prevalence increasing gradually around the world. More than 600 of these adults were obese. Between 1980 and 2014, the prevalence of obesity more than doubled globally. Obesity has been linked to a greatly increased risk of diastolic dysfunction in heart failure.^[3-5] Because of a hyperdynamic circulation, chronic volume overload, and an increase in peripheral resistance, an excess of body fat causes an increase in both preload and afterload.^[6,7] Impairment of cardiac function has been reported to correlate with BMI and duration of obesity with most studies reporting abnormal diastolic function.^[8,9] Hypertension and obesity

frequently coexist.^[10] Thus, in addition to contributing to or enhancing the effect of hypertension with regard to cardiac hypertrophy, obesity is another risk factor.^[11] They have a number of interconnected mechanisms that have a big impact on the LV wall thickness, LV geometry, and LV functions. The fact that alterations in LV function have been documented in both clinically asymptomatic patients and people with normal ejection fraction and intact global LV systolic function only makes the situation worse. The aforementioned elements highlight the significance of early detection of LV structural and function alterations in the high-risk population as well as the requirement for a non-invasive method to accomplish this goal.^[12] Echocardiography is considered a validated and sensitive technique for measuring and classifying changes in the LV structure and functions.^[13] Studies employing cardiac catheterization, echocardiography, and necropsy examination have demonstrated

relationships between morbid obesity, structural heart changes, and systolic performance.^[14-16] In order to aid in the earliest possible diagnosis of any dysfunction in asymptomatic individuals by non-invasive echocardiography, the current study was undertaken to investigate the impact of obesity on the systolic and diastolic functions of the LV in asymptomatic hypertensive adults.

MATERIALS AND METHODS

For six months, a cross-sectional study was carried out at the Department of Physiology Tertiary Care Institute in India. The institutional ethical committee provided its ethical approval, and each subject provided signed informed permission. 200 was determined to be the necessary sample size based on the earlier research.

Inclusion Criteria

Male and female volunteers with BMIs greater than 30 and ages greater than 20 were included.

Exclusion Criteria

- Hypertensive subjects, on antihypertensive therapy
- Diabetic subjects
- Subjects with evidence of coronary artery disease;
- CAD
- Subjects with evidences of valvular heart disease, thyroid disorders, dyslipidaemia and subjects with poor transthoracic echo window.

Age, gender, clinical status, and blood pressure were gathered as part of regular measurements and questionnaires for demographic information. Each eligible participant had a thorough medical history review, a physical examination, and a biological study. After a 12-hour fast, a venous blood sample was taken and sent for biochemical analysis to determine the levels of serum triglycerides, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, and low-density lipoprotein (VLDL) cholesterol (TG). A standard physical examination included a general physical, a systemic physical, and an anthropometric evaluation that included height, weight, and body mass index (BMI). To detect any subclinical heart abnormalities, transthoracic echocardiography and Doppler imaging were performed on each participant. To reduce errors in determining the subclinical pathology, a transthoracic 2-dimensional echocardiogram (TTE) with pulsed Doppler imaging (TDI) and 2D echocardiography was done. A certified registered cardiologist performed echocardiography utilising the GE Livid S6 commercially accessible machine while adhering to a set methodology. A parasternal long-axis view was used to measure the LV's linear dimensions in accordance with the American Society of Echocardiography's standards (ASE). Measurements

were made in M-Mode in parasternal view of the left atrium, left ventricular end diastolic diameter (LVEDD), and left ventricular end systolic diameter (LVESD). The predetermined Devereux and Reichek formula was used to determine the left ventricular mass (LVM). Using the monoplane area-length approach, measurements of the left ventricular end-diastolic volume (LVEDV), left ventricular endsystolic volume (LVESV), left ventricular ejection fraction (EF), and left ventricular fractional shortening (FS) were made. Body surface were multiplied by LA volume to produce the LA volume index (LAVI). The sample volume was positioned at the mitral valve leaflets while measuring transmitral inflow velocities using pulsed-wave Doppler (PWD) in the apical 4-chamber view. Measurements included isovolumetric relaxation time (IVRT), deceleration time, and transmitral early diastolic rapid filling (E-wave) and atrial contraction late filling (A-wave) velocities (DT). The level of the lateral and septal mitral valve annulus, as well as the peak early diastolic (E') velocities, were chosen for the placement of the pulsed TDI sample volume. The ratio between the E and E' (E/E') was calculated as an index of LV filling pressures. For tissue Doppler imaging, the mitral annulus velocity was measured with a 2-mm sample volume placed at the septal side of the mitral annulus (E'). In apical 4-chamber view M-Mode of tricuspid valve annulus was taken (TAPSE) and in the same view, peak tissue velocity of tricuspid valve annulus was taken (S'TV). All the measurements are taken with correlation with ECG changes.

Statistical Analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2007) and then exported to data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA). For all tests, confidence level and level of significance were set at 95% and 5% respectively.

RESULTS

100 obese people (Group 1) and 100 controls with normal weights made up the study sample (Group 2). Age, sex, as well as systolic and diastolic blood pressure, were similar between groups. (Table 1) In line with expectations, both the obese subjects' weight and BMI were higher than those of the 100 non-obese referent normal healthy people. Measurements between the two groups showed a link between rising BMI and rising septal wall diameter, LV mass, LV mass index, and LA diameter. LVEDD, LVESD, ejection fraction, and fractional shortening did not show any appreciable alterations, nevertheless. ($p > 0.05$) BMI was independently correlated with greater A velocity, lower E/A ratio, shorter deceleration time, lower E', and higher E/E', both of which are indicators of LV filling pressure ($p < 0.05$). However, there were no

notable modifications in the isovolumic relaxation time. Higher BMI was linked to lower global longitudinal strain, greater Tei Index for LV, and higher LA volume index (all $p < 0.05$). Tei Index values for RV ($p > 0.05$), LVEDV ($p > 0.05$), and LVESV ($p > 0.05$) did not significantly alter. Compared to TAPSE, a higher BMI was associated with a decreased pulsed Doppler peak annulus velocity ($p < 0.05$). Right Atrial and Left Ventricular Morphology As BMI rose, the obese group's septal wall thickness, posterior wall thickness, LV mass, LV mass index, and LA diameter all increased in comparison to the normal weight controls. LVEDD and LVESD did not alter significantly. ($p > 0.05$) Function of the Left Ventricular Systole Between the obese and non-obese groups, there was no discernible change in the ejection percentage ($p > 0.05$) or fractional shortening ($p > 0.05$). (Table 2) Function of the Left Ventricular Diastole Our research showed a substantial increase in the A

velocity lowered E/A ratio ($p < 0.05$), a decrease in the deceleration time ($p < 0.05$), a reduction in the E', an increase in the E/E' ratio ($p < 0.05$), an increase in the LA filling volume ($p < 0.05$), and an increase in the Tei Index for LV. But E velocity ($p > 0.05$), IVRT ($p > 0.05$), LVEDV ($p > 0.05$), and LVESV ($p > 0.05$) did not show any significant variations.

Global Longitudinal Strain

Increasing BMI was consistent with a lower peak longitudinal strain.

RV Function

Obese subjects, with increasing BMI, showed a lower value of pulsed-Doppler peak velocity of annulus ($p < 0.05$) when compared to the normal weight subjects. However, there were no significant changes in the value for Tricuspid annular plane systolic excursion ($p < 0.05$) and Tei Index for RV ($p < 0.05$).

Table 1: Comparison of blood pressure between obese hypertensives and non-obese hypertensives

Blood pressure	Obese hypertensives (Mean±SD)	Non-obese hypertensives (Mean±SD)	P value
SBP (mmHg)	134.80±12.9	138.05±16.4	0.10
DBP (mmHg)	83.90±7.80	84.90±11.02	

DBP: Diastolic blood pressure, SBP: Systolic blood pressure
Statistically significance at $p < 0.05$

Table 2: Comparison of echo parameters between obese hypertensives and non-obese hypertensives

Echo Parameters	Obese hypertensives (Mean±SD)	Non-obese hypertensives (Mean±SD)	P value
ESV (ml)	45.10±31.9	41.92±18.10	0.12
EF (%)	58.9±8.4	59.2±6.45	0.23
IVSs (cm)	1.47±0.10	1.51±0.22	0.54
LVIDd (cm)	4.49±0.50	4.40±0.32	0.47
LVIDs (cm)	2.9±0.68	3.05±0.40	0.06
DT (ms)	240.44±54.5	224.48±47.2	0.05*
EDV (m)	103.86±41.1	99.1±26.50	0.36
IVRT (ms)	105.94±16.2	93.8±12.9	0.003*
E/A ratio	0.88±0.14	1.05±0.22	0.001*

* indicates statistically significance at $p < 0.05$

DISCUSSION

The leading independent risk factors for cardiovascular morbidity are obesity and hypertension, both of which are known to alter the anatomy and function of the heart. However, these modifications can continue to be subclinical for some time. Early diagnosis of the aforementioned alterations in patients who appear to be asymptomatic may benefit in the prompt implementation of corrective actions. In addition to being a simple and non-invasive procedure, echocardiography is a trustworthy instrument for evaluating heart structure and function. Consequently, it can be used for this type of assessment in the population at risk. The majority of echocardiographic investigations employing ejection phase measurements to assess systolic function in

obese people have produced normal results.^[18-22] Studies in which these indices have been found to be reduced, albeit modestly, were done on patients with a considerable degree of obesity, suggesting that left ventricular systolic function is affected late in the course of obesity.^[22] There were no discernible differences in the IVSs, IVSd, LVIDs, or LVIDd between the groups. Both EF and ESV were comparable between the two groups. In contrast to the obese normotensive group, obese hypertensives had considerably longer DT and IVRT as well as lower E/A and E/é values. This shows that even in the absence of obvious systolic or global impairment, obese hypertensives had considerably lower diastolic function measures. LVDD was present in roughly 58% of non-obese hypertensives and 78% of obese hypertensives, which is significantly greater than in the other group. It supports the theory that fat increases the likelihood

of diastolic dysfunction in hypertensive people. No significant difference in the prevalence of systolic dysfunction was noted between the groups. Garg et al., in their study, found diastolic dysfunction with normal ejection fraction in 53% of overweight subjects and in 35% of obese subjects.+ Zarich et al. observed a significant decrease in the maximum velocity of the passive mitral filling (E) among obese patients, whereas the values for active mitral filling (A) were not significantly affected, resulting in a decrease in the E/A ratio. Conversely, Chakko et al. did not find significant differences in the values of E, but values of A were increased, resulting in a decreased E/A ratio. Stoddard et al. found a significant increase in both E and A values, which were positively correlated with the percentage of body weight in excess of the ideal so that the E/A ratio was not altered.^[24] In regards to CVD, Wang et al. (2010) revealed statistically significant interactions between systolic blood pressure, diastolic blood pressure, and BMI. According to Adebisi et al., BP and BMI significantly synergistically affected cardiac geometry and the left ventricular mass (LVM) index.^[25] According to Kossaiy et al.^[22] analysis of left ventricular diastolic function in 99 patients divided into BMI-based groups, values for LV mass, LV mass index, and septal wall thickness (SWT) were significantly higher in the overweight/obese groups than in the normal group. When compared to the group with normal BMI, the E' in the overweight/obese groups had a considerably lower TDI value. Wong et al.^[26] In this study comparing 109 overweight or obese subjects with 33 referents using transthoracic echocardiography, myocardial Doppler derived systolic and early diastolic velocity, found LV wall thickness, diameters, volumes and LV mass indexed to height increased with increasing BMI. Garg et al. in their study observed that when hypertension coexisted with obesity, there was a significant increase in left ventricular posterior wall thickness during diastole, relative wall thickness, and LVM/height. It was also noted that a higher number of obese hypertensive subjects had concentric hypertrophy.^[23] The current investigation confirms that obese hypertensives have worse left ventricular diastolic dysfunction than those without obesity. In people who are clinically asymptomatic, the changes in the left ventricular function metrics are still there. The study underlines the need for cardiac examination in order to identify structural and functional alterations early on, before symptoms manifest. In the population at risk of cardiovascular morbidity, echocardiography, a noninvasive and accurate method for assessing LV shape and function, may be used effectively. BMI was the only metric used to determine obesity; measurements of the distribution of body fat were not taken. A better relation might have been found between abdominal obesity and echocardiographic alterations. Investigation of cytokines, leptin, sympathetic nervous system activity, and renin–

angiotensin– aldosterone system activity could have added information about the underlying mechanisms.

CONCLUSION

In hypertensives, obesity has a considerable impact on the LV shape and diastolic functioning. Obesity and hypertension together dramatically increase the likelihood of having LVDD. Even in people with maintained systolic function, these changes can be seen on an echocardiogram.

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