A Study to Assess the Effects of Obesity on Ventricular Function by 2D Echocardiogharphy

Shilpa Patil[1], Arjun Mandade[2], Abhijeet Shelke[3], Ramesh Kawade[4], Kasturi Sanyal[5]

ABSTRACT

Introduction: Obesity constitutes a major health issue in the modern world. The purpose of this prospective study was to assess the effect of obesity on ventricular function by conventional 2D echocardiography and to predict the incidence of subclinical risk factors for cardiac complications in obese individuals.

Material and methods: For the study 30 subjects (cases) with a BMI>30 and no clinical evidence of cardiac disease were studied with BMI in the range 20-24.9 were included as controls, study was conducted over a amount of 2 months, from August 2016 to September 2016.

Result: Left Ventricular Systolic Function: Increasing BMI showed increase in septal wall thickness, posterior wall thickness, LV mass, LV mass index and LA diameter among the obese group compared to the normal weight controls.

Left Ventricular Diastolic Function: Significant increase in A velocity, reduced E/A ratio, decrease in deceleration time, reduced E', increased E/E' ratio, increase in LA filling volume and increase in Tei Index for LV. RV Function**:** Showed a lower value of pulsed-Doppler peak velocity of annulus but no significant changes in the value for Tricuspid annular plane systolic excursion and Tei Index for RV

Conclusion: The current study concluded that conventional 2D echocardiographic parameters of individuals with BMI>30 reveal increased wall thickness and mass of Left Ventricle (LV), significantly higher risk of LV diastolic dysfunction, evidence of Left Atrial (LA) enlargement associated with diastolic dysfunction, evidence of subclinical LV systolic dysfunction and Right Ventricular (RV) dysfunction.

Keywords: 2D Echocardiography, Obesity, Ventricular Function

INTRODUCTION

Obesity constitutes a major health issue in the modern world because of its association with morbidity, mortality and cardiovascular diseases.[1,2] The worldwide prevalence of obesity has been increasing steadily, with WHO estimates of more than 1.9 billion people were overweight from adults aged eighteen years and older. Of these over 600 adults were obese. The worldwide prevalence of obesity between 1980 and 2014 is more than doubled. Obesity has been associated with heart failure[3,4], with a significantly higher risk of diastolic dysfunction.[5] The excess in body fat determines a rise in each preload and afterload thanks to a hyperdynamic circulation, chronic volume overload and increase in peripheral resistance.[6,7] Impairment of cardiac function has been reported to correlate with BMI and duration of obesity[8,9], with most studies reporting abnormal diastolic function.

Hemodynamically, in obesity an increase in total blood volume is seen along with increased cardiac output, which in turn is fuelled by increased metabolic demand due to increased body weight. Also, in obesity, a shift to left is seen in the 'Frank-Sterling curve' due to incremental increase in the left ventricular filling pressure and volume. This over time leads to dilatation of the chamber which causes increased wall stress, subsequently leading to ventricular hypertrophy of eccentric type. As a result, left ventricular (LV) dilation and increased LV mass are frequent findings in individuals with obesity[10-12], with both eccentric and concentric LV geometric patterns described in these conditions. LV diastolic dysfunction might represent one of the pathophysiological links between an increase in body weight and an increased incidence of heart failure.

Our aim was to match the impact of body mass index (BMI) on ventricular functions among healthy subjects with conventional 2D echocardiography (echo). Cardiac abnormalities of the obese adults include the echocardiographically revealed early and preclinical LV or septal hypertrophy, and left or right ventricular dysfunction.[13,14] It is noted that developed subclinical cardiac effects in different studies reverse the process during weight loss.[15-20] In addition, 2D Echo sensitive echocardiographic modality, which allows the assess regional and myocardial function with strain.[18-20] These strain parameters are affected to a much lesser extent by cardiac rotation and passive cardiac motion.[18]

The role of obesity as an independent predictor for LV hypertrophy, left atrial (LA) enlargement, and subclinical impairment of LV systolic and diastolic function as well as microvascular cardiac changes is not fully established. This study aims to determine a link between echocardiographically demonstrated subclinical cardiac impairment and increasing BMI of normotensive, non-diabetic healthy individuals

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without any other cardiac comorbidities.

Study of ventricular function in non-hypertensive, non-diabetic and individuals without other associated co morbidities is important to detect any subclinical, unapparent pathology which may progress to further cardiac complications. Early detection and a well-structured treatment plan may help prevent such complications.

Aims and Objectives

The aims of the study were –

1. To assess the effect of obesity on left ventricular and right ventricular function by conventional 2D echocardiography.
2. To predict the incidence of subclinical risk factors for cardiac complications in obese non-hypertensive, non-diabetic healthy individuals.

MATERIALS AND METHODS

For the study 30 normotensive, non-diabetic healthy subjects (cases) with a BMI>30 and no clinical evidence of cardiac disease were studied. A total 30 apparently healthy age and sex matched subjects with BMI in the range 20-24.9 were included as controls. This case-control study was designed to see the prevalence of asymptomatic cardiac abnormalities with relation to obesity indices. This was a case-control prospective, data based study conducted over a period of two months, from August 2016 to September 2016. Written and informed consent for participation was obtained and this study was approved by the institute ethical committee.

Inclusion criteria for case population

Both male and female volunteers with BMI>30 and age above 20 years were included.

Exclusion Criteria For Case Population

- Hypertensive subjects, on antihypertensive therapy;
- Diabetic subjects;
- Subjects with evidence of coronary artery disease;
- CAD [excluded by history of angina, chest pain, Electrocardiogram (ECG) changes and abnormal Treadmill test (TMT) results];
- Subjects with evidences of valvular heart disease, thyroid disorders, dyslipidaemia and subjects with poor transthoracic echo window.

Clinical Assessment

Demographic details like age, gender, clinical status and blood pressures were taken by standard measurements and questionnaires. A detailed medical history was collected from each eligible subject, and they underwent physical examination and biological investigation. Venous blood sample was collected after a 12-hour fast and sent for biochemical analysis for estimation of Fasting glucose levels, TSH levels, Total serum cholesterol (TC), High-density lipoprotein (HDL) cholesterol, Low density lipoprotein (LDL) cholesterol, Very low density lipoprotein (VLDL), and serum triglyceride levels (TG).

Physical examination included routine general examination, systemic examination and anthropometric evaluation including height (meter), weight (kilogram) and calculation of BMI (Quetelet Index).

Echocardiography (Echo)

All subjects were investigated by transthoracic echocardiography and Doppler imaging, to assess any subclinical cardiac changes. A transthoracic 2-dimensional echocardiogram (TTE) with pulsed Doppler Imaging (TDI) and 2D echocardiography was performed to minimize the errors in assessing the subclinical pathology. Echocardiography was performed using a commercially available system, GE livid S6, by a trained registered cardiologist following a standard protocol.

LV linear dimensions were measured from a parasternal long-axis view as per the recommendations by American Society of Echocardiography (ASE). The left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD) and the left atrium (LA) diameter was measured in parasternal view in M-Mode. The left ventricular mass (LVM) was calculated using the predefined Devereux and Reichek formula. LVM was divided by the body surface area to obtain the left ventricular mass index (LVMI g/m2).

Measurement of LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), the LV ejection fraction (EF) and the fractional shortening (FS) were computed from apical two chamber (A2C) views using the monoplane area-length method. LA volume was multiplied by the body surface are to obtain the LA volume index (LAVI).

Pulsed-wave Doppler (PWD)-derived transmitral inflow velocities were measured in the apical 4-chamber view, with the sample volume placed at the mitral valve leaflets. Measurements included the transmitral early diastolic rapid filling (E-wave) and the atrial contraction late filling (A-wave) velocities to calculate E/A ratio, isovolumetric relaxation time (IVRT) and deceleration time (DT). Pulsed TDI sample volume was placed at the level of the lateral and septal mitral valve annulus and the peak early diastolic (E′) velocities were measured and average was taken. 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

Data analysis was done for mean, percentage, standard deviation, unpaired 't' square test, multiple correlation and multivariate analysis, by using SPSS-16 (Statistical Package for the Social Sciences) for Windows.

Multivariate stepwise linear regression analysis was performed to assess the adjusted correlations between echocardiography variables and BMI.

The 't' test was applied to study qualitative data, with p value <0.05 considered statistically significant.

Data is presented in the form of mean ± standard deviation.

RESULTS

The study sample included 30 obese subjects (Group 1) and 30 normal weight controls (Group 2). Characteristics of both groups studied are presented in Figure-1. Groups were similar with respect to age, sex, systolic and diastolic blood pressure. As expected, weight and BMI were elevated in the obese subjects (p<0.0001)

The study sample included 30 obese (BMI>29.9) and 30 non-obese (BMI<25) referent normal healthy individuals. Measurements comparing both groups revealed correlation between increasing BMI and increased septal wall diameter (p<0.0001), increased posterior wall diameter (p=0.0005), increased LV mass (p=0.0003), increase in LV mass index (p=0.0112), increased LA diameter (p=0.0274). However, there were no significant changes found in LVEDD (p=0.5633), LVESD (p=0.2042), ejection fraction (p=0.6769) and fractional shortening (p=0.1484).

BMI was independently associated with higher A velocity (p<0.0001), lower E/A ratio (p=0.0010), lower deceleration time (p=0.0069), lower E' (p=0.0005) and increased E/E' (p=0.0015), indicator of LV filling pressure. However, there were no significant changes in the isovolumic relaxation time (p=0.3569).

Higher BMI was associated with a higher LA volume index (p=0.0486) and a higher Tei Index for LV (p<0.0001) and a lower global longitudinal strain (p=0.0209). No significant changes were found in Tei Index for RV (p=0.4748), LVEDV (p=0.9067) and LVESV (p=0.5652).

A higher BMI was related to a lower pulsed Doppler peak velocity of annulus (p=0.0094) with no significant changes in TAPSE (p=0.1542)

Echocardiographic Findings

Left Ventricular and Left Atrial Morphology

Increasing BMI showed increase in septal wall thickness (p<0.0001), posterior wall thickness (p=0.0005), LV mass (p=0.0003), LV mass index (p=0.0112) and LA diameter (p=0.0274) among the obese group compared to the normal weight controls. There were no significant changes in LVEDD ((p=0.5633) and LVESD (p=0.2042) (Figure-2).

Left Ventricular Systolic Function

There was no significant difference in ejection fraction (p=0.6769) and fractional shortening (p=0.1484) across the obese and non-obese groups (Figure-3).

Left Ventricular Diastolic Function

Our study demonstrated a significant rise in A velocity (p<0.0001), reduced E/A ratio (p=0.0010), decrease in deceleration time (p=0.0069), reduced E' (p=0.0005), increased E/E' ratio (p=0.0015), increase in LA filling volume (p=0.0486) and increase in Tei Index for LV (p<0.0001). However, no significant changes were observed in E velocity (p=0.4506), IVRT (p=0.3569), LVEDV (p=0.9067) and LVESV (p=0.5652) (Figure-4).

Global Longitudinal Strain

Increasing BMI was consistent with a lower peak longitudinal

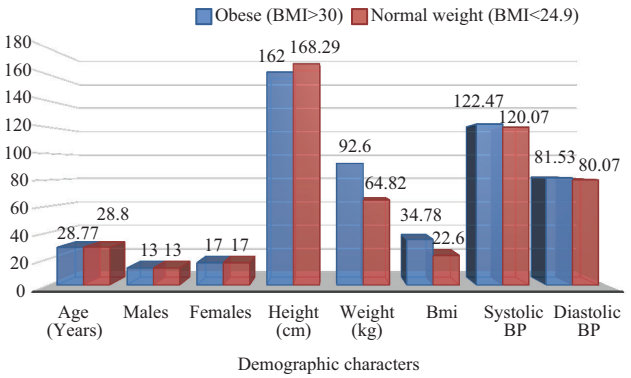

Figure-1: Demographic Characters

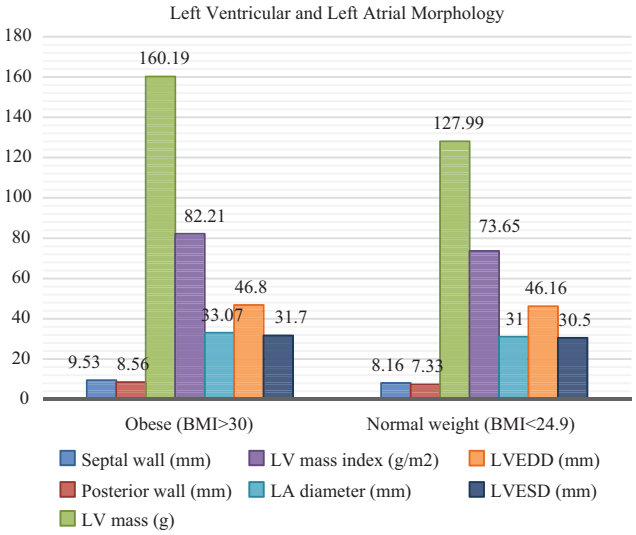

Figure-2: Left Ventricular and Left Atrial Morphology

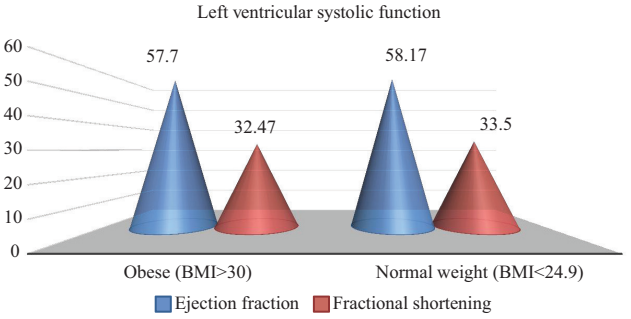

Figure-3: Left Ventricular Systolic Function.

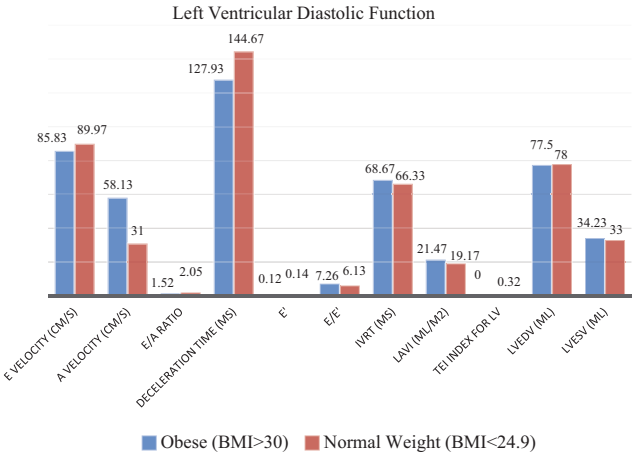

Figure-4: Left Ventricular Diastolic Function

strain (p=0.0209) (Figure-5).

RV Function

Obese subjects, with increasing BMI, showed a lower value of pulsed-Doppler peak velocity of annulus (p=0.0094) when compared to the normal weight subjects. However, there were no significant changes in the value for Tricuspid annular plane systolic excursion (p=0.1542) andTei Index for RV (p=0.4748) (Figure-6).

DISCUSSION

The results of this study demonstrate subclinical changes in the LV structure and function in obese healthy subjects with BMI>30 who do not have any other clinically appreciable cause of heart disease.

LV Morphology

Results from conventional 2D Echocardiography analysis reveal an increase in septal wall thickness, posterior wall thickness, LV mass and LV mass index in study group of obese volunteers as compared to the control group, all of which demonstrate a significant increase in LV size and LV hypertrophy.

To meet raised metabolic desires, circulating blood volume, plasma volume, and cardiac output all increase. The increase in blood volume successively will increases venous return to the right and the left ventricles, eventually producing dilation of these cardiac cavities, increasing wall tension. This ends up in LVH, that is in the course of decrease in diastolic chamber compliance, eventually resulting in an rise in left ventricular filling pressure and left ventricular enlargement. As long as LVH adapts to left ventricular chamber enlargement, systolic function is preserved. When LVH fails to keep pace with progressive left ventricular dilation, wall tension increases even more and systolic dysfunction may ensue. The risk of sudden cardiac death is also increased in obesity.[27]

Left Ventricular Systolic Function

The result of the current study shows no significant changes in the indices for LV ejection fraction and fractional shortening in the obese group as compared to the control group, albeit a slight decrease in both indices. The ejection fraction normality was in accordance with the normal relative wall thickness in our obese groups, which indicates preservation of systolic function.[28]

Most echocardiographic studies using measurements of the ejection phases to seethe systolic function in obese subjects have shown normal results. In some Studies these indices have been found to be decreased, albeit modestly, were done on patients with a moderate degree of obesity, suggesting in the course of obesity the left ventricular systolic function is affected late in course.[28]

Our study demonstrated a significantly lower value of peak longitudinal strain in the obese group as compared to the normal weight subjects, which points to subclinical LV dysfunction in correlation with increasing BMI.

Conventional methods such as LVEF and fractional shortening, which are relatively less sensitive and thus not able to pick up early preclinical changes. The more sensitive,

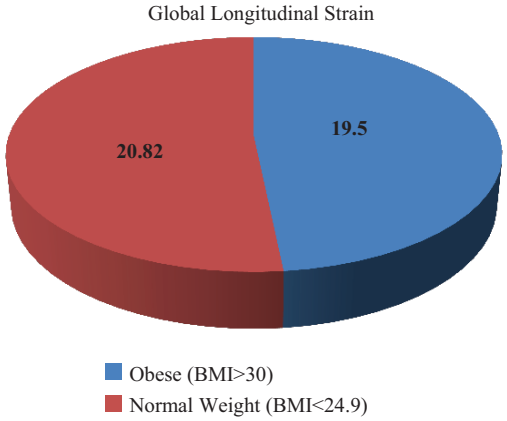

Figure-5: Global Longitudinal Strain

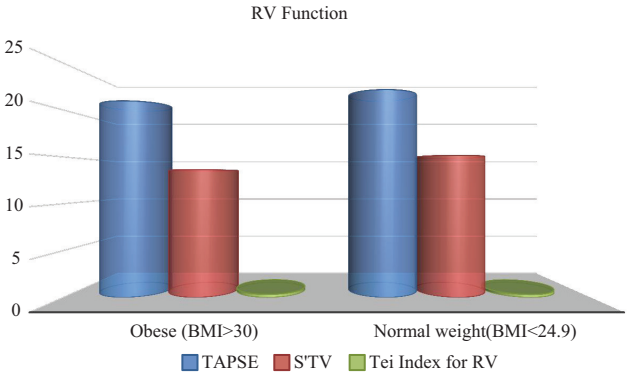

Figure-6: RV Function

newer echo techniques and strain imaging have been used in a recent study to demonstrate the presence of subclinical LV changes in young obese women.

Left Ventricular Diastolic Function

The result from the current study revealed lower values of E velocity, significantly higher A velocity, lower indices of E/A ratio, lower values of deceleration time, lower E', higher E/E' ratio, higher values of LA volume index and higher values of Tei Index for LV in the obese group, which is consistent with diastolic dysfunction seen with increasing BMI.

The correlation between BMI and diastolic function parameters is continuous and not dependent on cardiovascular risk factors that cluster with obesity, such as hypertension, diabetes and LV hypertrophy. The use of E/E′ ratio, a widely used indicator of LV filling pressure and an independent predictors of cardiac events like heart failure and myocardial infarction, revealed higher LV filling pressures in obese patients than in normal weight subjects. Cardiovascular risk factors were significantly more prevalent in obese individuals than in normal weight subjects.BMI was the principle predictor of LV mass and was not dependent on factors like the presence of hypertension and diabetes. In relationship between BMI and diastolic function parameters, Although the increased LV mass might be a contributor to the impairment of diastolic function observed in the overweight and obese groups, was only slightly weakened by the adjustment for LV mass and other covariates, suggesting that many mechanisms may link the increase in BMI with the impairment in LV diastolic properties.[5]

Left Atrial Morphology

The current study has demonstrated a significant increase in left atrial diameter in obese subjects as compared to normal weight individuals. The association of LA enlargement with decreased diastolic function is more consistent with current understanding of the left atrium as a biomarker of filling pressure than are previous findings of isolated LA enlargement without diastolic changes using less sensitive conventional methods.[24]

Right Ventricular Function

Results from the current study reveal a significant decrease in values of pulsed-Doppler peak velocity of annulus S'TV with a slight decrease in tricuspid annular plane systolic excursion (TAPSE) and a slight increase in Tei Index for RV in the obese group in contrast to the indices observed in the control group, which points to subclinical RV dysfunction in individuals with excess weight.

Right Ventricular dysfunction in relation with obesity as an independent parameter has not been studied extensively in previous studies.

Russo et al[5] In their case control study of 250 subjects grouped according to increasing BMI found that BMI was independently associated with higher E,A and E/E'. Overweight and obese had lower E' (both p<0.01) and higher E/E' (p<0.01) than normal weight subjects. Compared to normal weight subjects E/A was lower in obese (p<0.01). Similarly in our study obese subjects had a higher A velocity (p<0.0001), lower E/A ratio (p=0.0010), lower E' (p=0.0005) and increased E/E' (p=0.0015), indicator of LV filling pressure.

Kossaify et al[25] Evaluation of left ventricular diastolic function in 99 patients sub grouped as per their BMI revealed that values of LV mass, LV mass index, and septal wall thickness (SWT) were considerably superior in overweight/obese groups compared to values within the normal group. TDI showed a significantly lower E′ in overweight/obese groups compared to the E′ in the normal BMI group (P = 0.043). Impaired relaxation was encountered in thirty (30.64%) subjects. Similarly, in the current study, increased septal wall diameter (p<0.0001), increased LV mass (p=0.0003), increase in LV mass index (p=0.0112) were encountered. Increasing BMI was independently associated with a lower E' (p=0.0005) between obese and non-obese subjects.

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Wong et al[24] 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. These morphological measures, except for LVEDD, were significantly different in mildly and severely obese groups as compared with the referents. Ejection fraction did not disagree considerably across the BMI subgroups. However, there have been vital variation between obese and referent groups with relevance to septal, inferior, and average regional strain and systolic myocardial tissue velocity sm. Increasing degrees of obesity were related to a significant increment of IVRT and nonlinear changes in E and the E/A ratio, probably reflecting the impact of loading conditions. However, the findings on tissue Doppler measures were consistent with an association between diastolic dysfunction and obesity, evidenced by reduced mitral annular velocity (e′), myocardial early diastolic velocity (em), and elevated filling pressures, approximated by E/e′. Diastolic myocardial velocity (em) was significantly decreased in all the obese subgroups compared with the referents as well as between the subgroups. This corresponds to the results found in the current study with increasing BMI and increased septal wall diameter (p<0.0001), increased posterior wall diameter (p=0.0005), increased LV mass (p=0.0003), increase in LV mass index (p=0.0112), increased LA diameter (p=0.0274). However, there were no significant changes found in LVEDD (p=0.5633), LVESD (p=0.2042), ejection fraction (p=0.6769) and fractional shortening (p=0.1484).

BMI was independently associated with higher A velocity (p<0.0001), lower E/A ratio (p=0.0010), lower deceleration time (p=0.0069), lower E' (p=0.0005) and increased E/E' (p=0.0015), indicator of LV filling pressure. However, there have been no vital changes found in the isovolumic relaxation time (p=0.3569) and a lower global longitudinal strain (p=0.0209).

Russo et al[5] In their case control study of 250 subjects grouped according to increasing BMI found that BMI was independently associated with higher E,A and E/E'. Overweight and obese had lower E' (both p<0.01) and higher E/E' (p<0.01) than normal weight subjects. Compared to normal weight subjects E/A was lower in obese (p<0.01). Similarly in our study obese subjects had a higher A velocity (p<0.0001), lower E/A ratio (p=0.0010), lower E' (p=0.0005) and increased E/E' (p=0.0015), indicator of LV filling pressure.

CONCLUSION

The current study concluded that conventional 2D echocardiographic parameters of individuals with BMI>30 reveal increased Left Ventricular (LV) wall thickness and LV mass, significantly higher risk of LV diastolic dysfunction, evidence of Left Atrial (LA) enlargement associated with diastolic dysfunction, evidence of subclinical LV systolic dysfunction and Right Ventricular (RV) dysfunction.

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