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EARLY LEFT VENTRICULAR REMODELING AND ALTERED DIASTOLIC FILLING IN APPARENTLY HEALTHY OBESE ADULTS: A CROSS-SECTIONAL ECHOCARDIOGRAPHIC STUDY FROM WESTERN INDIA

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ABSTRACT

Background: Obesity is associated with adverse cardiac remodeling and may cause subclinical left ventricular (LV) structural and functional abnormalities before the onset of overt cardiovascular disease. This study evaluated the impact of obesity on LV structure and function in apparently healthy adults from Western India using conventional and tissue Doppler echocardiography.

Methods: In this cross-sectional study, 100 apparently healthy adults (50 obese and 50 non-obese) were recruited using consecutive sampling. Participants with systemic illnesses, including hypertension, diabetes mellitus, and coronary artery disease, were excluded. All subjects underwent standardized two-dimensional, M-mode, pulsed-wave Doppler, and tissue Doppler echocardiography. LV systolic function was assessed using left ventricular ejection fraction (LVEF) and mitral annular systolic velocity (s′), while diastolic function was evaluated using transmitral E and A velocities, E/A ratio, isovolumic relaxation time, and mitral annular e′ and a′ velocities. LV mass index (LVMI) was calculated using Devereux formula. Groups compared using independent-samples t-test, and multiple linear regression assessed independent associations between BMI and echocardiographic parameters after adjustment for age and sex.

Results: Obese participants had significantly higher LVMI, higher transmitral A-wave velocity, and lower E/A ratio than non-obese controls (all $p<0.001$), whereas LVEF and s′ remained comparable. BMI independently predicted LVMI ($\beta=3.91$; 95% CI: 3.02–4.80; $p<0.001$), A-wave velocity ($\beta=1.17$; 95% CI: 0.63–1.71; $p<0.001$), and E/A ratio ($\beta=-0.018$; 95% CI: −0.029 to −0.007; $p=0.001$).

Conclusions: Apparently healthy obese adults exhibit early LV structural remodeling and impaired diastolic relaxation despite preserved systolic function. Conventional echocardiography may facilitate early detection of obesity-related myocardial remodeling.

KEYWORDS: Obesity, left ventricular systolic function, left ventricular diastolic function, LV mass index, Echocardiography.

INTRODUCTION

Obesity has emerged as one of the most important global public health challenges, with its prevalence increasing rapidly across both developed and developing countries. The World Health Organization estimates that more than one billion adults worldwide are living with obesity,[1] a condition strongly associated with increased cardiovascular morbidity and mortality. [2] Beyond its well-recognized association with hypertension, diabetes mellitus, coronary artery disease, and heart failure, obesity exerts direct adverse effects on myocardial structure and function through hemodynamic overload, neurohormonal activation, systemic inflammation, insulin resistance, and adipokine dysregulation.[3] These mechanisms promote left ventricular (LV) remodeling even before overt cardiovascular disease becomes clinically apparent. [4]

Obesity-related cardiac remodeling is characterized by alterations in LV geometry, myocardial hypertrophy, increased LV mass, impaired myocardial relaxation, and eventually deterioration of ventricular function. Importantly, these structural and functional abnormalities may develop in otherwise asymptomatic individuals with preserved left ventricular ejection fraction (LVEF), emphasizing the need for early detection before irreversible myocardial damage occurs.[5] Contemporary evidence suggests that obesity-associated myocardial dysfunction represents a continuum beginning with subtle abnormalities in myocardial relaxation and ventricular remodeling rather than advanced systolic failure.[6]

Echocardiography remains the first-line imaging modality for evaluating obesity-related cardiac remodeling because it is non-invasive, widely available, and capable of assessing both cardiac structure and function.[7] Conventional Doppler echocardiography allows assessment of transmitral inflow patterns, whereas tissue Doppler imaging (TDI), three-

dimensional (3D) echocardiography, and speckle-tracking echocardiography provide increasingly sensitive measures of ventricular mechanics.[8,9] In particular, global longitudinal strain (GLS) has emerged as a sensitive marker of subclinical LV systolic dysfunction in obese individuals with preserved LVEF, frequently detecting myocardial impairment before conventional systolic indices become abnormal.[8] Likewise, assessment of left atrial function and epicardial adipose tissue (EAT) has enhanced understanding of obesity-associated myocardial remodeling because EAT acts as a metabolically active visceral fat depot capable of promoting local inflammation, myocardial fibrosis, and diastolic dysfunction.[10]

Despite these advances, most published studies have evaluated obese patients with accompanying metabolic disorders such as hypertension or diabetes mellitus, making it difficult to determine the independent effects of obesity on cardiac structure and function.[11,12] Furthermore, relatively limited data are available from healthy Indian adults, in whom body composition, visceral adiposity, and cardiovascular risk differ from Western populations.[13,14] Evaluation of obesity before the development of overt cardiometabolic disease may provide valuable insights into the earliest manifestations of cardiac remodeling and identify individuals who may benefit from preventive interventions.[15]

Therefore, the present study aimed to compare left ventricular structural and functional echocardiographic parameters between apparently healthy obese and non-obese adults from Western India. By evaluating conventional echocardiographic indices in individuals free from major cardiovascular comorbidities, this study seeks to characterize early obesity-associated alterations in LV remodeling while minimizing the confounding effects of established cardiovascular disease.

MATERIALS AND METHODS

Study design & population

This was an institution-based analytical cross-sectional study conducted between April 2023 and April 2024 in the Echocardiography Laboratory and Department of Physiology, Government Medical College, Vadodara, India.

Sample size was estimated using the comparison of mean left ventricular mass index reported by previous studies.[4,14] Assuming an expected standardized effect size of 0.70, a two-sided α error of 0.05, and study power of 80%, the minimum required sample size was calculated as 33 participants per group using G*Power version 3.1. After considering approximately 20% attrition or incomplete examinations, at least 40 participants were required in each group. To improve statistical precision, 50 obese and 50 non-obese participants were finally recruited.

Eligible participants were recruited using consecutive sampling, whereby all apparently healthy adults who fulfilled the eligibility criteria and provided written informed consent during the study period were invited to participate until the target sample size was achieved.

Participants were subsequently categorized according to the World Health Organization BMI criteria into two groups: obese (BMI ≥30 kg/m²) and non-obese (BMI <30 kg/m²). Recruitment continued until 50 obese and 50 non-obese participants were enrolled.

Individuals with hypertension, diabetes mellitus, ischemic heart disease, bronchial asthma, hypercholesterolemia, congenital or acquired heart disease, endocrine disorders, acute or chronic systemic illness, or those receiving medications known to influence cardiac function were excluded. Competitive athletes were also excluded to minimize the influence of physiological cardiac remodeling. Institutional ethics committee approved the protocol of this study vide letter no. IECBHR/193-2023. Written informed consent was obtained from all the participants before the commencement of the study.

All participants underwent a detailed clinical evaluation that included demographic characteristics, medical history, family history, smoking and alcohol consumption, physical activity, and medication use. Cardiovascular and endocrine disorders were excluded through clinical examination and review of available medical records.

Anthropometric measurements were performed using standardized techniques. Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale with participants wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared (kg/m²).

Echocardiography: All participants underwent comprehensive two-dimensional (2D), M-mode, pulsed-wave Doppler and tissue Doppler echocardiography using a commercially available Siemens ACUSON ultrasound system equipped with a phased-array transducer (2–4 MHz). All examinations were performed according to the recommendations of the American Society of Echocardiography. All measurements were performed offline by a single experienced observer who was blinded to participants' clinical and anthropometric characteristics. Measurements were averaged over three consecutive cardiac cycles to minimize beat-to-beat variability.

The following parameters of left ventricular structure and function were evaluated:

Left ventricular systolic function was assessed by measuring:

- Left ventricular ejection fraction (LVEF)
- Mitral annular systolic velocity (s′ or MASV) using pulsed-wave tissue Doppler imaging from the septal mitral annulus in the apical four-chamber view.

Left ventricular mass (LVM) was calculated using the Devereux corrected cube formula recommended by the American Society of Echocardiography:

$$\text{LVM (g)} = 0.8 \times [1.04 \times ((IVSd + LVIDd + PWTd)^3 - (LVIDd)^3)] + 0.6$$

Left ventricular mass index (LVMI) was calculated by indexing LVM to body surface area (g/m²).

Left Ventricular Diastolic Function

Diastolic function was evaluated using pulsed-wave Doppler and tissue Doppler imaging.

Transmitral inflow was recorded from the apical four-chamber view with the Doppler sample volume positioned at the tips of the mitral valve leaflets. The following parameters were measured:

- Peak early diastolic transmitral velocity (E wave)

- Peak late diastolic transmitral velocity (A wave)
- E/A ratio
- Isovolumic relaxation time (IVRT) (if measured)

Using pulsed-wave tissue Doppler imaging, the following mitral annular velocities were obtained:

- Early diastolic velocity (e′)
- Late diastolic velocity (a′)
- Systolic velocity (s′)

The average E/e′ ratio was calculated as an estimate of left ventricular filling pressure.

Statistical analysis

Data were entered into Microsoft Excel and analysed using Jamovi software (Version 2.6). The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables are presented as mean ± standard deviation (SD), while categorical variables are presented as frequencies and percentages. Comparisons between obese and non-obese participants were performed using the independent samples Student's t-test for continuous variables and the Chi-square test for categorical variables, where applicable. Pearson's correlation coefficient (r) was calculated to evaluate the relationship between BMI and echocardiographic parameters.

To determine whether BMI was independently associated with echocardiographic indices, multivariable linear regression analysis was performed after adjusting for age and sex, Regression coefficients with 95% confidence intervals (CI) were reported. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Study Population

A total of 100 apparently healthy adults were enrolled in the study, comprising 50 obese and 50 non-obese participants. The overall study population included 64 males and 36 females, with an age range of 20–60 years. There was no statistically significant difference in age between the two groups (Table 1). Body weight and BMI were significantly higher in the obese group (both p<0.001), whereas height was comparable between groups.

Table- 1 : Demographic data of study population. (values are Mean±SD).

S.No.	Parameters	Non obese (N=50)	Obese (N=50)	P value
1.	Age (years)	42.8 ± 13.5	45.0 ± 8.64	0.267
2.	Height (cm)	166.4 ± 9.2	163.6 ± 11.02	0.169
3.	Weight (kg)	67.48 ± 13.6	100.6 ± 17.9	<0.001
4.	BMI (kg/m^2)	23.9± 3.9	37.5 ± 5.5	<0.001

BMI-Body mass index

Left Ventricular Structural Parameters

Obese participants demonstrated significantly greater left ventricular mass index (LVMI) than non-obese participants (171.2 ± 51.6 vs. 124.9 ± 39.2 g/m², p<0.001), mean difference 46.3 g/m² (95% CI 28.7–63.9; p<0.001), indicating early structural cardiac remodeling associated with obesity (Table 2). The increase in LVMI remained the most prominent structural difference observed between the study groups.

TABLE- 2: Comparison of Echocardiography parameters in obese and nonobese participants (values are Mean±SD).

Echocardiographic parameters	Non obese (N=50)	Obese (N=50)	P-value
LV Mass Index	124.9±39.2	171.2±51.6	<0.001
Ejection fraction	63.1 ± 6.0	61.1 ± 5.0	0.07
Mitral early diastolic (E) wave	83 ± 19.8	85.6 ± 30.1	0.73
Mitral late diastolic (A)wave	68.3±17.9	88.5±25.6	<0.001
E/A ratio	1.33 ±0.57	0.9 ±0.31	<0.001
Mitral Annular Systolic Velocity (MASV or s')	8.7±2.1	8.8±2.5	0.82
Isovolumic relaxation time (IVRT)	81.8 ±13.7	80.7±11.9	0.66
Early diastolic velocity (e′)	9.3 ±2.2	8.7±2.2	0.17
Late diastolic velocity (a′)	10 ± 2.9	10.7±2.8	0.22
E/e' ratio	9.3 ± 2.8	10.1 ± 4.9	0.15

Left Ventricular Systolic Function

Conventional indices of left ventricular systolic function remained preserved in both groups. Left ventricular ejection fraction did not differ significantly between obese and non-obese participants (61.1 ± 5.0% vs. 63.1 ± 6.0%, p=0.07). Likewise, tissue Doppler-derived mitral annular systolic velocity (s′) was comparable between groups (p=0.82), suggesting preserved global systolic despite increased LV mass.

Left Ventricular Diastolic Function

Assessment of transmitral inflow demonstrated significant differences in diastolic filling characteristics between the groups. Obese participants had significantly higher late diastolic mitral inflow velocity (A wave) than non-obese participants (88.5 ± 25.6 vs. 68.3 ± 17.9 cm/s, p<0.001), whereas early filling velocity (E wave) was similar between groups (p=0.73).

Consequently, the E/A ratio was significantly lower in obese individuals (0.90 ± 0.31 vs. 1.33 ± 0.57, p<0.001), suggesting impaired left ventricular relaxation.

Although IVRT, e′ velocities, and E/e′ ratio showed trends toward altered diastolic function in obese participants, these differences were not statistically significant.

Correlation between BMI and Echocardiographic Parameters

Pearson correlation analysis demonstrated a significant positive correlation between BMI and LV mass index (r = 0.67, p<0.001), indicating progressive structural remodeling with increasing adiposity (Table 3).

Table 3: Correlation of echocardiographic parameters with BMI in study participants. (N=100)

Echocardiography parameters	r value	P value
LV mass index	0.67	<0.001
Ejection fraction	0.06	0.57
Mitral early Diastolic E wave	0.07	0.47
Mitral late Diastolic A wave	0.4	<0.001
E/A ratio	-0.33	<0.001
Mitral Annular Systolic Velocity (MASV or s')	0.06	0.56
IVRT	-0.08	0.43
Early diastolic velocity (e′)	-0.23	0.023
Late diastolic velocity (a′)	0.13	0.19
E/e'	0.26	0.008

BMI was also positively correlated with mitral A-wave velocity (r = 0.40, p<0.01) and negatively correlated with the E/A ratio (r = –0.33, p<0.01), suggesting worsening diastolic relaxation with increasing BMI.

No significant correlation was observed between BMI and other parameters

Multivariable regression analysis

Multiple linear regression analyses were performed to determine whether BMI was independently associated with left ventricular structural and functional parameters after adjustment for age and sex (Table 4).

Table 4. Multiple linear regression analysis showing independent predictors of echocardiographic parameters

Dependent Variable	Predictor	β Coefficient (95% CI)	p-value
LV Mass Index (g/m²)	Age	0.141 (−0.526 to 0.808)	0.676
	BMI	3.911 (3.024 to 4.797)	<0.001
	Sex	−24.280 (−39.919 to −8.640)	0.003
	Model fit	R^2 = 0.503; Adjusted R^2 = 0.488; F(3,96)=32.4	<0.001
Mitral A-wave velocity (cm/s)	Age	0.154 (−0.253 to 0.561)	0.455
	BMI	1.166 (0.625 to 1.708)	<0.001
	Sex	2.804 (−6.750 to 12.357)	0.562
	Model fit	R^2 = 0.169; Adjusted R^2 = 0.143; F(3,96)=6.50	<0.001
E/A Ratio	Age	−0.012 (−0.020 to −0.004)	0.004
	BMI	−0.018 (−0.029 to −0.007)	0.001
	Sex	−0.095 (−0.285 to 0.095)	0.322
	Model fit	R^2 = 0.184; Adjusted R^2 = 0.159; F(3,96)=7.22	<0.001

Multiple linear regression models were adjusted for age and sex. Regression coefficients (β) are presented with 95% confidence intervals.

For left ventricular mass index (LVMI), the overall model was statistically significant (R^2 = 0.503; adjusted R^2 = 0.488; F(3,96)=32.4; p<0.001). BMI emerged as the strongest independent predictor of LVMI (β = 3.911, 95% CI: 3.024–4.797; p<0.001), indicating that each 1 kg/m² increase in BMI was associated with an average increase of approximately 3.9 g/m² in LVMI. Sex also independently predicted LVMI (β = −24.280, 95% CI: −39.919 to −8.640; p=0.003), whereas age was not significantly associated with LVMI (p=0.676).

For mitral A-wave velocity, the regression model was also statistically significant (R^2 = 0.169; adjusted R^2 = 0.143; F(3,96)

=6.50; p<0.001). BMI remained an independent positive predictor of A-wave velocity (β = 1.166, 95% CI: 0.625–1.708; p<0.001), while neither age nor sex demonstrated significant associations.
For E/A ratio, the regression model explained 18.4% of the variance (R^2 = 0.184; adjusted R^2 = 0.159; F(3,96) =7.22; p<0.001). Both BMI (β = −0.018, 95% CI: −0.029 to −0.007; p=0.001) and age (β = −0.012, 95% CI: −0.020 to −0.004; p=0.004) were independently associated with lower E/A ratio, whereas sex was not a significant predictor (p=0.322).

DISCUSSION

This cross-sectional study evaluated the influence of obesity on left ventricular (LV) structure and function in apparently healthy adults using conventional and tissue Doppler echocardiography. The principal findings were: (i) obese participants had significantly greater left ventricular mass index (LVMI), indicating early structural cardiac remodeling; (ii) conventional measures of LV systolic function, including ejection fraction (LVEF) and mitral annular systolic velocity (s′), remained preserved; (iii) obese individuals showed altered diastolic filling, characterized by increased transmitral A-wave velocity and reduced E/A ratio, consistent with impaired LV relaxation; and (iv) multivariable regression confirmed that BMI was an independent predictor of LVMI, A-wave velocity, and E/A ratio after adjustment for age and sex. Together, these findings indicate that increasing adiposity is independently associated with early structural remodeling and subtle diastolic impairment, occurring well before any overt systolic dysfunction develops.

LV mass and structural remodeling

The increase in LVMI among obese participants is consistent with prior evidence that obesity independently promotes myocardial remodeling, even in the absence of hypertension or diabetes. Abdeen and Marghalani (2024) reported significantly greater LV wall thickness and mass in obese adults compared with normal-weight controls,[6] and Prajapati et al. (2025) demonstrated progressive remodeling across increasing obesity classes.[15] Similar findings have been reported in large population-based cohorts such as the Framingham Heart Study, where increasing adiposity was linked to adverse cardiac geometric changes independent of traditional cardiovascular risk factors.[16] Several other studies have likewise shown increased LV mass in obese individuals regardless of age or sex,[6,14] although a small number have reported no significant difference between obese and non-obese groups.[17] Collectively, this findings supports the hypothesis that obesity itself, independent of comorbidities, contributes substantially to myocardial structural remodeling.
The likely mechanisms underlying this increase in LV mass include relative expansion of intravascular volume, hyperinsulinemia, and disrupted leptin signaling, all of which are common in obesity and capable of inducing myocardial hypertrophy.[12] More broadly, chronic volume overload raises myocardial wall stress and drives compensatory hypertrophy, while activation of the renin–angiotensin–aldosterone system, sympathetic overactivity, insulin resistance, low-grade systemic inflammation, oxidative stress, and adipokine dysregulation all contribute to hypertrophy and interstitial fibrosis.[12] Lipotoxicity from ectopic myocardial fat deposition further impairs myocardial energetics and ventricular compliance.[18] The elevated LVMI observed in this study therefore most likely represents an early, compensatory remodeling response rather than established myocardial failure.
Notably, despite the relatively young age of our participants and the absence of major cardiovascular and metabolic comorbidities, obese individuals demonstrated evidence of early left ventricular structural remodeling, consistent with previous reports in otherwise healthy obese populations.[19] Studies have shown structural LV remodeling has been associated with adverse cardiovascular outcomes and prognosis in both normotensive and hypertensive cohorts.[11,20] Although previous longitudinal studies suggest that persistent obesity may be associated with progressive myocardial remodeling and eventual heart failure in susceptible individuals, the present cross-sectional study cannot establish temporal progression or causality.[21,22,23]
This study used multivariable regression to test whether obesity influences remodeling independent of age and sex. BMI remained the strongest independent predictor of LVMI, with each 1 kg/m² increase in BMI associated with an approximate 3.9 g/m² increase in LVMI; the model explained nearly half of the observed variability (adjusted R^2 = 0.488). This suggests that adiposity is a major independent contributor to myocardial structural remodeling, a finding that aligns with recent echocardiographic and cardiac MRI studies reinforcing obesity as an independent driver of LV hypertrophy.[24]

LV Systolic function

Despite the increase in LV mass, conventional systolic function was preserved in obese participants: LVEF and mitral annular systolic velocity (s′) did not differ significantly between groups. Previous studies have shown similar LVEF, mitral inflow velocities, isovolumic relaxation time, and E-wave deceleration time between obese and non-obese groups, suggesting that otherwise-healthy obese individuals can have normal conventional systolic and diastolic indices alongside an elevated LV mass index.[6,7,14,15]
However, preserved LVEF should not be equated with the absence of myocardial involvement, since conventional systolic indices often remain normal until relatively advanced myocardial disease has developed. Speckle-tracking studies have repeatedly demonstrated impaired global longitudinal strain (GLS) in obese individuals despite normal LVEF, pointing to subclinical myocardial dysfunction.[9] Abomandour et al. showed that speckle-tracking echocardiography can detect early impairment of myocardial deformation in obese adults with preserved ejection fraction, underscoring the limitations of relying on conventional systolic parameters alone.[8] Our findings therefore suggest that preserved LVEF in this cohort reflects maintained global pump function rather than the absence of early myocardial abnormality.
A further consideration is that commonly used indices of LV function including LVEF and mitral inflow velocities are influenced by preload and afterload (e.g., elevated blood pressure or wall stress).[11] Abnormalities in these indices may therefore partly reflect altered loading conditions rather than a true change in myocardial contractility. This study addressed that limitation in part by incorporating tissue Doppler-derived indices of systolic and diastolic function, which are relatively

load-independent.[25]

LV Diastolic function

Obesity was also associated with impaired LV diastolic filling. Obese participants had significantly higher transmitral A-wave velocity and lower E/A ratio than non-obese controls, consistent with impaired myocardial relaxation. Multivariable regression confirmed that BMI remained independently associated with both parameters after adjustment for age and sex: each unit increase in BMI was associated with a 1.17 cm/s increase in A-wave velocity and a 0.018-unit reduction in E/A ratio, indicating progressively impaired relaxation with increasing adiposity. Age was independently associated with lower E/A ratio, reflecting the expected physiological decline in diastolic function with age, while sex had no independent effect on diastolic indices after adjustment. These findings suggest obesity impairs LV relaxation over and above the effects of normal ageing.

This is consistent with previous reports of increased A-wave velocity and reduced E/A ratio in obese adults without overt cardiovascular disease.[14,15] However, current American Society of Echocardiography/European Association of Cardiovascular Imaging (ASE/EACVI) recommendations specify that comprehensive grading of diastolic function requires integrating transmitral inflow velocities with tissue Doppler e′ velocity, E/e′ ratio, left atrial volume index (LAVI), and peak tricuspid regurgitation velocity.[26] While tissue Doppler parameters were included in this study, LAVI and tricuspid regurgitation velocity were not available; our findings should therefore be interpreted as evidence of altered LV relaxation rather than formal grading of diastolic dysfunction.

Although the study population comprised apparently healthy adults without major cardiovascular or metabolic comorbidities, the observational cross-sectional design limits causal inference, and the temporal evolution of the observed myocardial remodeling cannot be determined.

Emerging evidence highlights epicardial adipose tissue (EAT) as a potential mediator of obesity-associated myocardial remodeling. EAT is a metabolically active visceral fat depot adjacent to the myocardium and coronary arteries that secretes inflammatory cytokines, profibrotic mediators, and adipokines capable of directly affecting myocardial structure and function.[27,28] A recent multimodal review emphasized a strong association between increased EAT thickness and LV hypertrophy, impaired diastolic function, atrial remodeling, and adverse cardiovascular outcomes.[10,27] EAT was not measured in this study, so its contribution to the structural and functional changes observed here could not be evaluated; future studies incorporating routine EAT assessment may offer additional mechanistic insight.

It is also worth noting that severe obesity can meaningfully reduce transthoracic echocardiographic image quality and agreement with cardiac MRI, suggesting that multimodal imaging may be warranted when assessing individuals with severe obesity.[29]

The present findings have important clinical implications. Although conventional systolic function remained preserved, the presence of increased LV mass index and altered diastolic filling characteristics suggests that obesity-related cardiac remodeling may begin before overt cardiovascular disease becomes clinically evident. This highlights the potential value of targeted cardiovascular evaluation in obese individuals, particularly those with additional risk factors or prolonged obesity. Conventional and tissue Doppler echocardiography may facilitate identification of individuals at an early stage of myocardial remodeling, allowing timely implementation of weight reduction strategies, regular physical activity, dietary modification, and optimization of cardiovascular risk factors before irreversible structural changes occur.

Limitations

Several limitations should also be considered. First, the cross-sectional design precludes conclusions regarding causality or temporal progression of myocardial remodeling. Second, obesity was defined using BMI alone without assessment of waist circumference, waist-to-hip ratio, visceral adiposity, or body fat percentage, which may better reflect metabolically adverse obesity. Third, advanced echocardiographic techniques such as global longitudinal strain, left atrial strain, comprehensive right ventricular functional assessment, three-dimensional echocardiography, and epicardial adipose tissue quantification were not available. Fourth, LV mass was indexed to body surface area in accordance with ASE recommendations; however, indexing by height$^{2.7}$ may better characterize obesity-related hypertrophy and should be considered in future studies. Finally, although multivariable regression adjusted for age and sex, residual confounding from unmeasured factors cannot be excluded. Large multicenter prospective studies incorporating advanced echocardiographic techniques and long-term clinical follow-up including follow-up after weight-loss or lifestyle interventions and studies in specific groups may validate and extend these findings.

CONCLUSION

Apparently healthy obese adults demonstrate early left ventricular structural remodeling and subtle impairment of diastolic function despite preserved conventional systolic function. BMI is independently associated with increased LV mass index and altered diastolic filling characteristics after adjustment for age and sex. These findings suggest that conventional and tissue Doppler echocardiography may facilitate early detection of obesity-related myocardial remodeling before overt cardiac dysfunction becomes clinically apparent. Early recognition of these subclinical changes may support timely lifestyle interventions and cardiovascular risk reduction strategies aimed at preventing progression to clinically significant cardiac dysfunction.

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