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ASSOCIATION OF VISCERAL OBESITY WITH LEFT VENTRICULAR HYPERTROPHY AND DIASTOLIC DYSFUNCTION

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ABSTRACT

Introduction: Visceral obesity is increasingly recognized as an important cardiovascular risk factor associated with adverse cardiac remodeling, left ventricular hypertrophy (LVH) and diastolic dysfunction. Early identification of obesity-related cardiac abnormalities may help prevent progression to overt cardiovascular disease and heart failure. Aim of the study was to evaluate the association of visceral obesity with left ventricular hypertrophy and diastolic dysfunction.

Materials and Methods: This hospital-based cross-sectional observational study was conducted in the Department of General Medicine among 100 adult subjects. Clinical history, anthropometric measurements including body mass index (BMI), waist circumference and waist–hip ratio were recorded. Visceral obesity was assessed using standard anthropometric criteria. All participants underwent echocardiographic evaluation for assessment of left ventricular mass index and diastolic function. Data were analyzed using SPSS software, and p-value <0.05 was considered statistically significant.

Results: Visceral obesity was present in 68% of the study population. Left ventricular hypertrophy was observed in 46% and diastolic dysfunction in 52% of subjects. Patients with visceral obesity had significantly higher prevalence of LVH (87.0% vs 51.9%, $p<0.001$) and diastolic dysfunction (84.6% vs 50.0%, $p<0.001$). Waist circumference showed significant positive correlation with left ventricular mass index and E/e′ ratio. Visceral obesity was identified as an independent predictor of both LVH and diastolic dysfunction.

Conclusion: Visceral obesity is strongly associated with left ventricular hypertrophy and diastolic dysfunction. Early cardiovascular evaluation in individuals with visceral obesity may help detect subclinical cardiac dysfunction and reduce future cardiovascular risk.

Keywords: Visceral obesity; Left ventricular hypertrophy; Diastolic dysfunction; Echocardiography; Waist circumference; Cardiovascular risk; Left ventricular mass index.

INTRODUCTION

Obesity has emerged as one of the most important global public health problems and is strongly associated with cardiovascular morbidity and mortality. In recent years, increasing attention has been focused not only on generalized obesity but also on visceral obesity, which represents excessive accumulation of adipose tissue within the abdominal cavity surrounding internal organs. Visceral adipose tissue is metabolically active and plays a major role in the development of insulin resistance, dyslipidemia, hypertension, chronic inflammation and cardiovascular disease (1). Compared to subcutaneous fat, visceral fat produces higher levels of inflammatory cytokines, free fatty acids and adipokines, which contribute to endothelial dysfunction, oxidative stress and myocardial remodeling (2). Therefore, visceral obesity is considered a more significant predictor of cardiovascular risk than body mass index alone.

Cardiovascular involvement associated with visceral obesity includes structural as well as functional abnormalities of the heart. One of the major structural alterations is left ventricular hypertrophy, which is characterized by an increase in left ventricular mass resulting from chronic hemodynamic and metabolic stress. Left ventricular hypertrophy is a well-recognized independent predictor of heart failure, arrhythmias, ischemic heart disease and sudden cardiac death (3). Traditionally, hypertension has been considered the primary cause of left ventricular hypertrophy; however, recent evidence suggests that visceral obesity independently contributes to left ventricular remodeling even in the absence of overt hypertension (4). Increased blood volume, elevated cardiac output, activation of the renin–angiotensin–aldosterone system, sympathetic overactivity and chronic inflammatory pathways associated with visceral adiposity may promote myocardial hypertrophy and fibrosis (5).

Diastolic dysfunction is another important cardiac abnormality associated with visceral obesity. It is characterized by impaired relaxation and reduced compliance of the left ventricle, leading to elevated ventricular filling pressures. Diastolic dysfunction often precedes systolic dysfunction and may remain asymptomatic for several years before progressing to overt heart failure (6). Visceral obesity has been identified as an important contributor to heart failure with preserved

ejection fraction, where impaired diastolic filling and myocardial stiffness play a central role (7). Excess visceral fat may lead to myocardial lipid accumulation, inflammatory activation and interstitial fibrosis, thereby impairing ventricular relaxation and compliance. In addition, obesity-related metabolic abnormalities such as insulin resistance and hyperinsulinemia further aggravate myocardial dysfunction (8).

Several studies have evaluated the relationship between visceral adiposity and cardiac abnormalities. Powell-Wiley et al. emphasized the strong association between obesity and cardiovascular remodeling, highlighting the role of visceral adipose tissue in the development of left ventricular hypertrophy and heart failure (1). Ren et al. described obesity cardiomyopathy as a distinct clinical entity characterized by myocardial hypertrophy, fibrosis and diastolic dysfunction resulting from obesity-associated metabolic and inflammatory changes (5). Sawada et al. demonstrated that visceral adiposity was significantly associated with impaired left ventricular diastolic function and altered adipokine levels in the general population (6). Nagata et al. further reported that visceral adiposity contributes to left ventricular diastolic dysfunction through inflammatory and fibrotic mechanisms affecting myocardial structure and function (7). Xu et al. found a significant association between visceral adiposity index and abnormal left ventricular geometry, left ventricular hypertrophy and diastolic dysfunction (9). Similarly, Wang et al. reported that increased metabolic visceral fat score was independently associated with left ventricular hypertrophy in patients with type 2 diabetes mellitus (10).

Although previous studies have demonstrated a relationship between obesity and cardiovascular abnormalities, important research gaps still remain. Many earlier studies assessed obesity using body mass index, which does not accurately reflect visceral fat distribution. Individuals with normal body mass index may still possess significant visceral adiposity and increased cardiovascular risk. Therefore, anthropometric indicators of visceral obesity such as waist circumference and waist–hip ratio may provide better assessment of obesity-related cardiac risk than body mass index alone (2). Furthermore, several studies evaluated either left ventricular hypertrophy or diastolic dysfunction independently, whereas fewer studies have simultaneously assessed both structural and functional cardiac abnormalities in relation to visceral obesity. There is also limited Indian data evaluating the association between visceral obesity and echocardiographic evidence of left ventricular hypertrophy and diastolic dysfunction despite the increasing prevalence of abdominal obesity and metabolic syndrome among South Asian populations (11).

Early identification of subclinical cardiac dysfunction associated with visceral obesity is clinically important because these changes may be reversible with lifestyle modification, weight reduction and risk factor control. Echocardiography provides a non-invasive and reliable method for assessing left ventricular mass and diastolic function and may help identify individuals at increased cardiovascular risk before the onset of symptomatic heart disease. Understanding the relationship between visceral obesity and cardiac remodeling may therefore contribute to improved prevention and management strategies for obesity-related cardiovascular disease.

Hence, the present study is undertaken to evaluate the association of visceral obesity with left ventricular hypertrophy and diastolic dysfunction using clinical, anthropometric and echocardiographic parameters.

MATERIALS AND METHODS

Study Design

The present study was designed as a hospital-based cross-sectional observational study conducted to evaluate the association of visceral obesity with left ventricular hypertrophy and diastolic dysfunction.

Study Setting

The study was conducted in the Department of General Medicine at a tertiary care teaching hospital. Patients attending the outpatient and inpatient departments of General Medicine who fulfilled the eligibility criteria were included in the study.

Study Duration

The study was conducted over a period of ___6___ months from __February 2025__ to __July 2025__.

Sample Size

A total of 100 subjects were included in the study.

Study Population

Adult patients attending the Department of General Medicine were screened for visceral obesity and enrolled in the study after obtaining informed written consent. Subjects were evaluated clinically and underwent anthropometric assessment and echocardiographic evaluation for detection of left ventricular hypertrophy and diastolic dysfunction.

Inclusion Criteria

- Patients aged more than 18 years.
- Patients willing to participate in the study and provide informed consent.
- Patients with visceral obesity based on anthropometric parameters such as increased waist circumference and/or waist–hip ratio according to standard criteria.
- Patients undergoing echocardiographic evaluation during the study period.

Exclusion Criteria
- Patients with known ischemic heart disease.
- Patients with congenital heart disease.
- Patients with significant valvular heart disease.
- Patients with cardiomyopathy or heart failure with reduced ejection fraction.
- Patients with chronic kidney disease.
- Patients with chronic liver disease.
- Pregnant women.
- Patients with severe systemic illness or malignancy.
- Patients unwilling to participate in the study.

Study Tools
The following tools and instruments were used for data collection and evaluation:
- Pre-designed and pre-tested structured proforma.
- Measuring tape for waist circumference and hip circumference measurements.
- Weighing machine and stadiometer for body mass index calculation.
- Sphygmomanometer for blood pressure measurement.
- Standard laboratory investigations including blood glucose and lipid profile.
- Two-dimensional echocardiography with Doppler assessment for evaluation of left ventricular hypertrophy and diastolic dysfunction.

Data Collection Procedure
- Detailed history regarding demographic profile, lifestyle factors, comorbidities and cardiovascular risk factors was obtained from all study participants.
- General physical examination and systemic examination were carried out in all patients.
- Anthropometric measurements including height, weight, waist circumference and hip circumference were recorded using standard methods.
- Body mass index was calculated using the formula:

$$\text{BMI} = \frac{\text{Weight in kg}}{(\text{Height in meter})^2}$$

- Waist–hip ratio was calculated by dividing waist circumference by hip circumference.
- Visceral obesity was assessed based on waist circumference and waist–hip ratio according to standard guidelines.
- Blood pressure was recorded in the sitting position after adequate rest.
- Relevant laboratory investigations including fasting blood glucose and lipid profile were performed.
- All subjects underwent transthoracic two-dimensional echocardiography performed by a qualified cardiologist.
- Left ventricular hypertrophy was assessed by calculating left ventricular mass index and evaluating left ventricular wall thickness.
- Diastolic dysfunction was assessed using Doppler echocardiographic parameters including E/A ratio, E/e′ ratio, deceleration time and left atrial size.
- Findings were recorded systematically in the study proforma for further analysis.
- Outcome Measures
- The primary outcome measures assessed in the study were:
- Presence of left ventricular hypertrophy.
- Presence and grade of left ventricular diastolic dysfunction.
- Association between visceral obesity parameters and echocardiographic abnormalities.

Statistical Analysis
The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 23.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Chi-square test was used for comparison of categorical variables, and independent t-test was used for comparison of continuous variables between groups. Correlation analysis was performed to assess the relationship between visceral obesity parameters and echocardiographic findings. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n = 100)

Variable	Value
Age (years)	52.6 ± 11.4
Male	62 (62.0%)
Female	38 (38.0%)
BMI (kg/m²)	29.4 ± 4.2
Waist circumference (cm)	101.8 ± 10.6
Waist–hip ratio	0.98 ± 0.08

Hypertension	54 (54.0%)
Diabetes mellitus	42 (42.0%)
Dyslipidemia	48 (48.0%)
Smoking	28 (28.0%)
Alcohol consumption	24 (24.0%)

The present study included 100 participants with a mean age of 52.6 ± 11.4 years, indicating that the majority of subjects belonged to the middle-aged population. Male participants constituted 62% of the study population, while females accounted for 38%. The mean body mass index was 29.4 ± 4.2 kg/m², with increased mean waist circumference (101.8 ± 10.6 cm) and waist–hip ratio (0.98 ± 0.08), suggesting a high prevalence of visceral obesity among the study subjects. Common associated cardiovascular risk factors included hypertension (54%), dyslipidemia (48%) and diabetes mellitus (42%). In addition, 28% of participants were smokers and 24% reported alcohol consumption, which may further contribute to adverse cardiovascular outcomes and cardiac remodeling.

Table 2: Anthropometric and Metabolic Parameters According to Presence of Visceral Obesity

Parameter	Visceral Obesity Present (n=68)	Visceral Obesity Absent (n=32)	p-value
BMI (kg/m²)	31.2 ± 3.8	25.8 ± 2.9	<0.001*
Waist circumference (cm)	108.6 ± 8.4	89.4 ± 6.8	<0.001*
Waist–hip ratio	1.02 ± 0.06	0.89 ± 0.05	<0.001*
Systolic BP (mmHg)	142.6 ± 14.2	128.4 ± 10.6	<0.001*
Diastolic BP (mmHg)	88.2 ± 8.4	80.6 ± 6.2	<0.001*
Fasting blood glucose (mg/dL)	132.4 ± 32.6	108.2 ± 24.4	0.002*
Triglycerides (mg/dL)	198.6 ± 42.8	146.2 ± 34.6	<0.001*
HDL cholesterol (mg/dL)	38.4 ± 6.2	46.8 ± 7.4	<0.001*

Patients with visceral obesity demonstrated significantly higher anthropometric and metabolic risk parameters compared to those without visceral obesity. The mean BMI, waist circumference and waist–hip ratio were markedly elevated in the visceral obesity group, indicating greater central adiposity. Blood pressure levels were also significantly higher among subjects with visceral obesity, suggesting increased cardiovascular risk. Metabolic abnormalities including elevated fasting blood glucose and triglyceride levels were more common in patients with visceral obesity, while HDL cholesterol levels were significantly lower. All these differences were statistically significant, highlighting the strong association between visceral obesity and adverse cardiometabolic profile.

Table 3: Echocardiographic Characteristics of the Study Population (n = 100)

Echocardiographic Parameter	Mean ± SD / n (%)
Interventricular septal thickness (mm)	11.8 ± 2.1
Posterior wall thickness (mm)	11.2 ± 1.8
Left ventricular mass index (g/m²)	122.4 ± 28.6
Relative wall thickness	0.46 ± 0.08
Left atrial diameter (mm)	39.2 ± 5.6
Ejection fraction (%)	58.6 ± 5.4
E/A ratio	0.88 ± 0.24
E/e′ ratio	13.6 ± 3.8
Deceleration time (ms)	232.4 ± 34.6
Left ventricular hypertrophy	46 (46.0%)
Diastolic dysfunction	52 (52.0%)

The echocardiographic evaluation in the present study demonstrated significant structural and functional cardiac abnormalities among the study population. The mean interventricular septal thickness and posterior wall thickness were elevated, indicating increased myocardial wall thickness associated with cardiac remodeling. The mean left ventricular mass index was 122.4 ± 28.6 g/m², and left ventricular hypertrophy was observed in 46% of subjects, suggesting a high prevalence of obesity-related myocardial hypertrophy. Diastolic dysfunction was present in 52% of participants, with reduced E/A ratio and elevated E/e′ ratio indicating impaired left ventricular relaxation and increased filling pressures. Although the mean ejection fraction remained preserved, these findings highlight the presence of subclinical cardiac dysfunction associated with visceral obesity and increased cardiovascular risk.

Table 4: Association of Visceral Obesity with Left Ventricular Hypertrophy

Variable	LVH Present (n=46)	LVH Absent (n=54)	p-value
Visceral obesity	40 (87.0%)	28 (51.9%)	<0.001*
Increased waist circumference	38 (82.6%)	24 (44.4%)	<0.001*
Increased waist–hip ratio	36 (78.3%)	22 (40.7%)	<0.001*
Hypertension	34 (73.9%)	20 (37.0%)	<0.001*
Diabetes mellitus	24 (52.2%)	18 (33.3%)	0.048*

Dyslipidemia	28 (60.9%)	20 (37.0%)	0.019*

The present study demonstrated a significant association between visceral obesity and left ventricular hypertrophy (LVH). Visceral obesity was observed in 87.0% of subjects with LVH compared to 51.9% among those without LVH, which was statistically highly significant (p<0.001). Similarly, increased waist circumference and elevated waist–hip ratio were significantly more common among patients with LVH, indicating the important role of central adiposity in cardiac remodeling. Traditional cardiovascular risk factors such as hypertension, diabetes mellitus and dyslipidemia also showed significant association with LVH. These findings suggest that visceral obesity, along with associated metabolic abnormalities, contributes substantially to the development of left ventricular hypertrophy and adverse cardiovascular changes.

Table 5: Association of Visceral Obesity with Diastolic Dysfunction

Variable	Diastolic Dysfunction Present (n=52)	Diastolic Dysfunction Absent (n=48)	p-value
Visceral obesity	44 (84.6%)	24 (50.0%)	<0.001*
Increased waist circumference	42 (80.8%)	20 (41.7%)	<0.001*
Increased waist–hip ratio	40 (76.9%)	18 (37.5%)	<0.001*
Hypertension	36 (69.2%)	18 (37.5%)	0.002*
Diabetes mellitus	26 (50.0%)	16 (33.3%)	0.041*
Dyslipidemia	30 (57.7%)	18 (37.5%)	0.044*

The present study showed a significant association between visceral obesity and left ventricular diastolic dysfunction. Visceral obesity was present in 84.6% of patients with diastolic dysfunction compared to 50.0% among those without diastolic dysfunction, which was statistically highly significant (p<0.001). Increased waist circumference and elevated waist–hip ratio were also significantly more common among subjects with diastolic dysfunction, indicating that central adiposity plays an important role in impaired ventricular relaxation and increased filling pressures. Among the associated comorbidities, hypertension, diabetes mellitus and dyslipidemia showed significant association with diastolic dysfunction. These findings suggest that visceral obesity and related metabolic risk factors contribute substantially to subclinical myocardial dysfunction and adverse cardiac remodeling.

Table 6: Correlation Between Anthropometric Parameters and Echocardiographic Variables

Variables Compared	Correlation Coefficient (r)	p-value
Waist circumference vs LV mass index	+0.62	<0.001*
Waist circumference vs E/e′ ratio	+0.58	<0.001*
Waist–hip ratio vs LV mass index	+0.54	<0.001*
Waist–hip ratio vs E/A ratio	−0.48	<0.001*
BMI vs LV mass index	+0.42	0.002*
BMI vs E/e′ ratio	+0.38	0.006*

Correlation analysis in the present study demonstrated a significant relationship between anthropometric measures of obesity and echocardiographic parameters of cardiac remodeling and diastolic dysfunction. Waist circumference showed a strong positive correlation with left ventricular mass index (r = +0.62) and E/e′ ratio (r = +0.58), indicating that increasing visceral adiposity was associated with greater myocardial hypertrophy and elevated left ventricular filling pressures. Waist–hip ratio also demonstrated significant positive correlation with left ventricular mass index and a negative correlation with E/A ratio, suggesting worsening diastolic relaxation with increasing central obesity. Body mass index showed moderate positive correlation with both left ventricular mass index and E/e′ ratio. All correlations were statistically significant, emphasizing the strong association between visceral obesity and adverse structural and functional cardiac changes.

Table 7: Multivariate Logistic Regression Analysis for Predictors of Left Ventricular Hypertrophy

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Visceral obesity	4.82	2.01 – 10.64	<0.001*
Hypertension	3.64	1.72 – 7.92	0.002*
Diabetes mellitus	1.88	0.92 – 3.84	0.086
Age > 50 years	2.14	1.08 – 4.42	0.031*
Male gender	1.42	0.68 – 2.96	0.218

Multivariate logistic regression analysis in the present study identified visceral obesity as a strong independent predictor of left ventricular hypertrophy, with an odds ratio of 4.82 (95% CI: 2.01–10.64, p<0.001). Hypertension was also found

to be a significant independent risk factor for LVH, increasing the risk by approximately 3.6 times. Age greater than 50 years showed significant association with left ventricular hypertrophy, indicating increased susceptibility to cardiac remodeling with advancing age. Although diabetes mellitus and male gender showed higher odds for LVH, their associations were not statistically significant after adjustment for other confounding variables. These findings highlight the independent contribution of visceral obesity to the development of left ventricular hypertrophy beyond conventional cardiovascular risk factors.

Table 8: Multivariate Logistic Regression Analysis for Predictors of Diastolic Dysfunction

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Visceral obesity	5.26	2.24 – 11.82	<0.001*
Hypertension	3.18	1.46 – 6.94	0.004*
Diabetes mellitus	2.22	1.08 – 4.68	0.036*
Age > 50 years	2.64	1.28 – 5.42	0.012*
Dyslipidemia	1.84	0.92 – 3.66	0.074

Multivariate logistic regression analysis demonstrated that visceral obesity was an independent and significant predictor of diastolic dysfunction, with an odds ratio of 5.26 (95% CI: 2.24–11.82, p<0.001). Hypertension and diabetes mellitus were also independently associated with increased risk of diastolic dysfunction, indicating the important contribution of metabolic and hemodynamic abnormalities to impaired ventricular relaxation. Age greater than 50 years showed a significant association with diastolic dysfunction, reflecting the progressive effect of aging on myocardial compliance and ventricular filling. Although dyslipidemia showed increased odds for diastolic dysfunction, the association did not reach statistical significance after multivariate adjustment. These findings emphasize that visceral obesity is a major independent determinant of left ventricular diastolic dysfunction and subclinical cardiac impairment.

DISCUSSION

The present study was conducted to evaluate the association of visceral obesity with left ventricular hypertrophy (LVH) and diastolic dysfunction among 100 patients attending the Department of General Medicine. The findings of the present study demonstrated a strong association between visceral obesity and adverse cardiac structural and functional changes, particularly LVH and left ventricular diastolic dysfunction. These findings are consistent with several recent PubMed-indexed studies that have highlighted the pathogenic role of visceral adiposity in cardiovascular remodeling.

In the present study, the mean age of the participants was 52.6 ± 11.4 years, with the majority of subjects belonging to the middle-aged and elderly age groups. Similar observations were reported by Omidi et al., who noted that obesity-related cardiac abnormalities were more common among middle-aged individuals due to prolonged metabolic exposure and increasing cardiovascular risk burden with age (12). Increasing age contributes to myocardial stiffness, reduced ventricular compliance and progressive ventricular remodeling, which may potentiate the adverse effects of visceral obesity on cardiac function.

The present study showed male predominance, with males constituting 62% of the study population. Similar gender predominance was observed in the study by Cai et al., where obesity-associated LVH was more frequently observed among men with hypertension (13). This may be explained by the greater tendency for visceral fat accumulation among males compared to females, leading to increased metabolic and inflammatory cardiovascular burden.

In the present study, patients with visceral obesity had significantly higher body mass index, waist circumference and waist–hip ratio compared to those without visceral obesity (p <0.001). Waist circumference and waist–hip ratio were strongly associated with echocardiographic abnormalities. These findings correlate with the observations of Nagata et al., who demonstrated that visceral fat area was more strongly associated with LV remodeling and diastolic dysfunction than body mass index alone (14). Similarly, Goldberg et al. reported that visceral adiposity was independently associated with impaired ventricular relaxation and increased cardiac stiffness, emphasizing the importance of central adiposity as a predictor of cardiovascular dysfunction (15).

The present study also demonstrated significantly higher systolic and diastolic blood pressure levels among individuals with visceral obesity. Hypertension was observed in 54% of the study population and showed significant association with LVH and diastolic dysfunction. Similar findings were reported by Cai et al., who observed that obesity significantly increased the risk of incident LVH in hypertensive individuals (13). Hypertension contributes to increased afterload, myocardial workload and compensatory ventricular hypertrophy. When combined with visceral obesity, the adverse hemodynamic and inflammatory effects may synergistically accelerate cardiac remodeling.

In the present study, dyslipidemia and diabetes mellitus were more common among patients with visceral obesity and were significantly associated with LVH and diastolic dysfunction. This finding is consistent with the study by Verma et al., who emphasized that visceral adiposity and insulin resistance play a central role in the development of cardiac stiffness, myocardial fibrosis and heart failure with preserved ejection fraction (16). Visceral fat contributes to chronic systemic

inflammation, oxidative stress and endothelial dysfunction, thereby aggravating myocardial injury and impaired ventricular compliance.

Echocardiographic evaluation in the present study revealed that the mean left ventricular mass index was significantly elevated, and LVH was present in 46% of the study population. The prevalence of LVH was significantly higher among patients with visceral obesity compared to those without visceral obesity (87.0% vs 51.9%, $p < 0.001$). Similar observations were made by Zhu et al., who reported significantly greater epicardial adipose tissue volume and myocardial fibrosis among hypertensive patients with LVH (17). Their study demonstrated that visceral adiposity independently contributed to LVH even after adjustment for conventional cardiovascular risk factors. Likewise, Cai et al. observed that obesity increased the risk of incident LVH by more than two-fold among hypertensive individuals (13).

The pathophysiological mechanisms underlying obesity-related LVH involve increased blood volume, elevated cardiac output, activation of neurohormonal pathways, chronic inflammation and myocardial fibrosis. Visceral adipose tissue acts as an endocrine organ producing inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, which contribute to myocardial remodeling and hypertrophy. Ayton et al. highlighted the role of epicardial adipose tissue in obesity-related cardiac dysfunction and myocardial inflammation, particularly in the development of LVH and heart failure with preserved ejection fraction (18).

Diastolic dysfunction was identified in 52% of the study population in the present study. Patients with visceral obesity demonstrated significantly greater prevalence of diastolic dysfunction compared to non-viscerally obese individuals (84.6% vs 50.0%, $p < 0.001$). The mean E/e′ ratio was elevated and E/A ratio was reduced among patients with visceral obesity, indicating impaired ventricular relaxation and elevated filling pressures. Similar findings were reported by Nagata et al., who demonstrated that visceral fat area was strongly associated with worse LV diastolic function, higher LV mass index and elevated BNP levels (14). Their study showed that visceral fat rather than subcutaneous fat was a more important determinant of diastolic dysfunction.

The findings of the present study are also supported by the observations of Sawada et al., who demonstrated that visceral adiposity was independently associated with LV diastolic dysfunction and altered adiponectin levels (19). Reduced adiponectin levels and increased inflammatory mediators in obesity contribute to myocardial fibrosis, increased ventricular stiffness and impaired relaxation. These metabolic alterations explain why many obese individuals develop heart failure with preserved ejection fraction despite preserved systolic function.

Correlation analysis in the present study demonstrated significant positive correlation between waist circumference and left ventricular mass index ($r = +0.62$, $p < 0.001$), as well as between waist circumference and E/e′ ratio ($r = +0.58$, $p < 0.001$). These findings indicate that increasing visceral adiposity is associated with progressive worsening of cardiac structure and diastolic function. Similar findings were reported by Martinez-Dominguez et al., who demonstrated that visceral adipose tissue mediated the relationship between insulin resistance and subclinical cardiovascular remodeling (20).

Multivariate logistic regression analysis in the present study identified visceral obesity as an independent predictor of both LVH and diastolic dysfunction even after adjustment for hypertension, diabetes and dyslipidemia. Visceral obesity showed an odds ratio of 4.82 for LVH and 5.26 for diastolic dysfunction. These findings are comparable to the study by Xu et al., who demonstrated significant association between visceral adiposity index and abnormal LV geometry, heart failure risk and diastolic dysfunction (21). The independent predictive value of visceral obesity observed in the present study highlights the importance of assessing central adiposity in routine clinical practice.

The present study therefore confirms that visceral obesity is strongly associated with adverse cardiac remodeling and impaired ventricular function. Echocardiographic screening among individuals with visceral obesity may help identify subclinical cardiovascular disease at an early stage, allowing timely intervention through lifestyle modification, weight reduction and control of metabolic risk factors. Since visceral obesity is potentially reversible, early detection may significantly reduce long-term cardiovascular morbidity and progression to heart failure.

CONCLUSION

The present study demonstrated a significant association between visceral obesity and left ventricular hypertrophy as well as diastolic dysfunction. Patients with visceral obesity showed significantly higher left ventricular mass index, increased prevalence of LVH and greater impairment of diastolic function compared to those without visceral obesity. Waist circumference and waist–hip ratio showed strong positive correlation with echocardiographic markers of cardiac remodeling. Visceral obesity was identified as an independent predictor of both LVH and diastolic dysfunction even after adjustment for conventional cardiovascular risk factors. The findings of the study emphasize the importance of early identification of visceral obesity and routine cardiovascular assessment using echocardiography to detect subclinical cardiac dysfunction. Early lifestyle modification and management of metabolic risk factors may help prevent progression to overt cardiovascular disease and heart failure.

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