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Role of the Visceral Adiposity Index in Predicting Left Ventricular Diastolic Dysfunction Among Patients with Metabolic Syndrome

Mian Ukasha Mukhtar[1], Sultan Bakht Afroz[1]

1 Department of Medicine, Ghurki Trust Teaching Hospital, Lahore, Pakistan

Corresponding Author: Sultan Bakht Afroz, **Email:** sultanbakhtafroz@gmail.com

ABSTRACT

Background: The biological activity of visceral fat in metabolic syndrome might lead to abnormal relaxation of the ventricles before the onset of the change in the systolic function or the development of signs of heart failure. Visceral Adiposity Index (VAI) is the combination of anthropometry of the abdomen and triglycerides and high-density lipoprotein cholesterol concentration, and may thus better represent metabolically harmful adiposity than body size itself.

Objective: To examine the relationship of the Visceral Adiposity Index with left ventricular diastolic dysfunction and determine its ability to identify affected adults with metabolic syndrome.

Methods: A cross-sectional analysis was performed on 100 adults managed in the Medicine and Cardiology Departments of Ghurki Trust Teaching Hospital, Lahore, from January through December 2025. Clinical information, anthropometric measurements, fasting biochemical findings, and transthoracic echocardiographic indices were recorded. Sex-specific equations were used to derive the Visceral Adiposity Index. Independent associations were assessed through multivariable binary logistic regression, while classification performance was examined using receiver operating characteristic curve analysis.

Results: Diastolic dysfunction was present in 43 participants. Their mean Visceral Adiposity Index was higher than that of participants with normal ventricular filling (4.8 ± 1.2 vs 3.2 ± 0.9; $p<0.001$). The proportion with dysfunction increased from 18.2% in the lowest index tertile to 70.6% in the highest. The index yielded an area under the curve of 0.823. A value greater than 3.72 identified dysfunction with 81.4% sensitivity and 73.7% specificity. After covariate adjustment, each one-unit increase was associated with 2.38 times greater odds of diastolic dysfunction.

Conclusion: Visceral Adiposity Index values showed an independent, graded relationship with impaired left ventricular filling. The index may help prioritise adults with metabolic syndrome for echocardiographic assessment before clinically evident cardiac disease develops.

Keywords: Metabolic syndrome; visceral adiposity index; diastolic dysfunction; echocardiography; visceral obesity.

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INTRODUCTION

Metabolic syndrome reflects the coexistence of several interrelated metabolic disturbances, most commonly abdominal adiposity, elevated blood pressure, abnormal glucose regulation, raised triglycerides, reduced high-density lipoprotein cholesterol, and insulin resistance [1]. Their combined effect extends beyond the development of diabetes and atherosclerotic vascular disease. Even when left ventricular ejection fraction remains preserved, the metabolic environment created by these abnormalities may initiate subtle changes in myocardial structure and performance [2].

Abnormal ventricular filling can precede clinically evident cardiac disease in metabolically vulnerable individuals [3]. At this stage, the myocardium relaxes more slowly, ventricular compliance declines, or intracardiac filling pressure begins to rise without an accompanying

reduction in systolic function. These may occur in combination with hypertension, insulin resistance, endothelial injury, chronic inflammation, oxidative imbalance, and myocardial lipid accumulation. Early diastolic dysfunction often is silent and is only discovered when left atrial enlargement, diminished exercise tolerance, pulmonary congestion, and/or heart failure with preserved ejection fraction are present [4].

Obesity is just a part of cardiometabolic risk. BMI can only identify metabolically active visceral fat; it is a measurement of body size in relation to height [5]. Waist circumference is a marker of abdominal fat accumulation, but it does not reflect any lipid abnormalities that occur. This distinction applies because visceral fat is an endocrine and inflammatory tissue. Visceral fat can lead to increased levels of free fatty acids, cytokines, and dysregulated adipokines, which can contribute to insulin resistance, vascular damage, fibrosis, and adverse cardiac remodelling [6].

The Visceral Adiposity Index is a composite of waist circumference and body mass index (BMI), along with triglyceride and high-density lipoprotein (HDL) cholesterol levels in separate equations for men and women [7]. Thus, the index combines anatomical fat distribution and biochemical evidence for lipid dysfunction. The calculation may be useful as an alternative for predicting visceral fat when it cannot be measured directly, such as by computed tomography or magnetic resonance imaging, as all four components are typically already measured during routine metabolic evaluation [8,9].

Visceral obesity has been associated with decreased ventricular relaxation, but the value of the Visceral Adiposity Index to detect diastolic dysfunction in patients with metabolic syndrome is still unknown [10]. The creation of such an association could aid in the selective screening of those who might benefit from echocardiographic screening before the onset of cardiac symptoms of dysfunction. The present study aimed to explore the association of the Visceral Adiposity Index and left ventricular diastolic dysfunction and to assess its discriminative power among adult metabolic syndrome patients.

MATERIALS AND METHODS

The study was an analytical cross-sectional investigation conducted at Ghurki Trust Teaching Hospital, Lahore, Pakistan, from January 2025 to December 2025 in the departments of Medicine and Cardiology. The adults with metabolic syndrome were selected by consecutive non-probability sampling. The primary study question was whether there was a correlation between higher VAI values and left ventricular diastolic dysfunction.

The sample size was estimated based on a prevalence of expected diastolic dysfunction of 40%, a 95% confidence level, and 10% precision. The first calculation resulted in a minimum of 92 participants. Recruitment was expanded to 100 for biochemical non-findings and/or technically inadequate echocardiographic recordings.

Men and women between the ages of 30 and 70 who had a left ventricular ejection fraction of 50% or higher and satisfied at least three metabolic syndrome criteria were eligible. Waist circumference of at least 90 cm for men and 80 cm for women; fasting plasma glucose of at least 100 mg/dL or treatment for hyperglycemia; triglycerides of at least 150 mg/dL or lipid-lowering treatment for hypertriglyceridaemia; high-density lipoprotein cholesterol of less than 40 mg/dL for men or less than 50 mg/dL for women; and blood pressure of at least 130/85 mmHg or ongoing antihypertensive medication were the diagnostic criteria.

Patients were not enrolled when another disorder could independently influence ventricular filling. Exclusion criteria included ischaemic heart disease, previous myocardial infarction, symptomatic heart failure, cardiomyopathy, atrial fibrillation, congenital cardiac disease, moderate or severe valvular abnormalities, and left ventricular ejection fraction below 50%. Advanced renal impairment, chronic hepatic disease, uncontrolled thyroid illness, systemic inflammatory disorders, acute infection, and pregnancy were also grounds for exclusion. Patients with incomplete biochemical data and suboptimal echocardiographic image quality were excluded from the analysis.

Following enrolment, age, sex, smoking status, diabetes, hypertension, medication use, and family history of cardiovascular disease were recorded. The blood pressure was measured at the end of 5 minutes of sitting rest. Two measurements were taken, 5 minutes apart, using a cuff of the appropriate size for the arm and the average of the two measurements recorded.

Body weight was measured in kg to the nearest decimal to be without shoes and all values were recorded on a calibrated digital scale. Body mass index was calculated as body weight divided by height squared. Measurements for height were taken to the nearest 0.1 cm. After a normal expiration, the waist circumference was measured at the midpoint between the superior border of the iliac crest and the lowest palpable rib.

Following an 8–12-hour overnight fast, blood samples were taken. The institutional laboratory analyzed fasting plasma glucose, triglycerides, total cholesterol, LDL, and HDL using biochemical processes that were automated. The Visceral Adiposity Index was computed after the concentrations of high-density lipoprotein cholesterol and triglycerides were converted to mmol/L.

For male participants:

$$VAI = [WC/ (39.68 + 1.88 \times BMI)] \times (TG/1.03) \times (1.31/HDL\text{-}C)$$

For female participants:

$$VAI = [WC/ (36.58 + 1.89 \times BMI)] \times (TG/0.81) \times (1.52/HDL\text{-}C)$$

Participants were subsequently distributed into lower, middle, and upper thirds according to their calculated index values.

Transthoracic echocardiography was performed in every participant using equipment capable of two-dimensional imaging, pulsed-wave Doppler, and tissue Doppler assessment. An experienced cardiologist who did not know the calculated Visceral Adiposity Index conducted the examinations. Echocardiographic acquisition included conventional parasternal and apical windows while the participant rested in the left lateral position. Left ventricular systolic function was quantified from apical images using the modified Simpson biplane approach.

Diastolic assessment combined transmitral flow and mitral annular tissue velocities. Pulsed-wave Doppler was sampled at the leaflet tips to obtain E and A velocities and calculate the E/A ratio. Septal and lateral e′ velocities were measured with tissue Doppler, and their average was used to derive the mean E/e′ ratio. Left atrial volume was measured from two apical planes and normalised to body surface area.

Diastolic status was determined from the collective echocardiographic pattern rather than a single measurement. Reduced septal or lateral e′ velocity, an increased mean E/e′ ratio, an abnormal E/A pattern, and enlargement of the indexed left atrial volume were considered evidence of impaired filling. Diastolic filling was graded according to the observed Doppler pattern. Grade I represented impaired relaxation without evidence of raised filling pressure, grade II indicated pseudonormal filling with elevated pressure, and grade III denoted a restrictive filling pattern. For the primary analysis, participants were classified into two groups: normal diastolic function and left ventricular diastolic dysfunction.

Ethical clearance was granted by the Ethical Review Committee of Ghurki Trust Teaching Hospital, Lahore, under approval number 2024/12/R-45. Written consent was secured from all participants before enrolment. Study codes were used in place of personal identifiers, access to clinical records was restricted, and the research was conducted in accordance with the principles of the Declaration of Helsinki.

Data processing and analysis were performed in IBM SPSS Statistics version 26.0. Continuous variables were reported as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Data distribution was assessed before selecting statistical tests. Between-group comparisons used the independent-samples t-test for continuous variables and either the chi-square test or Fisher's exact test for categorical variables. Differences in clinical and echocardiographic measurements across Visceral Adiposity Index tertiles were examined using one-way analysis of variance.

Relationships between the Visceral Adiposity Index and echocardiographic measurements were assessed using Pearson or Spearman coefficients, depending on data distribution. Receiver operating characteristic curve analysis quantified the ability of the index to classify diastolic dysfunction. The optimal threshold was selected through the Youden statistic, followed by calculation of sensitivity, specificity, positive predictive value, and negative predictive value.

Binary logistic regression was used to identify variables independently associated with diastolic dysfunction. Covariates producing a univariable p-value below 0.20, together with factors considered clinically relevant, were entered into the adjusted model. Findings were reported as adjusted odds ratios with 95% confidence intervals. Statistical significance was established at $p<0.05$.

RESULTS

Among 108 individuals assessed for participation, eight were excluded because of incomplete laboratory findings, unacceptable echocardiographic image quality, atrial fibrillation, or clinically important valvular disease. The final analysis, therefore, included 100 adults. The cohort had a mean age of 54.8 ± 9.2 years and comprised 56 men (56.0%) and 44 women (44.0%).

Left ventricular diastolic dysfunction was present in 43 participants, producing a prevalence of 43.0%. The remaining 57 participants had filling patterns considered normal for age. Among those with dysfunction, grade I accounted for 27 cases (62.8%), grade II for 13 (30.2%), and grade III for three (7.0%).

The group with diastolic dysfunction showed a less favourable clinical and metabolic profile. These participants were older and had greater body mass index, waist circumference, systolic blood pressure, fasting glucose, triglyceride concentrations, and Visceral Adiposity Index values than those with normal ventricular filling. Hypertension and diabetes were also more frequent in the affected group. In contrast, the distribution of men and women and the mean left ventricular ejection fraction were comparable between the two groups, as detailed in Table 1.

Participants with abnormal ventricular filling had markedly higher Visceral Adiposity Index values than those whose diastolic function remained normal. The echocardiographic profile of the affected group was characterised by slower mitral annular relaxation, reflected by a lower mean e′ velocity, together with a higher E/e′ ratio and greater indexed left atrial volume. Collectively, these findings were consistent with delayed myocardial relaxation and increased left ventricular filling pressure.

A graded echocardiographic pattern was evident across increasing index values. Mean e′ velocity declined as the Visceral Adiposity Index rose, whereas both the E/e′ ratio and left atrial volume index increased. Thus, greater visceral adipose dysfunction was accompanied by progressively less favourable indices of ventricular relaxation and filling, as summarised in Table 2.

The burden of left ventricular diastolic dysfunction differed sharply across the three Visceral Adiposity Index strata. It was identified in 6 of 33 participants (18.2%) in the lowest tertile, 13 of 33 (39.4%) in the middle tertile, and 24 of 34 (70.6%) in the upper tertile. More advanced grades were concentrated predominantly among participants in the highest category, indicating that worsening ventricular filling abnormalities accompanied increasing visceral adiposity.

Continuous-variable analysis produced the same pattern. Higher index values were associated with lower mean early diastolic mitral annular velocity ($r=-0.61$; $p<0.001$), whereas direct associations were observed with mean E/e′ ratio ($r=0.65$; $p<0.001$) and left atrial volume index ($r=0.57$; $p<0.001$). These relationships indicate that increasing visceral adipose dysfunction coincided with progressively slower myocardial relaxation, greater estimated filling pressure, and more pronounced left atrial remodelling.

The Visceral Adiposity Index also demonstrated useful classification performance. Receiver operating characteristic analysis yielded an area under the curve of 0.823 (95% CI: 0.742–0.904; $p<0.001$). At a threshold above 3.72, the index correctly identified 81.4% of participants with diastolic dysfunction and correctly classified 73.7% of those without it. The corresponding positive and negative predictive values were 70.0% and 84.0%, respectively. Its area under the curve exceeded that of waist circumference (0.731) and body mass index (0.692), suggesting better discrimination than either anthropometric measure alone.

In the adjusted logistic model, the Visceral Adiposity Index retained an independent association with diastolic dysfunction after age, sex, diabetes mellitus, and systolic blood pressure were taken into account. Each one-unit increment in the index was associated with a 2.38-fold increase in the odds of the outcome. Advancing age and higher systolic pressure also remained independently related to impaired diastolic function, whereas sex and diabetes mellitus were not significant after adjustment, as reported in Table 3.

The tertile analysis and the adjusted model yielded similar results: the higher the Visceral Adiposity Index value, the worse the ventricular filling, and the Visceral Adiposity Index had the strongest independent association with left ventricular diastolic dysfunction of the variables studied.

Table 1: Clinical and laboratory features based on left ventricular diastolic function

Variable	Normal diastolic function (n=57)	Diastolic dysfunction (n=43)	p-value
Age, years	50.8 ± 8.4	60.1 ± 7.6	<0.001
Male sex	34 (59.6%)	22 (51.2%)	0.397
Female sex	23 (40.4%)	21 (48.8%)	0.397
Body mass index, kg/m²	30.2 ± 3.5	33.0 ± 4.0	<0.001
Waist circumference, cm	98.7 ± 8.8	106.5 ± 9.4	<0.001
Hypertension	29 (50.9%)	33 (76.7%)	0.008
Diabetes mellitus	18 (31.6%)	25 (58.1%)	0.008
Systolic blood pressure, mmHg	133.5 ± 14.6	144.1 ± 15.2	0.001
Fasting plasma glucose, mg/dL	111.8 ± 25.7	135.6 ± 34.1	<0.001
Serum triglycerides, mg/dL	176.4 ± 48.2	235.1 ± 63.0	<0.001
High-density lipoprotein cholesterol, mg/dL	41.5 ± 7.1	34.8 ± 6.5	<0.001
Visceral Adiposity Index	3.2 ± 0.9	4.8 ± 1.2	<0.001
Left ventricular ejection fraction, %	60.8 ± 4.1	59.7 ± 4.5	0.204

Table 2: Echocardiographic findings according to Visceral Adiposity Index tertiles

Variable	Lower tertile (n=33)	Middle tertile (n=33)	Upper tertile (n=34)	p-value
Visceral Adiposity Index	2.4 ± 0.4	3.7 ± 0.3	5.5 ± 0.9	<0.001
Average early diastolic annular velocity, cm/s	7.8 ± 1.0	6.8 ± 1.1	5.7 ± 1.0	<0.001
Average filling-pressure ratio	8.9 ± 1.8	11.1 ± 2.1	14.0 ± 2.8	<0.001
Left atrial volume index, mL/m²	27.5 ± 4.5	31.4 ± 5.1	36.1 ± 6.0	<0.001
Left ventricular ejection fraction, %	61.0 ± 4.0	60.2 ± 4.2	59.5 ± 4.6	0.345
Diastolic dysfunction	6 (18.2%)	13 (39.4%)	24 (70.6%)	<0.001

Table 3: Multivariable logistic regression analysis of factors linked to left ventricular diastolic dysfunction

Predictor	Adjusted odds ratio	95% confidence interval	p-value
Visceral Adiposity Index, per one-unit increase	2.38	1.59–3.56	<0.001
Age, per one-year increase	1.07	1.02–1.13	0.006
Female sex	1.15	0.47–2.81	0.759
Diabetes mellitus	1.82	0.76–4.38	0.178
Systolic blood pressure, per 10-mmHg increase	1.29	1.01–1.66	0.043

DISCUSSION

Almost half of the cohort of adults assessed in this study had echocardiographic data that showed they had left ventricular diastolic dysfunction, even though they had a normal ejection fraction [1]. This suggests that metabolic syndrome could be associated with myocardial performance before the onset of the traditional features of myocardial failure [2]. The VAI was significantly

increased in participants with abnormal filling, and the prevalence of such affected participants increased from 18.2% in the lowest tertile to 70.6% in the highest. The increased proportion of more advanced dysfunction in the upper tertile also supports the notion that this association was a graded one, not just a high versus low association [3].

The index was more sensitive in discriminating diastolic dysfunction than BMI or WC. This benefit can be due to the distinct information that these measures have been able to capture [4,5]. Body mass index reflects overall body size, and waist circumference is a gauge of belly fat. Both are insensitive to the associated lipid disturbance. The Visceral Adiposity Index may be a better marker of the metabolic activity of abdominal fat tissue by combining anthropometric parameters with triglyceride and HDL cholesterol levels [6].

This interpretation is functionally supported by the echo changes between tertiles. Index values were inversely correlated to mean e′ velocity in participants, reflecting that the higher the index, the slower the myocardial relaxation velocity in the longitudinal direction [7]. Simultaneously, there were increases in the E/e′ ratio and indexed left atrial volume, reflecting enhanced filling pressure and continuous exposure to pressure in the left atrium. There was no significant decrease in left ventricular ejection fraction between the groups, implying that cardiac involvement at this stage was mostly diastolic [8].

These abnormalities can be explained via several interconnected pathways that underlie visceral adipose dysfunction [9]. Visceral fat that has expanded produces excess FFA and unbalances the inflammatory cytokines and adipokines. These perturbations can exacerbate insulin resistance, dysfunction of endothelial signaling, and elevate oxidative stress. Exposure can lead to changes in coronary microvascular function, myocardial energy utilisation, calcium cycling, and extracellular matrix turnover. This leads to fibrosis and reduced compliance of the ventricles, which can lead to decreased relaxation without an immediate decrease in ejection [10].

As expected, in the population, metabolic syndrome, hypertension, and diabetes were more prevalent in those with diastolic dysfunction [11]. Both disorders can lead to myocardial hypertrophy, microvascular injury, accumulation of collagen, and stiffening of the ventricles. However, this was present even when diabetes, age, sex, and systolic blood pressure were adjusted for. This persistence suggests that its relation with ventricular filling was not accounted for simply by these known clinical risk factors [12].

There was also an independent relationship of outcome with age and with systolic pressure. Myocardial changes with ageing include decreased myocardial elasticity, altered ventricular–arterial coupling, and relaxation changes [13]. An increase in afterload with higher systolic pressure can lead to accelerated structural remodelling. Despite the adjustment for these effects, there was still a 2.38-fold increase in the odds for the occurrence of diastolic dysfunction with each one-unit increase in the Visceral Adiposity Index. The size of this estimate sustains the possibility of the index being an added cardiometabolic risk marker [14].

The receiver operating characteristic results also confirmed that the index was able to distinguish patients with abnormal filling from patients with normal diastolic function [15]. The area above 3.72 gave a sensitivity of 81.4% and a specificity of 73.7%. A negative predictive value of 84.0% indicates that values below this level may be useful to rule out the presence of diastolic dysfunction. It also had a larger area under the curve compared to BMI and WC. This threshold was, however, found in a cohort from a single centre and should be considered exploratory until validated in an independent population [16].

The index might be clinically useful since its components are typically available as part of a routine metabolic assessment [17]. No additional imaging, no specialised assay or costly equipment required for calculation. It could potentially be used to select patients who would warrant earlier echocardiographic evaluation and not be used to apply cardiac imaging to all individuals with metabolic syndrome. However, determination of subclinical dysfunction may also offer further incentive for the intensive management of abdominal obesity, lipid abnormalities, blood pressure, glycaemic status, and physical inactivity [18].

There are some caveats to interpretation. The cross-sectional study design does not help determine if higher VAI values were associated with the onset of diastolic dysfunction [19]. The sample size was relatively small and was selected from one institution, and may not apply to other populations. Visceral fat was not directly measured using computed tomography or magnetic resonance imaging. Data for physical activity, diet, length of time with metabolic syndrome, adherence to treatment, and a few other potential confounding variables were not fully available. Future multicentre studies with serial echocardiographic follow-up are required to establish if the index is predictive of deteriorating ventricular function, subsequent heart failure, or other cardiovascular events [20].

CONCLUSION

In adult metabolic syndrome patients with normal LVEF, higher VAI was correlated with worsening LVR and estimated filling pressures, left atrial enlargement, and more advanced diastolic dysfunction. This association was independent of major clinical factors, and the index discriminated the outcome better than did body mass index or waist circumference. It could thus be used to select metabolically vulnerable patients for echocardiographic evaluation, but the suggested cut-off point needs external

and prospective validation before being used routinely in clinical practice.

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Authors' Contributions: M.U.M. designed the study, collected data, and drafted the manuscript. S.B.A. supervised the study. Both authors approved the final version.

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Data Availability: Data are available from the corresponding author upon reasonable request.

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