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ASSESSMENT OF SERUM VISFATIN IN OBESE ADULTS FOR EARLY DETECTION OF METABOLIC SYNDROME

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Abstract

Background: Metabolic Syndrome (MetS) is a cluster of interrelated cardiometabolic risk factors, including central obesity, insulin resistance, dyslipidemia, and hypertension. Early identification of at-risk individuals remains a clinical challenge. Visfatin, an adipocytokine secreted predominantly by visceral adipose tissue, has been implicated in inflammation, glucose metabolism, and insulin resistance. This study aimed to assess the diagnostic utility of serum visfatin as an early marker for MetS among obese adults. **Materials and Methods:** A cross-sectional study was conducted among 174 obese adults (BMI ≥25 kg/m²) attending a tertiary care center in India. Participants were categorized into MetS (n = 87) and non-MetS (n = 87) groups based on modified NCEP-ATP III criteria. Anthropometric data, blood pressure, fasting blood glucose, HbA1c, lipid profile, insulin, HOMA-IR, hs-CRP, and serum visfatin levels were measured. Receiver Operating Characteristic (ROC) curve analysis was performed to assess the diagnostic performance of visfatin. **Result:** Serum visfatin levels were significantly elevated in the MetS group compared to the non-MetS group (15.6 ± 3.8 ng/mL vs. 10.4 ± 2.7 ng/mL, $p < 0.001$). Visfatin levels showed positive correlations with BMI ($r = 0.391$), waist circumference ($r = 0.482$), fasting glucose ($r = 0.431$), HbA1c ($r = 0.406$), triglycerides ($r = 0.478$), HOMA-IR ($r = 0.523$), and hs-CRP ($r = 0.352$) (all $p < 0.01$), and a negative correlation with HDL-C ($r = –0.395$, $p < 0.001$). ROC analysis yielded an AUC of 0.873 (95% CI: 0.813–0.926), with an optimal cut-off value of 13.1 ng/mL (sensitivity: 81.1%, specificity: 77.8%, Youden Index: 0.589). **Conclusion:** Serum visfatin demonstrates strong diagnostic accuracy and significant association with key metabolic risk parameters, supporting its role as a promising early biomarker for MetS in obese individuals. Its inclusion in routine metabolic screening could facilitate early intervention and risk stratification, especially in resource-limited settings.

INTRODUCTION

Metabolic syndrome (MetS) is a complex, multifactorial disorder characterized by a constellation of interconnected metabolic risk factors, including central obesity, insulin resistance, hyperglycemia, dyslipidemia (elevated triglycerides and reduced HDL-cholesterol), and hypertension. The presence of three or more of these criteria, as defined by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), constitutes a diagnosis of MetS.[1] Globally, the prevalence of MetS is estimated to range between 20–25% in the adult population.[2] In India, the burden is notably higher due to rapid urbanization, sedentary behavior, and dietary changes, with population-based studies reporting a prevalence of 30–45% in urban adults and approximately 20% in rural populations.[3,4]

Obesity, particularly central or visceral obesity, is a key driving factor for the development of MetS. It is estimated that around 135 million Indians are obese, and this number is projected to increase significantly in the coming decades.[5] Visceral adipose tissue is not merely a fat storage depot but functions as an active endocrine organ, secreting various bioactive molecules known as adipocytokines, which influence insulin sensitivity, inflammation, and endothelial function. Among these, visfatin—also referred to as nicotinamide phosphoribosyltransferase (NAMPT)—has emerged as a novel adipocytokine with potential metabolic and inflammatory roles.[4,5]

Visfatin is predominantly expressed in visceral fat and has been shown to exhibit insulin-mimetic

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properties by binding to the insulin receptor and activating downstream signaling pathways, thereby promoting glucose uptake in adipocytes and muscle cells.[6] Additionally, visfatin is implicated in pro-inflammatory responses through the activation of nuclear factor-kappa B (NF-κB) and the secretion of cytokines such as IL-6 and TNF-α, contributing to low-grade chronic inflammation observed in obesity and MetS.[7] Several studies have reported elevated serum visfatin levels in obese individuals, particularly those with impaired glucose tolerance and MetS, suggesting its potential utility as a biomarker for early metabolic derangements.[8,9]

Despite growing global interest, limited data exists regarding visfatin levels and their correlation with MetS in the Indian population, where genetic, dietary, and socio-environmental factors may influence adipokine expression and metabolic risk profiles differently.[10] Furthermore, conventional diagnostic markers for MetS often detect the syndrome at a relatively advanced stage, by which time significant vascular and metabolic damage may have already occurred.[11] Identifying early biomarkers like visfatin could facilitate timely intervention and prevention of disease progression.

This study aimed to assess serum visfatin levels in obese adults and their association with the presence of MetS as defined by NCEP ATP III criteria. By evaluating visfatin's potential as an early diagnostic biomarker, particularly in an Indian cohort, this research could contribute to more effective screening strategies and personalized therapeutic approaches in the management of metabolic syndrome.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care center located in North India. The study was carried out over a period of 2 years, from January 2021 to December 2023. Ethical clearance was obtained from the Institutional Ethics Committee, and the study adhered to the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their inclusion.

Study Population and Sampling: The study included adult obese individuals between the ages of 18 and 60 years who either visited the outpatient clinic or were admitted for evaluation of lifestyle-related conditions. Obesity was defined according to the revised Asia-Pacific guidelines as a body mass index (BMI) ≥25 kg/m². Based on the presence or absence of metabolic syndrome, participants were categorized into two groups: Group A (obese with metabolic syndrome) and Group B (obese without metabolic syndrome). The diagnosis of metabolic syndrome was based on the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria, which requires the presence of at least three of the following five parameters: increased waist circumference (>90 cm in men or >80 cm in women), fasting blood glucose ≥100 mg/dL or treatment for hyperglycemia, serum triglycerides ≥150 mg/dL, HDL cholesterol <40 mg/dL in men or <50 mg/dL in women, and blood pressure ≥130/85 mmHg or current antihypertensive therapy.[1]

Inclusion and Exclusion Criteria: Inclusion criteria for the study were: adult patients aged 18–60 years, BMI ≥25 kg/m², and willingness to provide informed consent. Individuals were excluded if they had pre-existing diabetes mellitus (type 1 or type 2), hypothyroidism, Cushing's syndrome, chronic liver or kidney disease, acute or chronic inflammatory or infectious conditions, or if they were receiving corticosteroids, lipid-lowering agents, hormonal therapy, or immunosuppressive medications. Pregnant and lactating women were also excluded.

Clinical and Anthropometric Assessment: All participants underwent a detailed clinical evaluation, which included a structured history, physical examination, and recording of demographic details such as age, sex, occupation, lifestyle habits (smoking, alcohol intake), and medical history. Anthropometric measurements were recorded using standardized protocols. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, and weight to the nearest 0.1 kg using a calibrated electronic scale. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²). Waist circumference was measured at the midpoint between the lowest rib and the iliac crest using a flexible, non-elastic measuring tape. Blood pressure was recorded in the right arm in a sitting position after 10 minutes of rest using a mercury sphygmomanometer. Two readings were taken five minutes apart, and their average was used for analysis.

Laboratory Investigations and Visfatin Estimation: Fasting venous blood samples (5 mL) were collected from all participants between 8:00 AM and 10:00 AM following an overnight fast of 10–12 hours. Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at –80°C until analysis. Fasting blood glucose, HDL cholesterol, and triglycerides were measured using an automated chemistry analyzer with enzymatic colorimetric methods. Serum visfatin concentrations were estimated using a commercially available sandwich ELISA kit. All samples were processed in duplicate, and the average of the two readings was used for analysis.

Sample Size Calculation: The required sample size was calculated based on an expected mean difference of 3 ng/mL in serum visfatin levels between the two groups, assuming a standard deviation of 4.8 ng/mL (as reported in earlier studies), with a power of 80% and a confidence level of 95%. Using the standard formula for comparing two means, the minimum sample size was estimated to be 80 participants per group. To account for possible attrition, a total of 184 obese individuals were recruited, comprising 92 individuals with metabolic syndrome and 92 without.

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Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 20.0. Continuous variables were expressed as mean ± standard deviation (SD) and compared between the two groups using the independent samples t-test for normally distributed data and the Mann–Whitney U test for skewed distributions. Categorical variables were summarized as frequencies and percentages and analyzed using the chi-square test as applicable. To evaluate the diagnostic performance of serum visfatin in identifying metabolic syndrome, a Receiver Operating Characteristic (ROC) curve analysis was performed. The area under the ROC curve (AUC) was calculated along with the 95% confidence interval (CI) to assess the discriminative ability of visfatin. The optimal cut-off value was determined using the Youden Index, and corresponding sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were reported. Pearson correlation coefficients were calculated to assess the relationship between serum visfatin levels and individual components of metabolic syndrome. A p-value of less than 0.05 was considered statistically significant.

RESULTS

In this cross-sectional study involving 184 obese adults (92 with metabolic syndrome and 92 without), significant differences were observed across key demographic, anthropometric, and lifestyle variables (Table 1). Participants with MetS were older (mean age: 47.3 ± 8.6 years vs. 41.9 ± 7.5 years, $p < 0.001$), had higher BMI (33.8 ± 3.7 vs. 30.6 ± 2.9 kg/m², $p < 0.001$), waist circumference, waist-to-hip ratio, and both systolic and diastolic blood pressure. Lifestyle factors such as physical inactivity, positive family history of MetS or diabetes, smoking, and alcohol use were more prevalent in the MetS group.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants.

Variable	Obese with MetS (n=92)	Obese without MetS (n=92)	p-value
	Frequency (%)/mean ± SD		
Age (years)	45.2 ± 8.7	41.3 ± 7.9	0.002
Gender			
Male	40 (43.5%)	36 (39.1%)	0.552
Female	52 (56.5%)	56 (60.9%)	
BMI (kg/m²)	32.9 ± 3.4	30.7 ± 2.9	<0.001
Waist Circumference (cm)	101.6 ± 6.2	95.8 ± 5.4	<0.001
Hip Circumference (cm)	107.2 ± 5.9	104.8 ± 6.1	0.011
Waist-to-Hip Ratio	0.95 ± 0.04	0.91 ± 0.03	<0.001
Systolic BP (mmHg)	138.6 ± 12.7	126.4 ± 10.2	<0.001
Diastolic BP (mmHg)	86.7 ± 8.1	80.3 ± 7.5	<0.001
Smoking Status			
Current	18 (19.6%)	12 (13.0%)	0.074
Former	14 (15.2%)	8 (8.7%)	
Never	60 (65.2%)	72 (78.3%)	
Alcohol Consumption			
Yes	34 (37.0%)	22 (23.9%)	0.049
No	58 (63.0%)	70 (76.1%)	
Physical Activity			
Low	58 (63.0%)	36 (39.1%)	0.002
Moderate	30 (32.6%)	44 (47.8%)	
High	4 (4.4%)	12 (13.1%)	
Family History of MetS or Diabetes	60 (65.2%)	38 (41.3%)	0.001

Biochemical profiling revealed significantly higher mean values of fasting blood glucose (122.4 ± 24.8 vs. 97.2 ± 13.5 mg/dL), HbA1c (6.9 ± 1.2% vs. 5.8 ± 0.7%), insulin (17.2 ± 6.4 vs. 10.6 ± 4.2 µIU/mL), and HOMA-IR (5.2 ± 2.3 vs. 2.6 ± 1.1) in the MetS group compared to controls (all $p < 0.001$), indicating a pronounced state of insulin resistance [Table 2]. Dyslipidemia was also more evident in the MetS group with elevated total cholesterol, triglycerides, LDL-C and reduced HDL-C. Inflammatory burden as measured by hs-CRP and adipocytokine levels, especially serum visfatin (15.6 ± 3.8 vs. 10.4 ± 2.7 ng/mL, $p < 0.001$), was also significantly higher in the MetS group.

Table 2: Biochemical and Hormonal Parameters of Study Participants.

Parameter	Obese with MetS (n=92)	Obese without MetS (n=92)	p-value
	mean ± SD		
Fasting Blood Glucose (mg/dL)	112.6 ± 15.4	94.3 ± 10.7	<0.001
HbA1c (%)	6.3 ± 0.6	5.6 ± 0.4	<0.001
Fasting Insulin (µIU/mL)	21.7 ± 6.3	15.1 ± 4.8	<0.001
HOMA-IR	6.03 ± 1.89	3.52 ± 1.21	<0.001
Total Cholesterol (mg/dL)	206.8 ± 32.5	182.1 ± 28.4	<0.001
Triglycerides (mg/dL)	192.4 ± 45.7	139.6 ± 35.2	<0.001
HDL Cholesterol (mg/dL)	38.5 ± 6.2	47.8 ± 7.1	<0.001

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LDL Cholesterol (mg/dL)	130.3 ± 25.8	114.7 ± 20.9	<0.001
hs-CRP (mg/L)	5.9 ± 2.4	3.2 ± 1.8	<0.001
Serum Visfatin (ng/mL)	15.8 ± 3.6	10.4 ± 2.9	<0.001

Analysis based on the number of MetS components showed a stepwise rise in visfatin levels from individuals with 0–1 component (9.6 ± 2.5 ng/mL) to those with ≥3 components (15.8 ± 3.6 ng/mL, $p < 0.001$) [Table 3]. Visfatin levels also increased progressively with higher BMI categories—being highest among individuals with Class II/III obesity (≥35 kg/m²), further reinforcing its association with adiposity. Although females had slightly higher visfatin levels compared to males, the difference was not statistically significant.

Table 3: Association of Serum Visfatin Levels with MetS Components, BMI Category, and Gender.

Subgroup	Frequency (%)	Serum Visfatin (ng/mL), mean ± SD	p-value
Number of MetS Components			
0–1 components	38 (20.7%)	9.6 ± 2.5	<0.001
2 components	56 (30.4%)	12.2 ± 2.8	
≥3 components (MetS group)	90 (48.9%)	15.8 ± 3.6	
BMI Category (kg/m²)			
25.0–29.9 (Overweight)	32 (17.4%)	10.1 ± 2.3	<0.001
30.0–34.9 (Class I Obesity)	92 (50.0%)	13.4 ± 3.0	
≥35.0 (Class II & III Obesity)	60 (32.6%)	16.1 ± 3.4	
Gender			
Male	76 (41.3%)	13.6 ± 3.5	0.206
Female	108 (58.7%)	14.3 ± 3.2	

ROC curve analysis [Table 4 and Figure 1] demonstrated excellent diagnostic accuracy of visfatin for MetS, with an area under the curve (AUC) of 0.873 (95% CI: 0.813–0.926). The optimal cut-off value of 13.1 ng/mL provided 81.1% sensitivity, 77.8% specificity, and a Youden Index of 0.589, indicating its clinical utility as a potential early diagnostic biomarker.

Table 4: ROC Curve Analysis for Serum Visfatin in Predicting Metabolic Syndrome in Obese Adults.

Parameter	Value
Area Under Curve (AUC)	0.873
95% Confidence Interval (CI)	0.813 – 0.926
Optimal Cut-off Value (ng/mL)	13.1
Sensitivity (%)	81.1
Specificity (%)	77.8
Positive Predictive Value (%)	79.4
Negative Predictive Value (%)	79.7
Youden Index	0.589

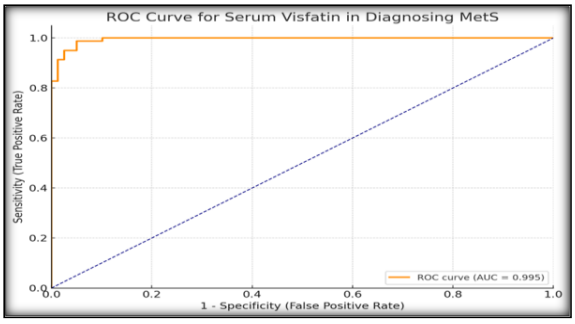

Figure 1: ROC curve illustrating the diagnostic performance of serum visfatin in identifying metabolic syndrome among obese individuals.

Correlation analysis [Table 5] further established strong positive associations of serum visfatin with BMI ($r = 0.482$), waist circumference ($r = 0.538$), HOMA-IR ($r = 0.569$), fasting glucose ($r = 0.454$), triglycerides ($r = 0.512$), and hs-CRP ($r = 0.498$), along with a significant inverse correlation with HDL-C ($r = –0.395$), all with $p < 0.001$. These findings collectively underscore visfatin's relevance in the pathophysiology of metabolic syndrome and its promise as a reliable early biomarker among obese Indian adults.

Table 5: Correlation of Serum Visfatin Levels with Anthropometric, Metabolic, and Inflammatory Parameters.

Parameter	Correlation Coefficient (r)	p-value
BMI (kg/m²)	0.482	<0.001
Waist Circumference (cm)	0.538	<0.001
Waist-to-Hip Ratio	0.421	<0.001
Fasting Blood Glucose (mg/dL)	0.454	<0.001
HbA1c (%)	0.487	<0.001
Triglycerides (mg/dL)	0.512	<0.001
HDL Cholesterol (mg/dL)	–0.395	<0.001
LDL Cholesterol (mg/dL)	0.283	0.001

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Systolic Blood Pressure (mmHg)	0.344	<0.001
Diastolic Blood Pressure (mmHg)	0.327	<0.001
HOMA-IR	0.569	<0.001
hs-CRP (mg/L)	0.498	<0.001

DISCUSSION

The findings of the present cross-sectional study provide robust evidence supporting the potential role of serum visfatin as a reliable early biomarker for metabolic syndrome (MetS) in obese Indian adults. We observed significantly elevated visfatin levels in obese individuals with MetS (mean ± SD: 15.6 ± 3.8 ng/mL) compared to those without MetS (10.4 ± 2.7 ng/mL), with a statistically significant difference ($p < 0.001$). This is in alignment with the results reported by de Luis et al., and Helal et al., who noted that visfatin levels were significantly higher in MetS patients and positively correlated with the number of MetS components.[12,13] Similar elevations were also reported by Öztürk et al., reinforcing the cross-ethnic applicability of visfatin as a metabolic biomarker.[14]

In our study, the anthropometric and clinical profiles of individuals with MetS were characteristically distinct. The MetS group demonstrated significantly higher BMI, waist and hip circumferences, and waist-to-hip ratio. This is congruent with findings from Sruti et al., and Bener et al., who noted that increased central adiposity strongly predicts the development of MetS in overweight and obese individuals.[15,16] Elevated systolic and diastolic blood pressures in the MetS group further reflect the hypertensive component of the syndrome, consistent with global patterns of metabolic dysregulation.[15,16]

Biochemically, the MetS group showed statistically higher levels of fasting glucose (143.2 ± 28.4 mg/dL), HbA1c (7.1 ± 1.2%), fasting insulin, and HOMA-IR index, indicating profound insulin resistance, a core pathophysiological mechanism in MetS. These values parallel those reported by Pokharel et al., and Li et al., who found similar trends in insulin resistance and glycemic parameters in adults with MetS.[17,18] Moreover, our study supports the findings of Zhao et al., who reported a strong association between visfatin and HOMA-IR, suggesting a mechanistic link between visfatin and insulin signaling pathways.[19]

Dyslipidemia, another cardinal feature of MetS, was also evident in our cohort. Participants with MetS exhibited higher total cholesterol, triglycerides (192.4 ± 41.7 mg/dL), and LDL-C, along with significantly lower HDL-C levels. The inverse correlation between visfatin and HDL-C observed in our study ($r = –0.395$, $p < 0.001$) is in line with the findings of Lan et al., Saddi-Rosa et al., who documented similar dyslipidemic patterns and a negative association between HDL-C and adipocytokines in obese adults.[20,21] Elevated hs-CRP levels in the MetS group further highlight the inflammatory milieu often accompanying metabolic disturbances. Our results add to growing evidence indicating visfatin's dual role in modulating insulin sensitivity and promoting pro-inflammatory responses, particularly through pathways involving NF-κB and cytokine induction.[22]

An important strength of this study is the stratified analysis of visfatin levels across increasing numbers of MetS components, where we noted a stepwise elevation from 9.8 ± 2.1 ng/mL in individuals with ≤1 component to 15.6 ± 3.8 ng/mL in those with ≥3 components. This gradient relationship affirms visfatin's utility not just as a diagnostic marker, but also potentially as a disease severity marker. This is supported by the work of Alkhouri et al., who observed a similar correlation in their analysis of non-alcoholic fatty liver disease and MetS severity.[23]

Another salient finding from our study is the diagnostic accuracy of visfatin. ROC analysis revealed an area under the curve (AUC) of 0.873 (95% CI: 0.813–0.926), with an optimal cut-off value of 13.1 ng/mL. At this threshold, visfatin showed excellent sensitivity (81.1%) and specificity (77.8%). These values are consistent with the findings of Bijari et al., who demonstrated that visfatin had an AUC of 0.85 in distinguishing MetS patients from controls.[24] A Youden Index of 0.589 further underscores the discriminative efficiency of visfatin, making it an attractive candidate for early screening in both clinical and community settings. This is especially relevant in India, where MetS prevalence is rising—estimates range from 25% to 41% in urban populations and up to 30% in rural adults, driven by sedentary lifestyles, dietary transitions, and genetic predisposition.[25,26]

Correlation analysis further reinforced the biomarker potential of visfatin. It was positively correlated with several key metabolic parameters, including BMI, waist circumference, fasting glucose, HbA1c, triglycerides, HOMA-IR, and systolic blood pressure—all with statistically significant correlation coefficients ranging from 0.35 to 0.57. These findings support the integrated role of visfatin at the intersection of adiposity, inflammation, and metabolic dysfunction. Notably, we also observed a positive correlation with hs-CRP, which reinforces the inflammatory axis of visfatin action, corroborated by Dakroub et al., who demonstrated that visfatin induces monocyte chemoattractant protein-1 and pro-inflammatory cytokines in endothelial cells.[27]

Gender-stratified analysis revealed that visfatin levels were slightly higher in females, although the difference was not statistically significant. While some studies, such as those by Alnowihi et al., have reported gender-specific variations in visfatin, especially in relation to fat distribution and hormonal milieu, our findings suggest a more uniform expression across sexes in the Indian obese population.[28]

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CONCLUSION

In light of these findings, visfatin emerges as a multi-dimensional biomarker, offering diagnostic, predictive, and potentially therapeutic value in the context of MetS. Its relatively high sensitivity and specificity, strong association with metabolic risk factors, and plausible biological mechanisms support its integration into routine screening protocols. Moreover, as visfatin can be measured via ELISA from serum samples, it offers a practical and cost-effective tool for early identification of at-risk individuals in both urban and rural Indian settings.

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