

Serum Visfatin as a Predictive Biomarker of Metabolic Dysregulation in Obesity: Correlation with Lipid Parameters and Insulin Resistance

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ABSTRACT

Background and Objective

Visfatin is a new adipocytokine primarily released by adipose tissue. It showed to affect both lipid and glucose metabolism and contribute to the chronic inflammatory condition associated with obesity. This study aimed to determine visfatin levels in obese and non-obese adult males and investigate their relationship with selected metabolic markers including insulin resistance and lipid profile.

Materials and Methods

Blood specimens were taken from 90 adult males categorized into two groups: obese and non-obese. Biochemical parameters were measured, including visfatin concentrations by using an enzyme-linked immunosorbent assay (ELISA) with a human visfatin ELISA kit, FBG, lipid profile, and hs-CRP using the Cobas 6000 c501 system, and FI using the Cobas 6000 c601 system.

Results

Serum visfatin levels were significantly elevated in obese subjects. FI, FBG, lipid profile, hs-CRP, HOMA-IR, and DBP are also significantly elevated in obese group. Visfatin showed a significant positive correlation with BMI, WC, FI, FBG, lipid profile, hs-CRP, HOMA-IR, and DBP, but a significant negative association with HDL-C.

Conclusion

The findings suggest that serum visfatin levels are markedly increased in obesity and are strongly linked with both insulin resistance and dyslipidemia. These results support the potential of visfatin as a predictive biomarker of metabolic dysregulation in obese individuals.

Keywords

Obesity; Adipokines; Visfatin; Lipid profile; Insulin Resistance.

INTRODUCTION

Excessive fat deposition in adipose tissue is the defining feature of the chronic condition known as obesity. This accumulation has a detrimental impact on the majority of an organism's systems, increasing the risk of cardiovascular disease (CVD), cancer, hypertension, stroke, non-alcoholic fatty liver, type II diabetes, and mental health issues¹⁻³. According to WHO, body mass index (BMI) is the standard method for classifying overweight and obesity in adults, which is calculated as weight (kg)/height (m²). Individuals are considered overweight and obese if their BMI is ≥ 25 kg/m² and ≥ 30 kg/m², respectively. According to WHO data published in 2022, 2.5 billion adults in the world are overweight, more than 890 million adults diagnosed with obesity, and obesity is recognized as a new non-infectious epidemic of the 21st century².

The term "adipose tissue" refers to a dynamic organ that is dispersed throughout the body, possessing an almost limitless ability to enlarge in the state of obesity⁴. While adipose tissue was once primarily recognized for its role in storing energy as triglycerides, it is now understood to also act as a vital endocrine organ. This is due to the secretion of hormone-like substances by adipose tissues, referred to as "adipokines" or "adipocytokines." For example, resistin,

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leptin, adiponectin, and visfatin contribute to various physiological processes⁵.

Cell-signaling proteins known as adipokines are essential regulators of numerous physiological functions, influencing mechanisms like lipid and glucose metabolism, energy and appetite regulation, insulin sensitivity, inflammation, angiogenesis, endothelial cell function, hemostasis, atherosclerosis progression, blood pressure, and metabolic syndrome⁶⁻⁸. In certain metabolic disorders, particularly obesity, the levels of these adipokines may be disrupted or irregular^{5,9}.

Visfatin is a protein initially identified as pre-Bcell colony-enhancing factor (PBEF) and showing nicotinamide phosphoribosyl transferase activity, composed of 491 amino acids, it has an approximate molecular mass of 52 kDa¹⁰ and is synthesized mainly by visceral adipose tissue^{11,12}. Visfatin has insulin mimic properties¹³. And also, it has a pro-inflammatory-atherosclerotic effect, indicating its involvement in the chronic inflammation commonly linked to obesity¹⁴. It regulates lipid homeostasis and is essential for glucose metabolism¹⁵. Several studies have demonstrated a significant positive relationship between obesity and serum visfatin levels^{16,17}. On the other hand, Pagano et al¹⁸ revealed that plasma visfatin levels were significantly lower in obese subjects.

Despite increasing interest, the clinical relevance of visfatin as a biomarker for metabolic dysfunction in obesity remains under investigation. In particular, its relationship with lipid parameters and insulin resistance indices, such as the HOMA-IR, needs further clarification.

Therefore, this study aimed to assess serum visfatin levels in obese and non-obese adult males and to investigate its correlation with lipid profile components and insulin resistance markers, with the goal of assessing visfatin's potential role as a predictive biomarker of metabolic dysregulation in obesity.

METHODOLOGY

Design of the study:

Ninety individuals, aged 18 to 44 years, participated in this comparative cross-sectional observational study conducted at Zakho General Hospital in Zakho City, Kurdistan Region, Iraq, from October 2024 to January

2025. Before their participation, all individuals were asked to provide written informed consent. Information regarding medical histories, health risk behaviors, including smoking habits, physical activity, and alcohol consumption, as well as key demographic information such as age and geographic region, was collected through direct interviews. A specifically designed questionnaire was used to gather these details from the participants.

In this study we created two main groups according to their BMI. The first group included 50 obese adult men with a BMI of more ≥ 30 kg/m², the second group included 40 healthy non-obese adult men with a BMI between 18 and 25 kg/m² as a control group. Participants having any additional disease, such as Diabetes Mellitus, cardiovascular disease, thyroid disorders, or those who used alcohol, tobacco, or their products, as well as those taking any medication, were not enrolled in the research.

Anthropometric measurements for all participants were recorded, which included waist circumference (WC) (cm), height (cm), and weight (kg). According to this law, weight (kg) divided by height (m²); and BMI were calculated, with data derived from self-reported and measured values. Participants' BMI was classified based on the WHO criteria: normal weight between 18.5-24.99 kg/m² while obese equal or more than 30 kg/m². Furthermore, for all participants, diastolic blood pressure (DBP) and systolic blood pressure (SBP) also recorded using a digital blood pressure monitor.

Following an overnight fast, six milliliters of venous blood were drawn from participants at Zakho General Hospital between 8:30 a.m. and 12:00 p.m. and then transferred to a sterile serum separation tube (gel tube). We kept it at room temperature until the clot forms, which should take around ten minutes. The samples were centrifuged (universal 320, Hettich) at 3000 rpm for 10 minutes at room temperature after clot formation to extract the serum. The extracted serum was taken out and put it into 1.5 ml Eppendorf tubes, that are used to divide the samples before being stored at -75° in a deep freezer at the Zakho Blood Bank until the analysis.

2.2 Biochemical analysis

The high-sensitivity C-reactive protein (hs-CRP), Fasting Blood Glucose (FBG), and lipid profile,

which includes High-Density Lipoprotein Cholesterol (HDL-C), Triglycerides (TG), and Total Cholesterol (TC), were analyzed by using the Cobas 6000 c501 autoanalyzer system (Roche Diagnostics, HITACHI). While fasting insulin (FI) and visfatin were measured by using the Cobas 6000 c601 autoanalyzer system (Roche Diagnostics, HITACHI) and the Enzyme-Linked Immunosorbent Assay (ELISA) with a human visfatin ELISA kit (Sunlong Biotech Co., Ltd, Catalog Number: SL1825Hu), respectively. Some parameters, which include Homeostasis Model Assessment of Beta-cell Function (HOMA- β), very low-density lipoprotein cholesterol (VLDL-C), Triglyceride-Glucose Index (TyG Index), low-density lipoprotein cholesterol (LDL-C), and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), were estimated by using these equations $HOMA-\beta = 20 \times \text{Fasting Insulin } (\mu\text{U/mL}) / \text{Fasting Glucose } (\text{mmol/L}) - 3.5$, $VLDL-C = TG/5$, $TyG \text{ Index} = \ln(\text{Triglycerides } (\text{mg/dL}) \times \text{Fasting Glucose } (\text{mg/dL}) / 2)$, $LDL-C = TC - HDL-C - TG/5$, and $(HOMA-IR) = FBS (\text{mg/dL}) \times FI (\text{mU/ml}) / 405^{19,20}$.

Analysis of Data

SPSS version 26.0 was used to analyze the data of this research. In order to assess the normality distribution of the study variables, Shapiro-Wilk test is used and are expressed either as mean \pm standard deviation (SD) or median and interquartile range for data that followed a normal distribution, and non-normal distributed respectively. Furthermore, the differences of biochemical parameters between the two groups, was achieved using the independent t-test for normal distributed of data and Mann-Whitney U test for data that did not follow the normal one. The relationship between visfatin and other variables was examined using Spearman's correlation test. The Receiver Operating Characteristic, ROC curve was conducted to determine the optimal serum visfatin cut-off point using the Youden formula²¹. P-value less than 0.05 is considered a statistically significant.

Ethical approval

Ethical approval for this study was obtained in November 2024 from the Research Ethics Committee, College of Medicine, University of Zakho (Ref: NOV2024/UOZE28).

RESULTS

The current research included 90 participants aged between 18 and 44 years, with 50 participants being obese and 40 being non-obese adults. Thirty participants were removed from the study because they had already been diagnosed with various diseases, refused to follow the study protocol, or had missing data. After the 30 subjects were eliminated, the final sample size was 90.

As shown in Table 1, obese individuals exhibited significantly higher weight, BMI, WC, and DBP in comparison with healthy group. No significant differences were found between healthy and obese groups regarding to the age, height, or SBP.

Table 1: Comparison of Anthropometric and Clinical Parameters Between Obese and Control Groups

Parameter (unit)	Control Group	obese Group	p-value
Age (years)	28.6 \pm 6.9	30.9 \pm 7.4	0.127
Weight (kg)	66.5 \pm 7.4	102.3 \pm 11.8	<0.001
Height (cm)	173 \pm 6.2	173 \pm 7.8	0.942
BMI (kg/m ²)	22.3(20.8-23.7)	33.5(32-35.9)	<0.001
WC (cm)	81.5(79-85.8)	112.5(108-117)	<0.001
SBP (mmHg)	123(117.3-127)	123(117.8-132)	0.174
DBP (mmHg)	72.8 \pm 9.1	81.1 \pm 10.04	<0.001

significant difference (p-value > 0.05), statistical significance (p < 0.05), and high significance (p < 0.01).

Table 2 shows that all lipid parameters and atherogenic indices were significantly elevated in the obese group. TC, LDL-C, VLDL-C, and TG were significantly higher (p < 0.001), while HDL-C was significantly lower. Ratios including LDL/HDL, TG/HDL, and TC/HDL were significantly increased in the obese group.

Table 2: Comparison of Lipid Profile and Atherogenic Indices Between Obese and Control Groups

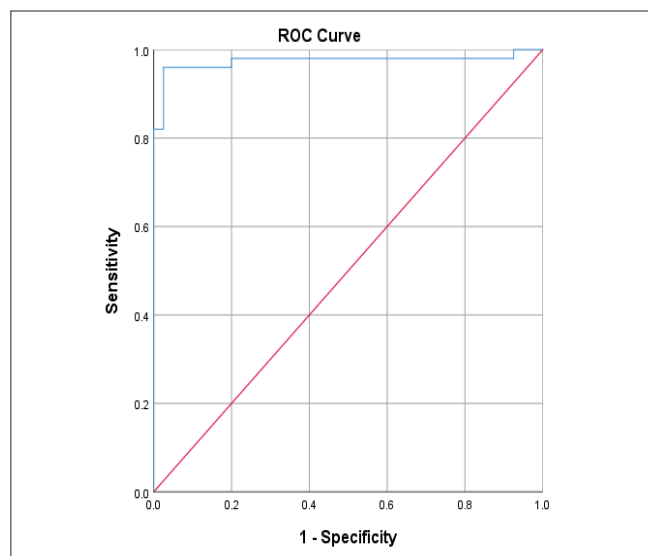
Parameter (unit)	Control Group	obese Group	p-value
TC (mg/dL)	137.4±23.98	174.7±29.5	<0.001
TG (mg/dL)	85.7(66.2-126.6)	176.2(130.3-230)	<0.001
HDL-C (mg/dL)	40(35-46.4)	34(30.6-36.8)	<0.001
LDL-C (mg/dL)	77.2 ±20.5	101.2 ±30.1	<0.001
VLDL (mg/dL)	17.13(13.24-25.31)	35.23(26.1-46)	<0.001
LDL/HDL Ratio	1.91±0.52	2.98±0.99	<0.001
TG/HDL Ratio	2.12(1.53-3.4)	5.41(3.4-6.8)	<0.001
TC/HDL	3.4±0.7	5.2±1.4	<0.001

As presented in Table 3, FBG, FI, HOMA-IR, HOMA- β , TyG index, and hs-CRP were all significantly elevated in obese individuals ($p < 0.001$). Serum visfatin concentrations are markedly elevated in the obese group compared to normal group [2.22 (2.02–2.41) ng/mL vs. 1.3 (1.1–1.5) ng/mL; $p < 0.001$].

Table 3: Comparison of Glycemic Indices, Inflammatory Marker, and Visfatin Levels Between Obese and Control Groups

Parameter (unit)	Control Group	obese Group	p-value
FBG (mg/dL)	82.5 ±6.11	90.1 ±9.2	<0.001
FI (μ IU/mL)	5.6(3.7-8.2)	20.7(15.4-27.6)	<0.001
HOMA-IR	1.2(0.8-1.6)	4.5(3.3-6.2)	<0.001
HOMA- β	103(73.1-130.3)	299.4(199.1-445.1)	<0.001
TyG Index	8.2±0.42	9±0.48	<0.001
hs-CRP (mg/L)	0.8(0-1.2)	2.6(1.6-5)	<0.001
Visfatin (ng/mL)	1.3(1.1-1.5)	2.22(2.02-2.41)	<0.001

The ROC curve analysis (Figure 1 and Table 4) revealed a high diagnostic accuracy of visfatin in distinguishing obese from non-obese individuals. The optimal cutoff value was 1.74 ng/mL, with 96% sensitivity, 97.5% specificity, and an AUC of 0.974 (95% CI: 0.936–1.000), showing an excellent discriminatory power.

**Figure 1:** ROC Curve for Visfatin in Predicting Obesity.**Table 4:** Cutoff Point for Visfatin

Cutoff Value (ng/mL)	Sensitivity (%)	Specificity (%)	p-value	Youden Index	AUC (95% CI)
1.74	96	97.5	<0.001	0.935	0.974 (0.936 – 1.000)

AUC = Area Under Curve; CI = Confidence Interval. Youden Index: Sensitivity + Specificity – 1.

As shown in Table 5, serum visfatin levels were significantly positively correlated with weight, BMI, WC, DBP, FI, HOMA-IR, HOMA- β , TyG index, hs-CRP, and all lipid parameters and their atherogenic indices (all $p < 0.001$). A significant negative correlation was observed with HDL-C ($r = -0.390$, $p < 0.001$).

DISCUSSION

In this study, we investigated serum visfatin levels in obese and non-obese adult males. We evaluated its correlation with a wide array of metabolic and biochemical parameters, including lipid profile, markers of insulin resistance, and systemic inflammation. Our findings reveal significantly elevated visfatin levels in the obese group than in the control group, supporting its classification as an obesity-associated adipocytokine. This result aligns with the findings reported by Tahir et al.¹⁰ and El-Foly et al.²², which similarly reported increased visfatin concentrations in individuals with higher adiposity.

The significant association of visfatin with BMI

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

parameters	r	p-value
Age (years)	0.076	0.475
Weight (kg)	0.686**	<0.001
Height (cm)	0.058	0.587
BMI (kg/m ²)	0.657**	<0.001
WC [cm]	0.650**	<0.001
SBP (mmHg)	0.007	0.951
DBP (mmHg)	0.333**	0.001
FBG (mg/dL)	0.446**	<0.001
TC (mg/dL)	0.434**	<0.001
TG (mg/dL)	0.513**	<0.001
HDL-C (mg/dL)	-0.390 **	<0.001
LDL-C (mg/dL)	0.363**	<0.001
VLDL-C(mg/dL)	0.513**	<0.001
LDL/HDL Ratio	0.497**	<0.001
TG/HDL Ratio	0.511**	<0.001
TC/HDL	0.555**	<0.001
hs-CRP (mg/L)	0.485**	<0.001
FI (μIU/mL)	0.726**	<0.001
HOMA-IR	0.735**	<0.001
HOMA-β	0.606**	<0.001
TyG Index	0.549**	<0.001

* Significant at the 0.05 level; ** significant at the 0.01 level.

and WC in our study suggests that visfatin is closely linked with both general and central obesity. These anthropometric indicators are well-known predictors of metabolic syndrome and cardiovascular risk. Our findings are in agreement with Sruti et al.¹⁷, who observed positive correlations between visfatin and abdominal fat accumulation. This relationship likely reflects increased visfatin production from visceral adipose tissue, notably exhibiting higher metabolic activity and a greater correlation with insulin resistance than subcutaneous fat.

Insulin resistance plays a central role in metabolic syndrome²³, and our results demonstrated significantly elevated levels of FI, HOMA-IR, and HOMA-β in the

obese group. Visfatin was strongly associated with these insulin resistance markers, indicating a potential role in glucose metabolism. Previous studies have proposed that visfatin might mimic insulin's action through its interaction with the insulin receptor, although this mechanism remains controversial²⁴. Our findings align with those of Alnowihi et al.²⁵, who reported that visfatin levels correlate positively with HOMA-IR in obese individuals. This supports the hypothesis that elevated visfatin levels may reflect an adaptive response to reduced insulin sensitivity or may be actively involved in the impairment of insulin signaling pathways.

In addition to insulin resistance, our study revealed profound alterations in lipid metabolism among obese participants. These individuals exhibited elevated TC, TG, LDL-C, and VLDL-C levels, along with reduced HDL-C. Visfatin was positively associated with all atherogenic lipids and inversely associated with HDL-C. These findings are consistent with those of Sruti et al.¹⁷, which reported elevated visfatin levels in dyslipidemic patients, correlating with adverse lipid profiles. The inverse association with HDL-C, a protective lipoprotein, may indicate that visfatin plays a role in promoting lipid imbalance and atherogenesis.

Importantly, we also observed significant relationships between visfatin and atherogenic lipid ratios, namely TC/HDL, TG/HDL, and LDL/HDL. These ratios are considered more informative predictors of cardiometabolic risk than individual lipid parameters. The TG/HDL ratio is recognized as a reliable marker for insulin resistance as well as cardiovascular disease, as it reflects the coexistence of hypertriglyceridemia and low HDL-C levels—two key features of atherogenic dyslipidemia^{26,27}. Our finding that visfatin correlates positively with TG/HDL suggests that elevated visfatin may indicate underlying insulin resistance and an increased atherogenic lipid burden. Likewise, higher LDL/HDL and TC/HDL ratios are indicative of pro-atherogenic lipid transport and enhanced cardiovascular risk. These associations support the interpretation that visfatin could function as an integrative biomarker, capturing the combined burden of cardiovascular disease, dyslipidemia, and risk insulin resistance in obese individuals.

Systemic inflammation is another hallmark of obesity, and based on our results, visfatin levels showed a positive correlation with hs-CRP, a sensitive marker of chronic inflammation. This supports earlier findings that

visfatin acts as a pro-inflammatory cytokine, promoting the expression of interleukins and other inflammatory mediators²⁸. The association between visfatin and hs-CRP further emphasizes its dual metabolic and immunologic roles in obesity. Taken together, these findings suggest that elevated visfatin reflects the chronic inflammation characteristic of visceral obesity and may play a role in advancing metabolic and vascular complications.

Furthermore, the diagnostic utility of visfatin was supported by the receiver operating characteristic (ROC) analysis, which indicated visfatin's strong ability to distinguish between obese and non-obese individuals. The high sensitivity and specificity observed reinforce its potential clinical value as a predictive biomarker for metabolic dysregulation, particularly in the context of obesity screening and risk stratification.

In this study, DBP was significantly higher in obese individuals than in controls, with a significant correlation between visfatin and DBP. The interaction of proinflammatory cytokines with the sympathetic nervous system and the renin-angiotensin system is one possible potential biological mechanism. These cytokines influence endothelial-derived factors and vascular function, which regulate blood pressure. Since endothelial dysfunction is a key factor in various types of hypertension, assessing visfatin levels could be valuable for detecting hypertension and understanding its underlying inflammatory processes. In contrast, SBP is more influenced by arterial stiffness and cardiac output, which may not differ significantly between the groups, especially if participants were younger. Additionally, visfatin's role in metabolic and cardiovascular pathways suggests it may be a biomarker for early hypertension-related vascular changes. The current study corroborates a previous study reported¹⁴. However, Ugur. et al.²⁹ demonstrated that obese adults exhibited significantly elevated DBP compared to their non-obese counterparts, whereas SBP showed no difference between the two groups.

Limitation

This study was conducted exclusively on adult male participants, which limits the generalizability of the findings to the female population. Sex-related differences in adipokine profiles, lipid metabolism, and insulin resistance are well-documented, and future research should address these differences. Additionally, the relatively small sample size and cross-sectional design limit the ability to infer causality.

CONCLUSION

Serum visfatin levels were significantly higher in obese adults compared to controls and showed strong positive correlations with BMI, waist circumference, fasting insulin, fasting blood glucose, lipid profile parameters, hs-CRP, HOMA-IR, and diastolic blood pressure, as well as a negative correlation with HDL-C. These findings suggest that visfatin may serve as a predictive biomarker of metabolic dysregulation in obesity. Future studies with larger and more diverse populations, including female participants, are needed to confirm these associations and clarify the underlying mechanisms.

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Conflict of interest: None.

Ethical approval

Ethical approval for this study was obtained in November 2024 from the Research Ethics Committee, College of Medicine, University of Zakho (Ref: NOV2024/UOZE28).

Research funding

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Informed Consent

Informed consent was obtained from all participants before their involvement.

Authors' contributions

The project framework was devised by Assistant Professor Dr. Lina Y. Mohammed, who also offered guidelines during the research period. Sozdar Ayoub Adil managed all facets of sample collection, biochemical testing, data analysis, and literature research. The manuscript of this study was composed by Sozdar Ayoub Adil. Both the author and the supervisor evaluated and ratified the final version.

Data Availability

The datasets utilized and/or produced in this study are not publicly available due to ethical and confidentiality considerations; however, they may be obtained from the corresponding author upon a reasonable request.

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