e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 2387-2393

Original Research Article

Serum Visfatin as a Marker of Hormonal and Metabolic Dysfunction in Women with Polycystic Ovary Syndrome: A Comparative Cross-Sectional Study

Rajeev Lohokare[1], Anwesha[2], Vandana Varma[3], Deepasha Shahi[4], Rajvardhan Sisodia[5], Pratiksha[6]

[1]Associate Professor & HOD, Department of Biochemistry, M.G.M. Medical College, Indore, Madhya Pradesh, India

[2]PG Resident, Department of Biochemistry, M.G.M. Medical College, Indore, Madhya Pradesh, India

[3]Associate Professor, Department of Biochemistry, M.G.M. Medical College, Indore, Madhya Pradesh, India

[4]Assistant Professor, Department of Biochemistry, M.G.M. Medical College, Indore, Madhya Pradesh, India

[5]MBBS Student, M.G.M. Medical College, Indore, Madhya Pradesh, India

[6]Intern, MBBS Graduate, Hi-Tech Medical College and Hospital, Bhubaneswar, Odisha, India

Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026

Corresponding author: Dr. Anwesha

Conflict of interest: Nil

Abstract

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder associated with hormonal imbalance, insulin resistance, and metabolic abnormalities. Visfatin, an adipokine involved in glucose metabolism and inflammation, may play a role in its pathophysiology.

Methods: This comparative cross-sectional study included 210 women (105 with PCOS and 105 age- and body mass index-matched healthy controls). Serum visfatin, luteinizing hormone (LH), follicle-stimulating hormone (FSH), testosterone, glycated hemoglobin (HbA1c), and lipid profile were measured. Group comparisons were performed using the independent samples t-test. Pearson's correlation and multivariable logistic regression analyses were used to assess the association of visfatin with hormonal and metabolic parameters.

Results: Age and BMI were comparable between the groups. Women with PCOS had significantly higher serum visfatin, LH, LH/FSH ratio, testosterone, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and HbA1c levels, while FSH and HDL cholesterol levels were significantly lower (all $p<0.001$). Serum visfatin showed significant correlations with hormonal and metabolic parameters and remained an independent predictor of PCOS after multivariable adjustment.

Conclusion: Serum visfatin is significantly elevated in women with PCOS and is independently associated with endocrine and metabolic dysfunction, suggesting its potential utility as a biomarker in PCOS.

Keywords: Polycystic ovary syndrome; Visfatin; Adipokine; Hyperandrogenism; Insulin resistance; Biomarker.

DOI: 10.25258/ijcpr.18.8.351

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with a prevalence ranging from 6–20% worldwide and up to 36% in certain Indian populations. It is characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology, and is increasingly recognized as a disorder with both reproductive and metabolic consequences.

Women with PCOS frequently experience menstrual irregularities, infertility, obesity, insulin resistance, and an increased risk of metabolic syndrome and type 2 diabetes mellitus. [1] Although the exact pathogenesis of PCOS remains unclear, insulin resistance is considered a key underlying mechanism. Approximately 50–70% of affected women exhibit varying degrees of insulin resistance, which contributes to compensatory hyperinsulinemia, increased ovarian androgen production, and impaired follicular development. [2] The close relationship between metabolic and reproductive abnormalities has prompted interest in identifying biomarkers that may help explain the pathophysiology of PCOS.

Adipose tissue is now recognized as an active endocrine organ that secretes several biologically active molecules known as adipokines. These molecules regulate glucose metabolism, insulin sensitivity, inflammation, and reproductive function. Altered adipokine secretion has been implicated in the development of obesity-associated insulin resistance and chronic low-grade inflammation, both of which are important features of PCOS. [3] Visfatin, also known as nicotinamide phosphoribosyltransferase (NAMPT), is an adipokine predominantly produced by visceral adipose tissue. Beyond its role in nicotinamide adenine dinucleotide (NAD^+) biosynthesis, visfatin has been shown to influence glucose homeostasis, inflammatory pathways, and cellular metabolism. Experimental studies suggest that visfatin possesses insulin-mimetic properties and may contribute to the regulation of energy balance and metabolic function. [4,5] Elevated circulating visfatin levels have been reported in obesity, metabolic syndrome, and type 2 diabetes mellitus, indicating a possible association with insulin resistance and inflammation. [6]

Several studies have investigated serum visfatin levels in women with PCOS; however, the findings remain inconsistent. While some researchers have reported significantly higher visfatin concentrations in women with PCOS independent of body mass index, others have suggested that observed differences are primarily related to obesity and associated metabolic disturbances. [7] Furthermore, visfatin has been identified in ovarian granulosa cells and may play a role in follicular development and steroidogenesis, suggesting a potential link between metabolic dysfunction and reproductive abnormalities in PCOS. [8]

Although previous meta-analyses have reported elevated serum visfatin levels in women with polycystic ovary syndrome (PCOS), the available evidence remains heterogeneous because of differences in ethnicity, obesity status, PCOS phenotype, and study methodology. Furthermore, data from the Indian population are limited, and few studies have comprehensively evaluated the association of visfatin with hormonal and metabolic parameters in age- and BMI-matched women. Therefore, the present comparative cross-sectional study was undertaken to compare serum visfatin levels between women with PCOS and healthy controls and to evaluate its association with hormonal and metabolic parameters, thereby providing additional evidence from an Indian population.

Materials and Methods

This comparative cross-sectional analytical study was conducted in the Department of Clinical Biochemistry in collaboration with the Department of Obstetrics and Gynaecology at Mahatma Gandhi Memorial Medical College and M.T.H. Hospital, Indore, over a period of one year. Ethical approval was obtained from the Ethics and Scientific Review Committee, M.G.M. Medical College and M.Y. Hospital, Indore (Approval No. ESC/MGM/MAY-24/155, dated May 30, 2024). Written informed consent was obtained from all participants before enrolment. The sample size was calculated using the formula for comparison of two independent means with a 95% confidence level and 80% power. Based on the expected difference in serum visfatin levels between women with PCOS and healthy controls reported in previous studies, the minimum required sample size was estimated to be 103 participants per group. To compensate for possible attrition, 105 participants were enrolled in each group.

Accordingly, a total of 210 women were included in the study, comprising 105 women diagnosed with polycystic ovary syndrome (PCOS) and 105 age- and body mass index (BMI)-matched healthy controls. Women with PCOS were recruited from the outpatient clinic of the Department of Obstetrics and Gynaecology. The diagnosis of PCOS was established according to the Rotterdam criteria, requiring the presence of at least two of the following features: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography. Healthy women without clinical or biochemical evidence of PCOS served as controls. Participants were recruited using a convenience sampling technique.

Women aged 18–35 years who provided written informed consent were eligible for inclusion. Pregnant or lactating women, women with malignancies, premature menopause, known endocrine disorders such as Cushing's syndrome, or those receiving hormonal therapy were excluded from the study.

Demographic and clinical information, including age, BMI, and relevant medical history, was recorded using a predesigned proforma. The primary outcome measure was serum visfatin concentration. Secondary outcome measures included serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), testosterone, glycated hemoglobin (HbA1c), and lipid profile parameters comprising total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C).

After an overnight fast of 8–10 hours, 5 mL of venous blood was collected from each participant between 8:00 and 10:00 AM under aseptic conditions. Blood samples were collected during the early follicular phase of the menstrual cycle (days 2–5) whenever feasible to minimize

physiological variations in reproductive hormone levels. Samples were allowed to clot for 30 minutes at room temperature and were centrifuged at 3000 rpm for 10 minutes. The separated serum was aliquoted and stored at −20°C until biochemical analysis. Serum visfatin concentrations were measured using a commercially available Human Visfatin (NAMPT/PBEF) sandwich enzyme-linked immunosorbent assay (ELISA) kit (Elabscience Biotechnology Inc., Houston, TX, USA) according to the manufacturer's instructions. The assay had a measurement range of 1–120 ng/mL, with intra-assay and inter-assay coefficients of variation of <10% and <12%, respectively. All samples were analyzed in duplicate, and absorbance was measured at 450 nm using a calibrated ELISA microplate reader. Internal quality-control procedures were followed throughout the analysis.

Serum LH, FSH, and testosterone concentrations were determined using chemiluminescent immunoassays on the UniCel DxI 800 Immunoassay System (Beckman Coulter, Brea, CA, USA) following the manufacturer's instructions. Serum total cholesterol, triglycerides, and HDL-C were estimated by enzymatic colorimetric methods using the AU5800 Clinical Chemistry Analyzer (Beckman Coulter, Brea, CA, USA). LDL-C and VLDL-C were calculated using the Friedewald equation wherever applicable. Glycated hemoglobin (HbA1c) was measured by ion-exchange high-performance liquid chromatography (HPLC) using the Bio-Rad D-10 HbA1c Analyzer (Bio-Rad Laboratories, Hercules, CA, USA). Internal quality-control materials, calibration standards, and routine maintenance procedures were employed throughout the study in accordance with standard laboratory protocols.

Data were entered into Microsoft Excel and analyzed using JAMOVI statistical software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between the PCOS and control groups were performed using the independent samples t-test. Effect sizes (Cohen's d) with 95% confidence intervals were calculated for group comparisons. Pearson's correlation coefficient was used to evaluate the relationship between serum visfatin levels and hormonal, anthropometric, and biochemical parameters within the PCOS group. To determine whether serum visfatin was independently associated with PCOS, multivariable logistic regression analysis was performed after adjustment for age, BMI, testosterone and HbA1c. A p-value of <0.05 was considered statistically significant.

Results

A total of 210 women were included in the study, comprising 105 women with PCOS and 105 healthy controls. There were no statistically significant differences between the two groups with respect to age or body mass index (BMI), indicating that the groups were comparable in terms of baseline sociodemographic and anthropometric characteristics (Table 1).

Women with PCOS demonstrated significant hormonal alterations compared with healthy controls. Serum luteinizing hormone (LH), LH/FSH ratio, testosterone, and serum visfatin levels were significantly higher, whereas follicle-stimulating hormone (FSH) levels were significantly lower in the PCOS group. Large effect sizes were observed for most hormonal parameters, particularly testosterone and LH/FSH ratio, indicating marked endocrine differences between the two groups (Table 2).

Assessment of metabolic parameters showed that women with PCOS had significantly less favorable metabolic profiles than healthy controls. They exhibited higher total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and HbA1c levels, along with significantly lower HDL cholesterol levels. The observed differences were statistically significant and demonstrated moderate to large effect sizes, suggesting clinically meaningful metabolic alterations in women with PCOS (Table 3).

Correlation analysis within the PCOS group revealed that serum visfatin was significantly positively correlated with LH, testosterone, BMI, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and HbA1c, while significant negative correlations were observed with FSH and HDL cholesterol.

No significant correlation was found between serum visfatin levels and the LH/FSH ratio (Table 4).

Multivariable logistic regression analysis demonstrated that serum visfatin remained independently associated with PCOS after adjustment for age, BMI, testosterone, and HbA1c. Testosterone was also identified as an independent predictor of PCOS, whereas age and HbA1c were not independently associated with disease status. BMI showed a modest independent association after adjustment for other variables (Table 5).

Table 1: Comparison of Sociodemographic and Anthropometric Parameters Between PCOS and Control Groups

Parameter	PCOS (n=105)	Control (n=105)	Mean Difference (95% CI)	Cohen's d	p-value
Age (years)	26.84 ± 4.12	26.31 ± 3.98	0.53 (−0.57 to 1.63)	0.13	0.412
BMI (kg/m²)	27.92 ± 3.85	27.11 ± 3.64	0.81 (−0.21 to 1.83)	0.22	0.138

Table 2: Comparison of Hormonal Parameters and Serum Visfatin Levels between PCOS and Control Groups

Parameter	PCOS (n=105)	Control (n=105)	Mean Difference (95% CI)	Cohen's d	p-value
LH (IU/L)	12.84 ± 4.21	7.62 ± 2.88	5.22 (4.23 to 6.21)	1.45	<0.001
FSH (mIU/mL)	5.92 ± 1.68	7.18 ± 2.01	−1.26 (−1.76 to −0.76)	−0.68	<0.001
LH/FSH Ratio	2.18 ± 0.74	1.06 ± 0.32	1.12 (0.96 to 1.28)	1.94	<0.001
Testosterone (ng/dL)	58.43 ± 14.25	32.76 ± 10.18	25.67 (22.36 to 28.98)	2.08	<0.001
Serum Visfatin (ng/mL)	36.78 ± 11.92	24.63 ± 8.47	12.15 (9.35 to 14.95)	1.17	<0.001

Table 3: Comparison of Metabolic Parameters between PCOS and Control Groups

Parameter	PCOS (n=105)	Control (n=105)	Mean Difference (95% CI)	Cohen's d	p-value
Total Cholesterol (mg/dL)	198.42 ± 34.11	172.36 ± 28.74	26.06 (17.45 to 34.67)	0.83	<0.001
Triglycerides (mg/dL)	162.58 ± 41.27	128.44 ± 36.12	34.14 (23.59 to 44.69)	0.88	<0.001
HDL Cholesterol (mg/dL)	38.21 ± 7.45	48.62 ± 8.11	−10.41 (−12.52 to −8.30)	−1.34	<0.001
LDL Cholesterol (mg/dL)	122.67 ± 26.38	98.43 ± 21.77	24.24 (17.62 to 30.86)	1.00	<0.001
VLDL Cholesterol (mg/dL)	32.52 ± 8.25	25.68 ± 7.22	6.84 (4.74 to 8.94)	0.88	<0.001
HbA1c (%)	5.84 ± 0.62	5.21 ± 0.48	0.63 (0.48 to 0.78)	1.13	<0.001
Adjusted odds ratios were obtained using a multivariable logistic regression model that included serum visfatin, age, BMI, testosterone, and HbA1c. Each estimate was adjusted for all other variables included in the model. OR, odds ratio; CI, confidence interval; BMI, body mass index; HbA1c, glycated haemoglobin. A p-value <0.05 was considered statistically significant.					

Table 4: Correlation of Serum Visfatin with Hormonal and Biochemical Parameters in PCOS Group

Variables	Correlation Coefficient (r)	p-value
Visfatin vs LH	0.42	0.006
Visfatin vs FSH	-0.28	0.004
Visfatin vs Testosterone	0.64	<0.001
Visfatin vs BMI	0.33	0.021
Visfatin vs Total Cholesterol	0.39	0.012
Visfatin vs Triglycerides	0.44	0.003
Visfatin vs HDL	-0.36	0.015
Visfatin vs LDL	0.41	0.007
Visfatin vs VLDL	0.43	0.005
Visfatin vs HbA1c	0.57	<0.001
Visfatin vs LH/FSH Ratio	-0.024	0.806

Table 5: Multivariable Logistic Regression Analysis Showing Factors Independently Associated with PCOS

Variable	Adjusted OR	95% CI	p-value
Serum visfatin	3.11	1.62 to 5.98	0.001
Age	0.87	0.69 to 1.11	0.274
BMI	0.64	0.41 to 0.99	0.046
Testosterone	1.17	1.06 to 1.30	0.003
HbA1c	2.26	0.34 to 15.05	0.399

Discussion

The present study evaluated serum visfatin levels in women with polycystic ovary syndrome (PCOS) and investigated their association with hormonal and metabolic parameters. Women with PCOS had significantly higher serum visfatin concentrations than healthy controls, together with characteristic hormonal alterations, dyslipidemia, and higher HbA1c levels.

Correlation analysis further demonstrated significant associations between serum visfatin and several endocrine and metabolic variables, while multivariable logistic regression confirmed that serum visfatin remained independently associated with PCOS after adjustment for age, BMI, testosterone, and HbA1c. These findings support the potential role of visfatin as a marker of endocrine and metabolic dysfunction in women with PCOS.

The absence of significant differences in age and BMI between the two groups indicates that the observed biochemical differences were unlikely to be explained by these baseline characteristics. Similar study designs using comparable control groups have been reported by Ali and Nori and Al-Ghazali et al., thereby reducing the influence of major confounding factors during group comparisons. [9,10]

Women with PCOS demonstrated significantly higher LH levels, LH/FSH ratio, testosterone, and serum visfatin concentrations, along with significantly lower FSH levels. These findings are consistent with the well-established endocrine abnormalities of PCOS, characterized by altered gonadotropin secretion and hyperandrogenism. Similar hormonal alterations have been reported by Rashad et al. and Kim et al., who also observed associations between elevated visfatin levels and androgen excess in women with PCOS. [6,11]

The principal finding of the present study was the significantly elevated serum visfatin concentration in women with PCOS. Visfatin, also known as nicotinamide phosphoribosyltransferase (NAMPT), is involved in glucose metabolism, inflammatory signaling, and cellular energy homeostasis. Elevated circulating visfatin levels may therefore reflect the combined effects of insulin resistance, chronic low-grade inflammation, and endocrine dysregulation that characterize PCOS. Our findings are consistent with those reported by Ali and Nori and Al-Ghazali et al., who demonstrated significantly higher serum visfatin levels among women with PCOS. [9,10] Furthermore, the meta-analyses by Sun et al. and Chen et al. concluded that circulating visfatin concentrations are generally higher in women with PCOS than in healthy controls, supporting its possible involvement in the pathophysiology of the disorder. [12,13] In contrast, Güdücü et al. and Kim et al. reported no significant differences in serum visfatin levels between women with PCOS and controls. [6,14] Variations in ethnicity, obesity status, diagnostic criteria, assay methodology, and study population characteristics may explain these inconsistent findings. Nevertheless, the present results add to the growing evidence supporting elevated serum visfatin as a common biochemical feature of PCOS.

Women with PCOS also demonstrated significantly less favorable metabolic profiles, including higher total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and HbA1c levels, together with lower HDL cholesterol levels. These findings are consistent with the increased cardiometabolic risk associated with PCOS and agree with the observations reported by Pareek et al., who described a high prevalence of dyslipidemia among women with the syndrome. [15]

Correlation analysis demonstrated that serum visfatin was positively associated with LH, testosterone, BMI, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and HbA1c, while negative correlations were observed with FSH and HDL cholesterol. Among these, the strongest positive correlation was observed with testosterone, suggesting a close relationship between visfatin and hyperandrogenism. Similar associations between serum visfatin and androgen excess have been reported by Ali and Nori and Rashad et al., who also observed positive correlations with testosterone in women with PCOS. [9,11] In addition, studies by Tsouma et al. demonstrated significant associations between visfatin and adverse lipid parameters, while Kuai et al. reported positive correlations between visfatin and body mass index in women with PCOS. [7,16] These findings collectively support the close relationship between elevated serum visfatin and the endocrine and metabolic abnormalities characteristic of PCOS.

An important finding of the present study was that serum visfatin remained independently associated with PCOS after adjustment for age, BMI, testosterone, and HbA1c in multivariable logistic regression analysis. This suggests that the observed association between visfatin and PCOS is not explained solely by differences in adiposity or glycemic status and supports the possibility that visfatin may represent an independent biochemical marker of the syndrome. However, these findings should be interpreted cautiously because observational studies cannot establish causal relationships.

Overall, the present findings indicate that elevated serum visfatin is closely associated with both

endocrine and metabolic abnormalities in women with PCOS. Significant associations with reproductive hormones, lipid abnormalities, BMI, and glycemic status support the concept that visfatin may act as an important link between metabolic dysfunction and reproductive disturbances. These findings suggest that serum visfatin may have potential clinical utility as a biomarker of endocrine and metabolic dysfunction in women with PCOS.

The strengths of the present study include its comparative cross-sectional design, adequate sample size, and inclusion of healthy controls with comparable baseline characteristics, which minimized the influence of major confounding factors. Furthermore, the simultaneous evaluation of serum visfatin, reproductive hormones, lipid profile, and HbA1c provided a comprehensive assessment of the endocrine and metabolic status of women with PCOS. The inclusion of effect sizes, confidence intervals, and multivariable regression analysis further strengthened the statistical interpretation of the findings. The study has several limitations. Convenience sampling may have introduced selection bias and may limit the generalizability of the findings. Although multivariable regression was performed to adjust for selected confounders, residual confounding cannot be excluded. In addition, the cross-sectional design demonstrates association rather than causation or predictive value, and therefore it cannot determine whether elevated serum visfatin precedes or results from PCOS. Direct measures of insulin resistance such as HOMA-IR and inflammatory biomarkers were not assessed, which may have provided additional insight into the underlying mechanisms. Future multicentric prospective longitudinal studies incorporating comprehensive metabolic and inflammatory assessments are warranted to further clarify the diagnostic and prognostic value of serum visfatin in PCOS.

Conclusion

Women with PCOS exhibited significantly higher serum visfatin levels than healthy controls. Serum visfatin was significantly associated with hormonal abnormalities, dyslipidemia, body mass index, and HbA1c, and remained independently associated with PCOS after adjustment for potential confounding factors. These findings suggest that serum visfatin may serve as a promising biomarker of endocrine and metabolic dysfunction in women with PCOS. However, prospective longitudinal studies are required to establish its diagnostic and prognostic utility.

References

1. Bharali MD, Rajendran R, Goswami J, Singal K, Rajendran V. Prevalence of Polycystic Ovarian Syndrome in India: A Systematic Review and Meta-Analysis. Cureus. 2022;14(12):e32351.
2. Khan MJ, Ullah A, Basit S. Genetic Basis of Polycystic Ovary Syndrome (PCOS): Current Perspectives. Appl Clin Genet. 2019;12:249-260.
3. Schüler-Toprak S, Ortmann O, Buechler C, Treeck O. The Complex Roles of Adipokines in Polycystic Ovary Syndrome and Endometriosis. Biomedicines. 2022;10(10): 2503.
4. Dakroub, A.; A. Nasser, S.; Younis, N.; Bhagani, H.; Al-Dhaheri, Y.; Pintus, G.; Eid, A.A.; El-Yazbi, A.F.; Eid, A.H. Visfatin: A Possible Role in Cardiovasculo-Metabolic Disorders. Cells 2020, 9, 2444.
5. Kumari, B., & Yadav, U. C. S. (2018). Adipokine Visfatin's Role in Pathogenesis of Diabesity and Related Metabolic Derangements. Current molecular medicine, 18(2), 116–125.
6. Kim JJ, Choi YM, Hong MA, Kim MJ, Chae SJ, Kim SM, Hwang KR, Yoon SH, Ku SY, Suh CS, Kim SH. Serum visfatin levels in non-obese women with polycystic ovary syndrome and matched controls. Obstet Gynecol Sci. 2018;61(2):253-60.
7. Kuai D, Tang Q, Wang X, Yan Q, Tian W, Zhang H. Relationship between serum apelin, visfatin levels, and body composition in Polycystic Ovary Syndrome patients. Eur J Obstet Gynecol Reprod Biol. 2024;297:24-29.
8. Reverchon, M., Cornuau, M., Cloix, L., Ramé, C., Guerif, F., Royère, D., & Dupont, J. (2013). Visfatin is expressed in human granulosa cells: regulation by metformin through AMPK/SIRT1 pathways and its role in steroidogenesis. Molecular human reproduction, 19(5), 313–326.
9. Ali AI, Nori W. Correlation of Serum Visfatin Level in Non-obese Women with Polycystic Ovary Syndrome and Matched Control. Reprod Sci. 2022;29:3285-3293.
10. Al-Ghazali BS, Mohammed AA, Fahad AM. The association of serum visfatin in women with polycystic ovary syndrome: A case-control study. Revis Bionatura. 2022;7(4):60.
11. Rashad NM, Abd-Elrahman MNA, El-Shal AS, Amin AI. Serum visfatin as predictive marker of cardiometabolic risk in women with polycystic ovary syndrome. Middle East Fertil Soc J. 2018;23(4):419–26.
12. Chen J, Hu L, Ling Y, Xu Y, et al. Risk correlation analysis between polycystic ovary syndrome (PCOS) and serum visfatin levels in middle-aged women: systematic review and

meta-analysis. Discov Med. 2023;35(175):168-75.
13. Sun Y, Wu Z, Wei L, Liu C, Zhu S, Tang S. High-visfatin levels in women with polycystic ovary syndrome: evidence from a meta-analysis. Gynecol Endocrinol. 2015;31(10):808-14.
14. Güdücü N, İşçi H, Görmüş U, Yiğiter AB, Dünder I. Serum visfatin levels in women with polycystic ovary syndrome. Gynecol Endocrinol. 2012;28(8):619-623.
15. Pareek B, Kabra C, Beniwal A, Verma M. Comparison of lipid profile in women suffering from polycystic ovarian syndrome and healthy women: A cross sectional study. Int J Pharm Clin Res. 2024;16(3):1745-1750.
16. Tsouma I, Kouskouni E, Demeridou S, Boutsikou M, Hassiakos DK, Chasiakou A, et al. Correlation of visfatin levels and lipoprotein lipid profiles in women with polycystic ovary syndrome undergoing ovarian stimulation. Gynecol Endocrinol. 2014;30(7):516-9.