



Student's Journal of Health Research Africa

e-ISSN: 2709-9997, p-ISSN: 3006-1059

Vol.6 No. 12 (2025): December 2025 Issue

<https://doi.org/10.51168/sjhrafrica.v6i12.2347>

Original Article

Evaluation of serum nesfatin-1 levels in polycystic ovarian syndrome women: A cross-sectional case-control study.

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Page | 1

Abstract

Background:

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder associated with metabolic and hormonal disturbances. Nesfatin-1 is emerging as a potential biomarker linked to insulin resistance and ovarian dysfunction.

Objective:

To evaluate serum nesfatin-1 levels in women with PCOS and assess their correlation with clinical, metabolic, and hormonal parameters.

Methods:

This cross-sectional case-control study included 50 women with PCOS and 50 age- and BMI-matched healthy controls. Clinical features, metabolic parameters, and hormonal profiles were assessed. Serum nesfatin-1 levels were measured using ELISA.

Results:

Mean serum nesfatin-1 levels were significantly higher in the PCOS group (6.03 ± 1.58 ng/ml) compared to controls (1.98 ± 1.00 ng/ml). Triglycerides (80.76 ± 12.32 mg/dl) and LDL cholesterol (99.86 ± 9.91 mg/dl) showed positive correlation, while HDL cholesterol showed negative correlation with nesfatin-1 levels. Hormonal parameters, including FSH (7.32 ± 2.92 mIU/ml), LH (6.58 ± 3.81 mIU/ml), and serum insulin (9.71 ± 7.56 μ U/ml), were positively correlated with nesfatin-1.

Conclusion:

Serum nesfatin-1 levels are significantly elevated in PCOS women and are associated with metabolic and hormonal disturbances.

Recommendations:

Nesfatin-1 may serve as a useful biomarker for early detection and metabolic risk stratification in PCOS patients.

Keywords: Polycystic ovarian syndrome, Nesfatin-1, insulin resistance, metabolic parameters, hormonal profile

Submitted: September 17, 2025 **Accepted:** October 10, 2025 **Published:** December 30, 2025

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Introduction

Polycystic ovarian syndrome (PCOS) is an endocrine abnormality which necessitates the presence of two out of the following three criteria: - oligo - and/or anovulation, - hyperandrogenism (biochemical and/or clinical), or ultrasonic polycystic ovaries, after exclusion of other causes of androgen excess and menstrual cycle irregularity or amenorrhoea.¹ It was estimated that 5–10% of women of reproductive age, whereas 15–20% of infertile women were affected by PCOS, with symptoms

usually seen after adulthood and adolescence.¹ High serum concentrations of androgenic hormones, such as testosterone, androstenedione, and dehydroepiandrosterone sulfate, may be noticed in those patients.¹ Irregular menstrual cycles, anovulation and acne are characteristics of average pubertal growth and PCOS. Numerous pathways can be associated with the pathophysiology of PCOS. It is believed that the syndrome is defined by complex correlations between hypothalamic-pituitary-ovarian or hypothalamic-pituitary-

adrenal axis activities and metabolic disturbances such as insulin resistance (IR), obesity, and compensatory hyperinsulinemia.²

Rotterdam “criteria are commonly used for PCOS diagnosis, involving at least 2 of the following criteria: chronic anovulation, hyperandrogenism, and polycystic ovaries.³ Young adults with symptoms of androgen overload and amenorrhea/oligomenorrhea are considered at risk for PCOS until a conclusive diagnosis of PCOS is established.

Nesfatin-1 is a novel hormone derived from nucleobindin-2 (NUCB2) precursor protein containing 82-aminoacids and have a molecular weight of 9.8 kDa.⁴ Nesfatin-1 is produced in various tissues and organs throughout the body including brain stem, forebrain, midbrain nuclei, pituitary, hypothalamus, peripheral adipose tissue, gastric mucosa, pancreatic endocrine beta cells, etc.⁸ Nesfatin-1 is a “potent anorexigenic peptide having antihyperglycemic effects and is associated with energy balance and homeostasis, glucose metabolism, obesity and probably gonadal functions.⁸

Recently, it was shown that nesfatin-1 promotes Ca²⁺ influx through L-type channels and enhances glucose-induced insulin secretion in mouse islet β -cells.⁹ Intracerebroventricular administration of nesfatin-1 induces decreases in food intake and body weight and increases in sympathetic nerve activity and mean arterial pressure.¹⁰ Nesfatin-1 has been shown to have a significant impact on glucose metabolism, influencing various aspects, energy dynamics, obesity, and low levels associated with delayed puberty suggest that Nesfatin-1 has a role in both normal and pathological ovarian function and reproduction. Regulation of the reproductive system, such as the initiation of puberty and gonadotropin secretion, can also be impaired.⁷ A microenvironment made up of several regulatory proteins, hormones, and metabolites provides the follicular fluid that supports oocyte development and maturation.^{11,12} The results of follicular fluid incorporation on oocyte maturation have suggested that Nesfatin-1 levels could be linked with ovarian dysfunction in PCOS patients such as oligo/anovulation, “polycystic ovaries,” etc.¹⁰ Previously, low nesfatin-1 levels were found to be associated with delayed puberty in female rats and nesfatin-1 injection had dramatic effects on the levels of luteinizing hormone (LH) and follicle stimulating hormone (FSH).¹⁰ These factors are critical in the development of polycystic ovarian syndrome.

The aim of the study is to investigate the serum levels of nesfatin-1 in women with PCOS and explore the potential correlations with various clinical, metabolic, and hormonal factors. Therefore, “the purpose of this study

was to evaluate serum Nesfatin-1 levels in polycystic ovarian syndrome and to compare its levels in non-polycystic ovarian syndrome women.”

Methods

Study design

This was a cross-sectional case-control study. Women diagnosed with PCOS constituted the case group, while age- and BMI-matched healthy women formed the control group to evaluate differences in nesfatin-1 levels and associated parameters.

Study setting

The study was conducted in the Department of Pathology in collaboration with the Department of Obstetrics and Gynaecology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India — a tertiary care referral hospital catering to urban and rural populations of eastern Uttar Pradesh and adjoining regions. The study was carried out over one year.

Participants

Cases included women aged 18–35 years diagnosed with PCOS based on Rotterdam criteria. Controls were healthy women without clinical or ultrasonographic features of PCOS, matched for age and BMI.

Variables

Clinical variables included BMI, acne, hirsutism, and acanthosis nigricans. Metabolic variables included fasting glucose and lipid profile. Hormonal variables included FSH, LH, testosterone, insulin, and TSH.

Data sources and measurements

Venous blood samples were collected after overnight fasting. Hormonal assays were performed using standard laboratory methods. Serum nesfatin-1 was measured using ELISA.

Bias

Selection bias was minimized by using standardized diagnostic criteria and matching controls for age and BMI. Laboratory measurements were performed uniformly.

Study size

Sample size was calculated using the formula:

$$n = Z^2 \times p \times q / d^2$$

Based on the estimated prevalence and 95% confidence interval, 50 cases and 50 controls were included.

Statistical analysis

SPSS version 16.0 was used. Student's t-test, Mann-Whitney U test, and Pearson correlation were applied. $p < 0.05$ was considered significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee of Baba Raghav Das Medical College, Gorakhpur (ethical clearance obtained prior to

commencement). Written informed consent was taken from all participants.

Results

A total of 130 women were screened during the study period. Of these, 100 women fulfilled eligibility criteria and consented to participate. Fifty women diagnosed with PCOS were included as cases, and fifty age- and BMI-matched healthy women were included as controls. Thirty women were excluded due to the presence of chronic illnesses, metabolic disorders, or refusal to participate.

Demographical characteristics and biochemical values of PCOS patients and healthy controls are shown in Table 1.

	CONTROL (N=50)	PCOS (N=50)
Age (years)	26.92 $\pm$ 3.90	24.48 $\pm$ 3.81
Weight (kgs)	47.92 $\pm$ 6.50	50.3 $\pm$ 7.19
Height (m)	155.72 $\pm$ 5.87	156.5 $\pm$ 6.03
BMI (kg/m ²)	19.91 $\pm$ 2.32	21.71 $\pm$ 2.42
FBS (mg/dl)	92.28 $\pm$ 5.41	94.58 $\pm$ 5.68
Trigly(mg/dl)	69.56 $\pm$ 14.40	80.76 $\pm$ 12.32
HDL (mg/dl)	53.4 $\pm$ 9.58	52.34 $\pm$ 8.16
LDL (mg/dl)	97.5 $\pm$ 10.9	99.86 $\pm$ 9.91
Free testosterone (ng/dl)	1.232 $\pm$ 0.75	1.46 $\pm$ 0.43
S.Insulin (Uu/ml)	8.484 $\pm$ 6.45	9.712 $\pm$ 7.56
FSH (mIU/ml)	6.312 $\pm$ 3.00	7.32 $\pm$ 2.92
LH (mIU/ml)	5.05 $\pm$ 3.00	6.58 $\pm$ 3.81
Nesfatin-1 (ng/ml)	1.98 $\pm$ 1.00	6.03 $\pm$ 1.58

The ages of the patients ranged from 18 to 33 years, with the mean age of 24.84 $\pm$ 3.55 years in PCOS women and 26.92 $\pm$ 3.90 years in the control group, respectively. The majority of women in the PCOS and non-PCOS groups

were between 20 and 30 years of age. In this study, 42% of PCOS patients had high BMI, wherein 34% were overweight, and 8% were obese, as compared to the control group with high BMI (10%).

Table 2: Comparison of mean Nesfatin-1 (ng/ml) with age group in both the PCOS and the control age group.

AGE GROUP (years)	PCOS GROUP (N=50)	CONTROL GROUP (N=50)
18-25	5.9 $\pm$ 1.3	0.60 $\pm$ 0.4
26-30	7.0 $\pm$ 2.1	0.72 $\pm$ 0.3
>30	7.3 $\pm$ 2.8	0.95 $\pm$ 1.3

For patients between 18 and 25 years, the mean Nesfatin-1 level in cases was 5.9 $\pm$ 1.3 ng/ml, while in controls it was significantly lower at 0.60 $\pm$ 0.4 ng/ml.

For patients between 26 and 30 years, the mean Nesfatin-1 level in cases was 7.0 $\pm$ 2.1 ng/ml, while in controls it was significantly lower at 0.72 $\pm$ 0.3 ng/ml.

Patients above 30 years, the mean Nesfatin-1 level in cases was 7.3 $\pm$ 2.8 ng/ml, while in controls it was significantly lower at 0.95 $\pm$ 1.3 ng/ml.

Table 3: **Nesfatin-1 levels and their significant differences between the subgroups of BMI.**

NESFATIN-1 LEVELS (ng/ml)					
	PCOS		CONTROL GROUP		
	GROUP				
	MEAN	STANDARD DEVIATION	MEAN	STANDARD DEVIATION	P VALUE
BMI < 25	6.443	3.39	1.461	0.23	0.014
BMI ≥ 25	5.011	0.36	2.13	0.44	0.038
BMI <20	5.864	1.08	2.21	0.78	0.010
20 ≤ BMI < 25	4.93	0.98	1.92	0.85	0.036
25 ≤ BMI < 30	5.00	0.36	1.98	0.25	0.009
BMI ≥ 30	0	0	0	0	0

Table 3 shows that in all the subgroups of BMI, nesfatin-1 was significantly lower in the control group than in PCOS cases.

Table 4: **Evaluation of metabolic parameters in relation to serum nesfatin-1.**

	CONTROL (N=50)	PCOS (N=50)
Trigly(mg/dl)	69.56 ± 14.40	80.76 ± 12.32
HDL (mg/dl)	53.4 ± 9.58	52.34 ± 8.16
LDL (mg/dl)	97.5 ± 10.9	99.86 ± 9.91
Nesfatin-1 (ng/ml)	1.98 ± 1.00	6.03 ± 1.58

Table 4 shows that LDL and triglyceride parameters are positively correlated with Serum Nesfatin 1 levels. There is a negative correlation between HDL levels [(PCOS group- 52.34 ± 8.16 and Control group – (53.4 ± 9.58)] and Serum Nesfatin-1.

Table 5: **Evaluation of Endocrine Profile in relation to serum nesfatin-1.**

	CONTROL (N=50)	PCOS (N=50)
Free testosterone (ng/dl)	1.232 ± 0.75	1.46 ± 0.43
S.Insulin (Uu/ml)	8.484 ± 6.45	9.712 ± 7.56
FSH (mIU/ml)	6.312 ± 3.00	7.32 ± 2.92
LH (mIU/ml)	5.05 ± 3.00	6.58 ± 3.81
Nesfatin-1 (ng/ml)	1.98 ± 1.00	6.03 ± 1.58

Table 5 shows that FSH (7.32 ± 2.92), LH (6.58 ± 3.81), Serum Insulin (9.712 ± 7.56), and nesfatin-1 (9.712 ± 7.56) had a positive correlation in the PCOS group.

Table 6: Evaluation of fasting blood glucose in relation to serum nesfatin-1

	CONTROL (N=50)	PCOS (N=50)
FBS (mg/dl)	92.28±5.41	94.58±5.68
Nesfatin-1 (ng/ml)	1.98 ± 1.00	6.03 ± 1.58

Mean fasting blood sugar level was slightly higher in the PCOS group (94.58±5.68) as compared to the control group (92.28±5.41).

Discussion

Polycystic ovarian syndrome is a heterogeneous endocrine disorder characterized by menstrual irregularity, hyperandrogenism, and chronic anovulation. It commonly affects women of reproductive age and is frequently associated with metabolic abnormalities, including insulin resistance and obesity. In the present cross-sectional case-control study, serum nesfatin-1 levels were found to be significantly higher in women with PCOS compared to healthy controls, suggesting a potential role of this peptide in the pathophysiology of the syndrome.

The majority of women in both the PCOS and control groups belonged to the 20–30-year age group, which is consistent with the typical reproductive age distribution reported in earlier studies.^{12,13} Increased prevalence of PCOS in younger women emphasizes the need for early metabolic screening and hormonal evaluation to prevent long-term complications.

In the present study, a higher proportion of PCOS women demonstrated increased body mass index compared to controls. Obesity and overweight are well-recognized contributors to insulin resistance and hyperandrogenism in PCOS. Previous studies have similarly reported a strong association between increased BMI and PCOS prevalence, particularly in urban populations with sedentary lifestyles.^{15,16,20} Lifestyle-related factors, including reduced physical activity and unhealthy dietary habits, are considered major contributors to rising PCOS incidence.

Serum nesfatin-1 levels in PCOS women were markedly elevated compared to controls. This finding is consistent with earlier research demonstrating altered nesfatin-1 concentrations in PCOS patients and their association with metabolic dysfunction.^{12–14} However, a few studies have reported lower nesfatin-1 levels in affected women, suggesting population-specific metabolic variations and genetic influences.¹⁰ These discrepancies may reflect differences in insulin sensitivity, obesity prevalence, and ethnic background among study populations.

The present study further demonstrated a positive correlation between nesfatin-1 levels and triglycerides as well as LDL cholesterol, while a negative correlation was

observed with HDL cholesterol. These findings suggest a link between nesfatin-1 and dyslipidemia in PCOS. Similar metabolic associations have been documented previously, supporting the role of nesfatin-1 in energy regulation and lipid metabolism.^{13,14} Nesfatin-1 is known to influence appetite control and glucose homeostasis, potentially contributing to altered metabolic profiles in PCOS patients.

Hormonal analysis revealed positive correlations between serum nesfatin-1 and gonadotropins, including FSH and LH, as well as serum insulin levels. These findings reinforce the concept that nesfatin-1 may interact with both endocrine and metabolic pathways involved in PCOS pathogenesis. Previous investigations have similarly highlighted its association with insulin resistance and reproductive hormone imbalance.^{12,14} Insulin resistance remains a central feature of PCOS, contributing to hyperandrogenism and ovulatory dysfunction.

Fasting blood glucose levels were slightly higher in PCOS women compared to controls, though within near-normal ranges. This may reflect compensatory hyperinsulinemia maintaining glucose homeostasis in early metabolic dysfunction. Long-term persistence of insulin resistance may predispose these women to type 2 diabetes mellitus, as demonstrated in earlier epidemiological studies.^{18,19} Cardiovascular parameters, including blood pressure and heart rate, were comparable between the two groups in the present study. However, previous research has indicated increased cardiovascular risk among PCOS patients, particularly in those with obesity and insulin resistance.¹⁴ This highlights the importance of early metabolic monitoring in affected women.

Overall, the elevated serum nesfatin-1 levels observed in PCOS patients, irrespective of other metabolic and hormonal parameters, suggest its potential role as a biomarker for metabolic dysregulation in PCOS. The findings support the hypothesis that nesfatin-1 participates in glucose metabolism, lipid homeostasis, and endocrine regulation, thereby contributing to the complex pathophysiology of the syndrome.

Generalizability

The findings may be applicable to similar tertiary care settings managing reproductive-age women with PCOS, particularly in developing countries.



Limitations

Small sample size and single-center design limit broader generalization.

Recommendations

Nesfatin-1 may be incorporated as a biomarker in the metabolic assessment of PCOS after validation in larger studies.

Conclusion

The present study demonstrates that serum Nesfatin 1 levels are significantly increased in women with Polycystic Ovarian Syndrome (PCOS) when compared with non-PCOS controls, highlighting its potential role in the pathophysiology of the disorder.

In addition, significant correlations were observed between serum nesfatin 1 and metabolic as well as hormonal parameters.

Overall, altered Nesfatin 1 levels may contribute to both the endocrine and metabolic disturbances seen in PCOS. Serum Nesfatin 1 could therefore be considered as a potential biomarker for metabolic risk assessment in PCOS women. However, further large-scale and longitudinal studies are required to clarify its exact role and to explore its potential as a therapeutic target.

Acknowledgement

The authors acknowledge the departments involved for technical and clinical support.

Abbreviations

PCOS – Polycystic ovarian syndrome

BMI – Body mass index

FSH – Follicle-stimulating hormone

LH – Luteinizing hormone

ELISA – Enzyme-linked immunosorbent assay

Funding

No external funding was received.

Conflict of interest

The authors declare no conflict of interest.

Data availability

Data are available from the corresponding author upon reasonable request.

Author contributions

All authors contributed to the study design, data collection, analysis, and manuscript preparation.

Author biography

All authors are faculty members or postgraduate trainees involved in clinical pathology and gynecology research.

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Student's Journal of Health Research Africa

e-ISSN: 2709-9997, p-ISSN: 3006-1059

Vol.6 No. 12 (2025): December 2025 Issue

<https://doi.org/10.51168/sjhrafrica.v6i12.2347>

Original Article

PUBLISHER DETAILS

Student's Journal of Health Research (SJHR)

(ISSN 2709-9997) Online

(ISSN 3006-1059) Print

Category: Non-Governmental & Non-profit Organization

Email: studentsjournal2020@gmail.com

WhatsApp: +256 775 434 261

Location: Scholar's Summit Nakigalala, P. O. Box 701432,
Entebbe Uganda, East Africa

