International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Ahmed N et al. Int J Reprod Contracept Obstet Gynecol. 2026 Jan;15(1):44-49
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20254258

Original Research Article

Association of ovarian volume with insulin resistance in PCOS patients

Naorin Ahmed[1]*, Dilruba Akhter[2], Nusrat Jahan Eva[3], Ahsan Habib Khan[4], M. Karimatun Nesa[5], M. Irin Nahar[6], Chowdhury Afsana Haider[4], Halima Jahan Ripa[7]

[1]Upazila Health Complex, Kaliakoir, Gazipur, Bangladesh
[2]Department of Obstetrics and Gynecology, Institute of Child and Mother Health (ICMH), Matuail, Dhaka, Bangladesh
[3]Department of Obstetrics and Gynecology, Government Unani and Ayurvedic Medical College, Dhaka, Bangladesh
[4]Medical Officer, Directorate General of Health Services (DGHS), Dhaka, Bangladesh
[5]Medical Officer, Upazila Health Complex, Nachole, Chapainawabganj, Bangladesh
[6]Department of Obstetrics and Gynaecology, Rajshahi Medical College Hospital, Rajshahi, Bangladesh
[7]Assistant Registrar, 250 Bedded General Hospital, Jamalpur, Bangladesh

Received: 11 November 2025
Accepted: 06 December 2025

***Correspondence:**
Dr. Naorin Ahmed,
E-mail: naorindishary@gmail.com

ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder in women, characterized by hyperandrogenism, menstrual irregularities, polycystic ovarian morphology, and often associated with insulin resistance. The purpose of this study was to assess the association between insulin resistance and ovarian volume in PCOS patients, comparing women with ovarian volume >10 cc to those with ≤10 cc. The aim of the study was to evaluate the association between ovarian volume and insulin resistance among patients diagnosed with polycystic ovary syndrome (PCOS).

Methods: This comparative cross-sectional study at the department of obstetrics and gynecology, ICMH, Dhaka (October 2022-September 2023) included 66 women with PCOS (18-35 years), divided by ovarian volume into Group A (>10 cc, n=34) and Group B (≤10 cc, n=32). Fasting glucose, insulin, HOMA-IR, hirsutism, BMI, and waist-to-hip ratio were assessed, analyzed with SPSS v27 ($p<0.05$).

Results: In 66 PCOS patients, groups were similar in age, BMI, waist-to-hip ratio, menarche, parity, and Ferriman-Gallwey score (all $p>0.05$); HOMA-IR was higher in group A (4.83 versus 3.59; $p=0.012$), correlated with ovarian volume ($r=0.685$, $p<0.001$), and HOMA-IR ≥3.8 increased odds of ovarian volume >10 cc (67.6% versus 21.9%; OR=7.468; 95% CI=2.476-22.522; $p<0.001$).

Conclusions: Higher insulin resistance (HOMA-IR ≥3.8) is significantly associated with larger ovarian volume (>10 cc) in PCOS patients, suggesting ovarian volume as a potential indicator of insulin resistance.

Keywords: Insulin resistance, Ovarian volume, Polycystic ovary syndrome

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder impacting both reproductive and metabolic health in women.[1] The prevalence of PCOS globally ranges from 8% to 13%, yet up to 70% of affected women remain untreated.[2] In the United States, prevalence is estimated between 6% and 12%, while in India it varies from 3.7% to 22.5% depending on the population studied and diagnostic criteria used.[3,4] In Bangladesh, PCOS was reported in 6.11% of gynecology out-patient visits and 35.39% among infertile women.[5] These geographical differences may be influenced by variations in diagnostic criteria, sample heterogeneity, socioeconomic status, healthcare access, risk factors, and awareness.[6]

PCOS is characterized by hyperandrogenism (clinical or biochemical), menstrual irregularities such as oligomenorrhea or anovulation, and polycystic ovarian morphology.[7] Persistent hyperandrogenism is associated with impaired hypothalamic-pituitary feedback, luteinizing hormone (LH) hypersecretion, premature granulosa cell luteinization, and abnormal follicular development.[8] Women with PCOS may also experience psychological issues such as anxiety, depression, disturbed body image, and reduced self-esteem. With age, PCOS shifts from primarily a reproductive disorder to a metabolic one, including insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, dyslipidemia, and cardiovascular risk factors.[9]

The pathogenesis of PCOS involves a complex interplay of genetic, hormonal, metabolic, and environmental factors. Disturbances in pulsatile GnRH release lead to LH hypersecretion, contributing to ovarian dysfunction and hyperandrogenism. Follicles may be resistant to FSH, possibly due to elevated intra-ovarian anti-Müllerian hormone (AMH). Genetic and epigenetic variants, including CYP11A1, CYP17A1, CYP19A1 polymorphisms and susceptibility loci such as THADA, DENND1A, LHCGR, FSHR, C9orf3, YAP1, GATA4, NEIL2, and ERBB4, further contribute to PCOS susceptibility.[10-12]

Insulin resistance is a key metabolic feature of PCOS and contributes to hyperandrogenism through synergistic action with LH on ovarian steroidogenesis.[13,14] Women with oligomenorrhea are particularly prone to insulin resistance. Insulin resistance can be assessed using the homeostatic model assessment for insulin resistance (HOMA-IR) or intravenous glucose tolerance testing.[13] Increased insulin levels amplify LH receptor expression, accelerate early follicular growth, and disrupt dominant follicle selection, contributing to follicular arrest.[15]

Ovarian morphology, including increased antral follicle count (AFC) and ovarian volume, is a cardinal feature of PCOS.[16] Under Rotterdam criteria, polycystic ovary morphology (PCOM) is defined as ≥12 follicles and/or ovarian volume >10 cc, while the 2014 AE-PCOS Society task force recommends ≥25 follicles and/or volume >10 cc.[17]

Understanding the association between insulin resistance and ovarian volume is clinically important, as increased ovarian volume correlates with hyperandrogenemia and may reflect metabolic dysfunction.[18,19] However, some studies have found no association between ovarian volume and insulin levels.[20]

Therefore, this study aimed to assess the association between insulin resistance and ovarian volume in PCOS patients, comparing women with ovarian volume >10 cc to those with ≤10 cc, with the goal of providing insights into PCOS pathophysiology and improving diagnostic and therapeutic approaches.

Objective

To evaluate the association between ovarian volume and insulin resistance among patients diagnosed with polycystic ovary syndrome (PCOS).

METHODS

This comparative, cross-sectional study was conducted at the department of obstetrics and gynecology, Institute of Child and Mother Health (ICMH), Matuail, Dhaka, Bangladesh, from October 2022 to September 2023. A total of 66 women aged 18-35 years, diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam criteria, were included. Participants were selected based on predefined inclusion and exclusion criteria to evaluate the association between insulin resistance and ovarian volume in PCOS patients. Based on ovarian volume measured by transvaginal sonography (TVS), participants were divided into two groups: group A (n=34) with ovarian volume >10 cc and group B (n=32) with ovarian volume ≤10 cc.

Inclusion criteria

Women aged 18-35 years with PCOS diagnosed according to the Rotterdam criteria. Willingness to provide written informed consent. Group A: ovarian volume ≥10 cc; Group B: ovarian volume <10 cc.

Exclusion criteria

Thyroid disorders, hyperprolactinemia, Cushing syndrome, congenital adrenal hyperplasia. Diabetes mellitus or other systemic diseases (e.g., chronic kidney disease, liver disease). Use of medications affecting insulin resistance or ovarian function within the last six months, including oral contraceptives, glucocorticoids, metformin, ovulation induction agents, anti-androgens, or lipid-lowering/anti-obesity drugs.

Study variables

The dependent variable was ovarian volume, and independent variables included fasting plasma glucose, fasting insulin, and insulin resistance assessed by HOMA-IR. Hirsutism was evaluated using the Modified Ferriman-Gallwey score, while BMI and waist-to-hip ratio were measured according to standard protocols.

Data collection and measurements

After an 8-hour fast, 6 ml of venous blood was drawn under aseptic conditions to measure plasma glucose and insulin levels at the biochemistry and molecular biology department, BSMMU. Transvaginal sonography was used to assess ovarian volume, and participants underwent structured interviews and clinical examinations to collect demographic, clinical, and obstetric information.

Statistical analysis

Data were analyzed using SPSS version 27. Descriptive statistics were presented as mean±SD, frequencies, and percentages. Group comparisons were performed using unpaired t-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables. P

earson's correlation was used to assess the relationship between HOMA-IR and ovarian volume, and odds ratios (OR) with 95% confidence intervals (CI) were calculated for categorical associations. A p value <0.05 was considered statistically significant.

Ethical considerations

Ethical approval was obtained from the institutional review board of ICMH, and written informed consent was obtained from all participants. Confidentiality was maintained using unique ID numbers, and participants were assured of minimal physical, psychological, or social risk, with the freedom to withdraw at any time.

RESULTS

Table 1 shows that no significant differences were observed in the socio-demographic characteristics between the two groups (p>0.05).

Table 1: Distribution of respondents according to socio-demographic characteristics by group (n=66).

Socio-demographic characteristics		Group A (n=34) N (%)	Group B (n=32) N (%)	P value
Age (years)	18-23	15 (44.1)	16 (50.0)	0.173[a]
	24-29	17 (50.0)	10 (31.3)	
	30-35	2 (5.9)	6 (18.8)	
	Mean±SD	24.47±3.13	25.66±4.36	0.207[b]
Education	Up to primary	4 (11.8)	3 (9.4)	0.530[a]
	SSC/equivalent	16 (47.1)	11 (34.4)	
	Secondary and above	14 (41.2)	18 (56.3)	
Occupation	Homemaker	32 (94.1)	25 (78.1)	0.136[a]
	Student	0 (0.0)	2 (6.3)	
	Service holder	2 (5.9)	5 (15.6)	
Monthly income (Tk.)	≤15,000	2 (5.9)	7 (21.9)	0.112[a]
	15,001-30,000	24 (70.6)	21 (65.6)	
	>30,000	8 (23.5)	4 (12.5)	

Table 2: Distribution of patients according to anthropometric measurements by group (n=66).

Parameters		Group A (n=34) N (%)	Group B (n=32) N (%)	P value
BMI (kg/m^2)	Normal (18.5-24.9)	7 (20.6)	13 (40.6)	0.163[c]
	Overweight (25.0-29.9)	18 (52.9)	15 (46.9)	
	Obese (≥30.0)	9 (26.5)	4 (12.5)	
	Mean±SD	27.49±3.46	26.07±3.22	0.090[b]
Waist-to-hip ratio	Mean±SD	0.91±0.08	0.89±0.09	0.223[b]

Table 3: Distribution of patients according to obstetric characteristics by group (n=66).

Parameters		Group A (n=34) N (%)	Group B (n=32) N (%)	P value
Age at menarche (years)	Mean±SD	12.35±0.89	12.23±0.68	0.548[b]
Parity	Nulliparous	29 (85.3)	23 (71.9)	0.183[c]
	Parous	5 (14.7)	9 (28.1)	

Table 2 presents the distribution of patients based on anthropometric measurements. The mean BMI was slightly higher in group A (27.5±3.5 kg/m^2) compared with group B (26.1±3.2 kg/m^2), although the difference was not statistically significant (p>0.05). Similarly, no significant difference was found between the groups regarding waist-to-hip ratio (p=0.223).

Table 3 shows the distribution of patients according to obstetric characteristics. The mean age at menarche was similar between the two groups (p=0.548). A greater proportion of participants in group A were nulliparous (85.3%) compared with group B (71.9%), though the difference was not statistically significant (p=0.183).

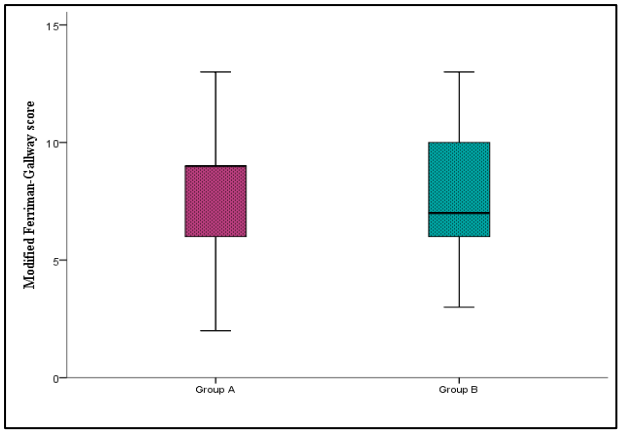

Figure 1: Box and whisker plot showing the distribution of respondents according to modified Ferriman-Gallwey Scores.

Figure 1 illustrates the distribution of modified Ferriman-Gallwey scores among the respondents. The mean±SD score was slightly higher in group A compared with group B (8.00±2.86 versus 7.72±2.92), but this difference was not statistically significant (p>0.05).

Table 4: Comparison of respondents according to insulin resistance index (HOMA-IR) by group (n=66).

Insulin resistance index	Group A (n=34) (Mean±SD)	Group B (n=32) (Mean±SD)	P value
HOMA-IR	4.83±2.21	3.59±1.67	0.012[b]

Table 4 compares the insulin resistance index between the two groups. The mean HOMA-IR was significantly higher in group A (4.83±2.21) compared with group B (3.59±1.67) (p=0.012).

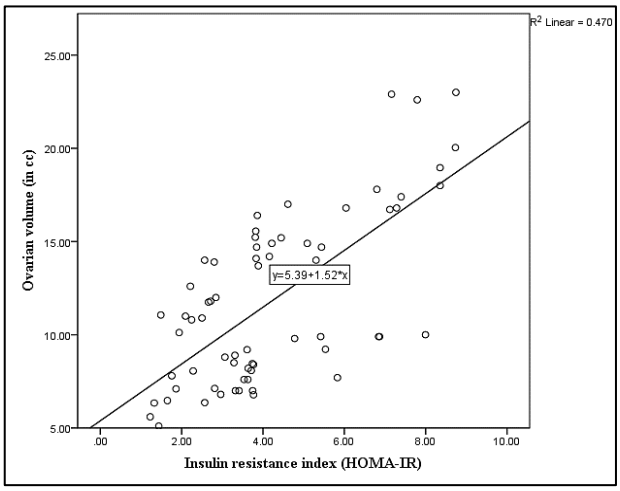

Figure 2: Scatterplot showing the correlation between HOMA-IR levels and ovarian volume.

Figure 2 demonstrates a moderate positive correlation between the insulin resistance index (HOMA-IR) and ovarian volume in PCOS patients (r=0.685, p<0.001). The coefficient of determination (R^2=0.470) indicates that approximately 47% of the variance in ovarian volume can be explained by variations in HOMA-IR levels.

A higher level of insulin resistance was observed in group A (67.6%) compared with group B (21.9%), a difference that was statistically significant (p=0.001). PCOS patients with high insulin resistance (HOMA-IR≥3.8) had 7.5 times higher odds of having an ovarian volume >10 cc (OR=7.468; 95% CI =2.476-22.522).

Table 5: Odds ratios (OR) and 95% confidence intervals (CI) for ovarian volume >10 cc according to insulin resistance in PCOS patients (group A =34, group B =32).

HOMA-IR	Group A (n=34) N (%)	Group B (n=32) N (%)	P value	OR (95% CI)
≥3.8	23 (67.6)	7 (21.9)	<0.001[c]	7.468 (2.476-22.522)
<3.8	11 (32.4)	25 (78.1)		

DISCUSSION

Polycystic ovary syndrome (PCOS) is characterized by a combination of chronic anovulation or oligomenorrhea, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology observed on ultrasound. Furthermore, in PCOS, ovarian volume is often increased, typically exceeding 10 cc, as seen through ultrasound imaging. Therefore, this cross-sectional comparative study was conducted to evaluate the association between insulin resistance and ovarian volume in patients with PCOS.

In this study, the mean age of women with PCOS having ovarian volume >10 cc was slightly lower (24.47±3.13 years) than that of women with ovarian volume ≤10 cc (25.66±4.36 years), though the difference was not statistically significant (p>0.05). According to Bhat et al, the mean age of the study group was 28.5±6.15 years and that of the control group was 29.2±5.54 years, which were comparable.[21]

The present study revealed that BMI was slightly higher in group A (27.5±3.5 kg/m^2) than in group B (26.1±3.2 kg/m^2), though the difference did not reach statistical significance (p>0.05). Similarly, there was no statistically significant difference in the waist-to-hip ratio between the two groups (0.91±0.08 in group A versus 0.89±0.09 in group B; p=0.223). When assessing the Modified

Ferriman-Gallwey score, a measure of hirsutism, the mean score in group A was slightly higher than that in group B (8.00±2.86 versus 7.72±2.92), but again, this difference was not statistically significant (p>0.05). Gencer et al reported similar findings, observing that PCOS women with insulin resistance had a significantly higher mean BMI (27.42±5.56) than those without insulin resistance (24.01±3.71) (p<0.05).[22]

In this study, women with larger ovaries had an age at menarche nearly identical to that of women with smaller ovaries (12.35±0.89 versus 12.23±0.68 years; p=0.548). The proportion of nulliparous women was higher in group A compared to group B (85.3% versus 71.9%; p=0.183). Roos et al similarly found that women with PCOS were more likely to be nulliparous compared to women without PCOS (53.0% versus 43.8%; p<0.001), consistent with the present findings.[23]

In the current study, PCOS patients with larger ovarian volume (group A) exhibited significantly higher insulin resistance (HOMA-IR) compared to those with smaller ovarian volume (4.83±2.21 versus 3.59±1.67; p=0.012). A moderate positive correlation was observed between HOMA-IR and ovarian volume in PCOS patients, indicating that as insulin resistance increased, ovarian volume also tended to increase (r=0.685, p<0.001). Using a cut-off value of 3.8 for high insulin resistance, participants with HOMA-IR≥3.8 had 7.5 times higher odds of having an ovarian volume >10 cc (OR=7.468; 95% CI =2.476-22.522). These findings underscore a strong association between elevated insulin resistance and increased ovarian volume in PCOS.

Gencer et al similarly demonstrated that insulin resistance was associated with fasting insulin, HOMA index, BMI, and ovarian volume in PCOS patients.[22] Hong et al also documented that women with PCOS had significantly higher 2-hour post-load glucose, fasting and post-load insulin levels, ovarian volume, and follicle number, along with lower insulin sensitivity, compared with controls (p<0.01).[24] In a cross-sectional study, Reid et al found that the odds ratio for elevated HOMA-IR among patients with a maximum ovarian volume >10 cc was 1.9 compared to those with ≤10 cc (95% CI 1.0-3.4).[25] Follicle number, however, was not significantly associated with any metabolic parameter. Neoklis et al also observed that indices of insulin resistance, including fasting insulin, fasting glucose/insulin ratio, QUICKI, and HOMA-IR, were linked to phenotype I in overweight or obese PCOS women.[25] This highlights the critical role of insulin resistance in the pathophysiology of PCOS, particularly in overweight or obese individuals.

Overall, the present findings suggest that higher insulin resistance is associated with larger ovarian volume, emphasizing the importance of assessing insulin resistance in the management of PCOS, particularly in patients with enlarged ovaries.

This study had several limitations. The study was conducted in a single tertiary care hospital; therefore, the results may not accurately reflect the status of the general population. The sample size was limited and could not be increased due to financial constraints. The Rotterdam FN criteria were used for diagnosing PCOS patients, which may be overly inclusive given current ultrasound technology and the newer guidelines recommending higher FN cut-off values.

Therefore, the findings of this study cannot be generalized to the entire population.

CONCLUSION

The findings of this study suggest a significant association between insulin resistance and ovarian volume, with higher insulin resistance (HOMA-IR≥3.8) being linked to larger ovarian volume (>10 cc) in PCOS patients. Measurement of ovarian volume may provide valuable insights into the risk of systemic hyperinsulinemia and could serve as a practical indicator for risk stratification and counselling in individuals with PCOS.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Choudhury AA, Rajeswari VD. Polycystic ovary syndrome (PCOS) increases the risk of subsequent gestational diabetes mellitus (GDM): a novel therapeutic perspective. Life Sci. 2022;310:121069.
2. World Health Organization. Polycystic ovary syndrome. Geneva: WHO; 2023. Available from: https://www.who.int/news-room/fact-sheets/detail/polycystic-ovary-syndrome. Accessed on 3 June 2025.
3. Rao M, Broughton KS, LeMieux MJ. Cross-sectional study on the knowledge and prevalence of PCOS at a multiethnic university. Progress Prevent Med. 2020;5(2):e0028.
4. Ganie MA, Rashid A, Sahu D, Nisar S, Wani IA, Khan J. Prevalence of polycystic ovary syndrome (PCOS) among reproductive age women from Kashmir valley: a cross-sectional study. Int J Gynecol Obstet. 2020;149(2):231-6.
5. Fatema K, Das TR, Kazal RK, Mahamood S, Pervin HH, Noor F, et al. Prevalence and characteristics of polycystic ovarian syndrome in women attending in outpatient department of obstetrics and gynecology of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. Int J Reprod Contracept Obstet Gynecol. 2021;10(3):830-6.
6. Bozdag G, Mumusoglu S, Zengin D, Karabulut E, Yildiz BO. The prevalence and phenotypic features of polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod. 2016;31(12):2841-55.

7. Meier RK. Polycystic ovary syndrome. Nurs Clin North Am. 2018;53(3):407-20.
8. Palomba S, Daolio J, La Sala GB. Oocyte competence in women with polycystic ovary syndrome. Trends Endocrinol Metab. 2017;28(3):186-98.
9. Louwers YV, Laven JS. Characteristics of polycystic ovary syndrome throughout life. Ther Adv Reprod Health. 2020;14:2633494120911038.
10. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, et al. Polycystic ovary syndrome. Nat Rev Dis Prim. 2016;2(1):1-8.
11. Heidarzadehpilehrood R, Pirhoushiaran M, Abdollahzadeh R, Binti Osman M, Sakinah M, Nordin N, et al. A review on CYP11A1, CYP17A1, and CYP19A1 polymorphism studies: candidate susceptibility genes for polycystic ovary syndrome (PCOS) and infertility. Genes. 2022;13(2):302.
12. Hayes MG, Urbanek M, Ehrmann DA, Armstrong LL, Lee JY, Sisk R, et al. Genome-wide association of polycystic ovary syndrome implicates alterations in gonadotropin secretion in European ancestry populations. Nat Commun. 2015;6(1):7502.
13. Dumesic DA, Padmanabhan V, Chazenbalk GD, Abbott DH. Polycystic ovary syndrome as a plausible evolutionary outcome of metabolic adaptation. Reproductive Biol Endocrinol. 2022;20(1):12.
14. Galluzzo A, Amato MC, Giordano C. Insulin resistance and polycystic ovary syndrome. Nutr Metab Cardiovasc Dis. 2008;18(7):511-8.
15. Diamanti-Kandarakis E. Polycystic ovarian syndrome: pathophysiology, molecular aspects and clinical implications. Exp Rev Mol Med. 2008;10:e3.
16. Chun S. Serum luteinizing hormone level and luteinizing hormone/follicle-stimulating hormone ratio but not serum anti-Müllerian hormone level is related to ovarian volume in Korean women with polycystic ovary syndrome. Clin Exp Reprod Med. 2014;41(2):86.
17. Dewailly D, Lujan ME, Carmina E, Cedars MI, Laven J, Norman RJ, et al. Definition and significance of polycystic ovarian morphology: a task force report from the Androgen Excess and Polycystic Ovary Syndrome Society. Hum Reprod Update. 2014;20(3):334-52.
18. Mansour A, Hashemi Taheri AP, Moradi B, Mohajeri-Tehrani MR, Qorbani M, Ghorbani Pashakolaee S, et al. Ovarian volume, not follicle count, is independently associated with androgens in patients with polycystic ovary syndrome. BMC Endocr Disord. 2022;22(1):298.
19. Villa P, Rossodivita A, Sagnella F, Moruzzi MC, Mariano N, Lassandro AP, et al. Ovarian volume and gluco-insulinaemic markers in the diagnosis of PCOS during adolescence. Clin Endocrinol. 2013;78(2):285-90.
20. Guastella E, Longo RA, Carmina E. Clinical and endocrine characteristics of the main polycystic ovary syndrome phenotypes. Fertil Steril. 2010;94(6):2197-201.
21. Bhat K, Bhandari M, Garg V. Prostate specific antigen: a diagnostic marker in polycystic ovarian syndrome. J Clin Diagn Res. 2019;13(1).
22. Gencer G, Serin AN, Gencer K. Comparison of insulin resistance and overian volume in patients with polycystic ovary syndrome with and without Hashimoto. Res Square. 2022.
23. Roos N, Kieler H, Sahlin L, Ekman-Ordeberg G, Falconer H, Stephansson O. Risk of adverse pregnancy outcomes in women with polycystic ovary syndrome: population based cohort study. BMJ. 2011;343.
24. Hong SH, Sung YA, Hong YS, Jeong K, Chung H, Lee H. Polycystic ovary morphology is associated with insulin resistance in women with polycystic ovary syndrome'. Clin Endocrinol. 2017;87(4):375-80.
25. Reid SP, Kao CN, Pasch L, Shinkai K, Cedars MI, Huddleston HG. Ovarian morphology is associated with insulin resistance in women with polycystic ovary syndrome: a cross sectional study. Fertil Res Pract. 2017;3(1):8.
26. Neoklis A, Anastasia K, Damianaki K, Nikolaos D, Markantes G, Papadopoulos V, et al. Polycystic ovarian morphology is associated with hyperandrogenemia and insulin resistance in women with polycystic ovary syndrome (PCOS). J Steroids Horm Sci. 2016;7(1):1-5.

Cite this article as: Ahmed N, Akhter D, Eva NJ, Khan AH, Nesa MK, Nahar MI, et al. Association of ovarian volume with insulin resistance in PCOS patients. Int J Reprod Contracept Obstet Gynecol 2026;15:44-9.