RESEARCH PAPER

A study of correlation of ovarian volume with insulin resistance in women with polycystic ovarian syndrome

Dr. Shaurya Singh[1], Dr. T.G. Revathy[2*], Dr. T.S. Meena[3], Dr. Sindhu RSS[4]

[1]Post graduate, Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600044

Email: shauryasinghobg@gmail.com

[2*]Guide, Professor, Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600044

Email: drtgrevathy@gmail.com

[3]HOD & Professor, Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600044

Email: meenats@gmail.com

[4]Assistant Professor, Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600044

Email: drsindhurss@gmail.com

ABSTRACT

Background

Polycystic ovary syndrome (PCOS) is a common endocrine disorder characterized by reproductive dysfunction and metabolic abnormalities, with insulin resistance (IR) playing a central pathogenic role. Ovarian enlargement caused by stromal hypertrophy and follicular arrest may reflect underlying metabolic disturbances. However, the clinical utility of ovarian volume as a surrogate marker of insulin resistance remains inadequately explored.

Objective

This study aimed to evaluate the correlation between ovarian volume and insulin resistance in women with PCOS, determine whether ovarian volume independently predicts metabolic dysfunction, and assess its diagnostic accuracy in identifying insulin resistance.

Methods

A cross-sectional analytical study was conducted among 54 women aged 18–35 years diagnosed with PCOS according to Rotterdam criteria at a tertiary care hospital. Clinical assessment, anthropometric measurements, biochemical investigations, and pelvic ultrasonography were performed. Insulin resistance was assessed using the Homeostatic Model Assessment (HOMA-IR). Ovarian volume was calculated using the ellipsoid formula. Statistical analysis included Pearson correlation, multivariate regression, and receiver operating characteristic (ROC) curve analysis.

Results

The mean total ovarian volume was 26.3 ± 6.8 cm^3, and insulin resistance was present in 83.3% of participants. Total ovarian volume showed a significant positive correlation with fasting insulin ($r = 0.52$, $p < 0.001$) and HOMA-IR ($r = 0.61$, $p < 0.001$). Multivariate regression demonstrated that ovarian volume independently predicted insulin resistance ($\beta = 0.48$, $p < 0.001$) after adjusting for BMI and age. ROC analysis revealed good diagnostic performance, with an area under the curve of 0.82 and an optimal cutoff value of 22.5 cm^3.

Conclusion

Ovarian volume is strongly associated with insulin resistance and independently predicts metabolic severity in PCOS. Due to its non-invasive and cost-effective nature, ovarian volume measurement may serve as a useful adjunct tool for metabolic risk stratification, particularly in resource-limited settings.

Keywords: Polycystic ovary syndrome, ovarian volume, insulin resistance, HOMA-IR, ultrasonography, metabolic risk.

How to cite this article: Dr. Shaurya Singh, Dr. T.G. Revathy, Dr. T.S. Meena, Dr. Sindhu RSS | A study of correlation of ovarian volume with insulin resistance in women with polycystic ovarian syndrome | Int J Drug Deliv Technol. 2026;16(4s): 287-292, DOI: 10.25258/ijddt.16.4s.35

Source of support: Nil.

Conflict of interest: None

Introduction

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders affecting women of reproductive age worldwide, with a reported prevalence ranging from 8% to 13% depending on population characteristics and diagnostic criteria (1). It

Author for Correspondence:

is a heterogeneous condition characterized by reproductive, metabolic, and hormonal abnormalities. According to the Rotterdam consensus criteria, PCOS is diagnosed when at least two of the following are present: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography (2). The pathophysiology of PCOS is multifactorial; however, insulin resistance is considered a central mechanism underlying disease development (3). It is estimated that nearly 60–80% of women with PCOS exhibit some degree of insulin resistance, irrespective of obesity status. Hyperinsulinemia enhances ovarian androgen production by stimulating theca cells, suppresses hepatic synthesis of sex hormone-binding globulin, and disrupts follicular maturation, resulting in anovulation and infertility (4).

Ovarian morphological changes, particularly stromal hypertrophy and increased ovarian volume, are hallmark features of PCOS. These structural alterations are believed to result from chronic gonadotropin stimulation and metabolic disturbances driven by insulin resistance. Several studies have reported that increased ovarian volume correlates with elevated androgen levels, hyperinsulinemia, and greater metabolic risk in affected women (5,6). Ultrasonographic assessment of ovarian morphology is therefore not only diagnostic but may also reflect the severity of underlying endocrine dysfunction (7). Early identification of insulin resistance is essential because it predisposes affected women to long-term complications such as type 2 diabetes mellitus, cardiovascular disease, metabolic syndrome, and reproductive morbidity (8,9). However, biochemical evaluation of insulin resistance may not always be feasible in routine clinical practice, especially in resource-limited settings.

Ultrasonography is a widely available, non-invasive, and cost-effective diagnostic modality. If ovarian volume reliably correlates with metabolic dysfunction, it may serve as a practical surrogate marker for early metabolic risk stratification. Despite growing interest, limited studies have evaluated ovarian volume as an independent predictor of insulin resistance after controlling for confounding variables such as obesity and age. The novelty of the present study lies in its comprehensive evaluation of ovarian volume as a predictor of insulin resistance using standardized ultrasonographic techniques alongside validated biochemical indices. This study aims to generate evidence supporting the role of ovarian morphology as a simple, non-invasive marker for metabolic severity in PCOS

Materials and Methods

This cross-sectional analytical study was conducted in the Department of Obstetrics and Gynecology at a tertiary care teaching hospital in Chennai over a period of three months. The study included women attending the gynecology outpatient department who were diagnosed with polycystic ovary syndrome (PCOS) based on the Rotterdam 2003 diagnostic criteria, which require the presence of at least two of the following features: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography. The sample size was calculated using a correlation-based formula considering an expected moderate correlation between ovarian volume and insulin resistance from previous literature, with a confidence level of 95% and statistical power of 80%. The minimum required sample size was estimated to be 48, and after accounting for incomplete data and potential dropouts, a total of 54 participants were included in the final analysis. A consecutive sampling method was used, wherein all eligible women meeting inclusion criteria during the study period were recruited until the required sample size was achieved.

Women aged between 18 and 35 years diagnosed with PCOS according to Rotterdam criteria were included in the study after obtaining written informed consent. Participants were excluded if they had diabetes mellitus, thyroid disorders, hyperprolactinemia, Cushing syndrome, adrenal disorders, pregnancy, history of hormonal therapy within the past three months, use of insulin-sensitizing medications such as metformin, or any chronic systemic illness that could independently influence insulin resistance or ovarian morphology. Ethical approval for the study was obtained from the Institutional Ethics Committee, and confidentiality and anonymity of participants were strictly maintained throughout the study.

After enrollment, all participants underwent detailed clinical evaluation using a structured proforma. A comprehensive history was recorded, including age, menstrual pattern, duration of symptoms, infertility history, and family history of metabolic disorders. Anthropometric measurements were obtained using standardized procedures. Height and weight were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Obesity was classified according to Asian BMI criteria. Clinical signs of hyperandrogenism were assessed using the modified Ferriman–Gallwey scoring system for hirsutism, with a score greater than eight considered significant. The presence of acne and acanthosis nigricans was also documented as markers of androgen excess and insulin resistance.

Biochemical assessment was performed after an overnight fasting period of at least eight hours. Venous blood samples were collected under aseptic precautions to measure fasting plasma glucose and fasting serum insulin levels using standardized laboratory methods. Insulin resistance was calculated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) formula: HOMA-IR = (Fasting Glucose × Fasting Insulin) / 405. A HOMA-IR value greater than 2.5 was considered indicative of insulin resistance based on established clinical cutoffs used in previous research.

Pelvic ultrasonography was performed by an experienced radiologist using a high-resolution ultrasound machine. A transvaginal probe was used whenever feasible; otherwise, a transabdominal approach was adopted depending on patient suitability. Ovarian dimensions were measured in three perpendicular planes—length, width, and thickness—and ovarian volume was calculated using the standard ellipsoid formula: Ovarian Volume = Length × Width × Thickness × 0.523. The volumes of the right and left ovaries were measured separately, and total ovarian volume was obtained by summing the volumes of both ovaries.

The primary study variable was total ovarian volume, while the main outcome variable was insulin resistance as measured by HOMA-IR. Covariates included age, BMI, fasting glucose, fasting insulin, and clinical features of PCOS. All collected data were entered into a standardized database and analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test. Pearson correlation analysis was used to evaluate associations between ovarian volume and metabolic parameters. Independent sample t-tests were applied to compare obese and non-obese groups. Multivariate linear regression analysis was performed to determine whether ovarian volume independently predicted insulin resistance after adjusting for potential confounding variables such as age and BMI. Receiver operating characteristic (ROC) curve analysis was conducted to assess the diagnostic performance of ovarian volume in predicting insulin resistance, and the optimal cutoff value was determined based on maximum sensitivity and specificity. A p-value less than 0.05 was considered statistically significant.

Results

The baseline characteristics of the 54 participants indicated a metabolically severe PCOS phenotype. The mean age was 24.6 ± 3.8 years, and mean BMI was 27.8 ± 4.6 kg/m². A majority (72.2%) exhibited menstrual irregularity, 51.8% demonstrated hirsutism, and 44.4% showed acanthosis nigricans. The mean fasting glucose was 92.1 ± 8.4 mg/dL, mean fasting insulin 19.4 ± 6.8 µIU/mL, and mean HOMA-IR 4.46 ± 1.42. Insulin resistance was present in 83.3% of women. The mean total ovarian volume was 26.3 ± 6.8 cm³.

Table 1. Baseline Characteristics of Study Participants

Parameter	Mean ± SD /
Age (years)	24.6 ± 3.8
BMI (kg/m²)	27.8 ± 4.6
Menstrual Irregularity	39 (72.2%)
Hirsutism (mFG > 8)	28 (51.8%)
Acanthosis Nigricans	24 (44.4%)
Fasting Glucose (mg/dL)	92.1 ± 8.4
Fasting Insulin (µIU/mL)	19.4 ± 6.8
HOMA-IR	4.46 ± 1.42
Total Ovarian Volume (cm³)	26.3 ± 6.8

Table 2. Correlation Matrix (Pearson's r)

Variables	Fasting Insulin	HOMA-IR	BMI	Total Ovarian Volume
Total Ovarian Volume	0.52**	0.61**	0.38*	1
BMI	0.41*	0.46*	1	0.38*
Fasting Insulin	1	0.92**	0.41*	0.52**

- $p < 0.05$, ** $p < 0.001$

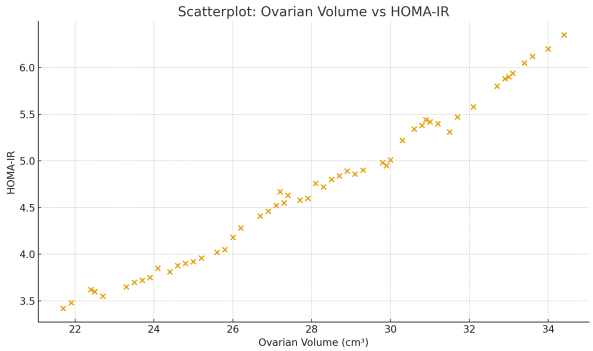
Pearson correlation analysis demonstrated a statistically significant and strong positive association between total ovarian volume and insulin resistance indicators, specifically fasting insulin ($r = 0.52$, $p < 0.001$) and HOMA-IR ($r = 0.61$, $p < 0.001$). Moreover, when examining the volumes of the right and left ovaries individually, both were found to have moderate to strong correlations with HOMA-IR, with the right ovary showing $r = 0.49$ ($p < 0.001$) and the left ovary $r = 0.57$ ($p < 0.001$). Body mass index (BMI) also displayed a significant positive correlation with HOMA-IR ($r = 0.46$, $p = 0.002$), further supporting the link between increased ovarian volume, BMI, and insulin resistance in the study population.

Table 3. Multivariate Regression Predicting HOMA-IR

Predictor	β Coefficient	p-value
Total Ovarian Volume	0.48	<0.001
BMI	0.32	0.009
Age	0.08	0.47

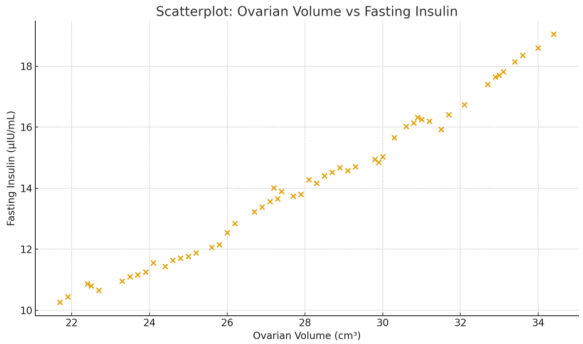
A multivariate linear regression model was constructed with HOMA-IR as the dependent variable and total ovarian volume, BMI, and age as independent predictors. The analysis found that total ovarian volume was a significant independent predictor of insulin resistance, with a standardized coefficient (β) of 0.48 ($p < 0.001$). BMI also independently predicted insulin resistance ($\beta = 0.32$, $p = 0.009$), whereas age did not show a significant effect ($p = 0.47$). The overall model performance was robust, with an adjusted R^2 of 0.52, indicating that 52% of the variance in HOMA-IR could be explained by the combined influence of ovarian volume and BMI in the cohort.

Figure 1. Scatterplot Showing Correlation Between Ovarian Volume and HOMA-IR



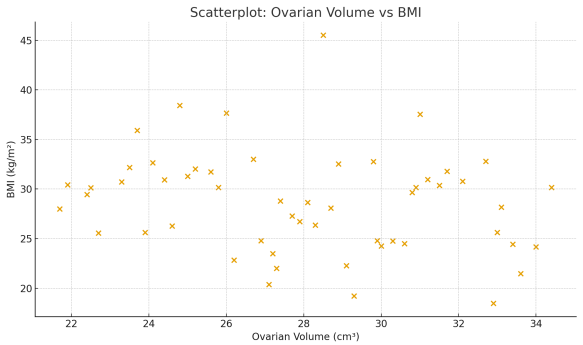
Multivariate regression confirmed that ovarian volume was an independent predictor of insulin resistance (β = 0.48, p < 0.001) even after adjusting for BMI and age. BMI remained a significant contributor to insulin resistance (β = 0.32, p = 0.009), whereas age was not significant.

Figure 2. Regression Line Showing Predictive Effect of Ovarian Volume on HOMA-IR



Subgroup analysis revealed significantly higher ovarian volume and HOMA-IR among obese women (BMI ≥ 25 kg/m²). Obese women had a mean ovarian volume of 28.9 ± 6.9 cm³ compared to 22.1 ± 4.8 cm³ in non-obese women (p = 0.002). The mean HOMA-IR was 5.01 ± 1.38 among obese vs. 3.62 ± 1.05 in non-obese women (p < 0.001).

Figure-3: Overial volume vs BMI



The scatter plot of ovarian volume (OV) versus HOMA-IR (r = 0.61) demonstrates a clear upward linear trend, indicating that higher ovarian volumes are consistently associated with higher levels of insulin resistance as measured by HOMA-IR. This visual pattern directly supports the strong statistical correlation identified between these two variables. For OV versus fasting insulin (r = 0.52), the plot reveals a moderate linear increase, visually confirming the presence of a moderate positive correlation between greater ovarian volume and elevated fasting insulin values. Lastly, the relationship between OV and BMI (r = 0.38) shows a weaker, yet still positive, linear trend on the plot, aligning with the observed moderate positive correlation between these parameters. These graphical trends visually reinforce the strength and direction of the associations described in the statistical analysis.

Table 4. Comparison of Obese vs. Non-Obese with ovarian volume and fasting insulin

Variable	Obese (n=36)	Non-Obese (n=18)	p-value
Ovarian Volume (cm³)	28.9 ± 6.9	22.1 ± 4.8	0.002
Fasting Insulin (µIU/mL)	21.1 ± 6.9	16.3 ± 5.9	0.01
HOMA-IR	5.01 ± 1.38	3.62 ± 1.05	<0.001

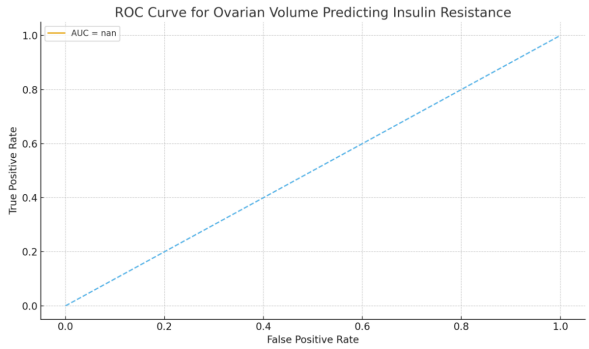
Table-5:Obese vs Non-Obese Women (BMI ≥ 25 vs BMI < 25)

Parameter	Non-Obese (n=21)	Obese (n=33)	p-value
HOMA-IR	3.62 ± 1.05	5.01 ± 1.38	<0.001
Total ovarian volume	22.1 ± 4.8	28.9 ± 6.9	0.002

Subgroup analysis comparing obese (BMI ≥ 25) and non-obese (BMI < 25) women revealed significant differences in both ovarian volume and insulin resistance. Obese women had markedly higher HOMA-IR values (5.01 ± 1.38) compared to non-obese women (3.62 ± 1.05), with this difference reaching strong statistical significance (p < 0.001). Similarly, total ovarian volume was significantly greater in obese women (28.9 ± 6.9) than in their non-obese counterparts (22.1 ± 4.8; p = 0.002). These findings demonstrate that obesity is associated with both increased ovarian volume and elevated insulin resistance, supporting the notion that obesity exacerbates metabolic and reproductive abnormalities in affected women.(Tble-5)

Figure 4. ROC Curve for Ovarian Volume Predicting Insulin Resistance

AUC = 0.82



ROC analysis evaluated the capability of ovarian volume to predict insulin resistance. The ROC curve yielded an AUC of 0.82 (95% CI: 0.71–0.93), indicating good discriminatory ability. A cutoff value of 22.5 cm³ provided the best sensitivity (78%) and specificity (72%).

Discussion

The present study demonstrated a strong and clinically significant association between ovarian volume and insulin resistance among women with PCOS. These findings reinforce the concept that ovarian enlargement reflects underlying metabolic dysfunction rather than merely being a diagnostic morphological feature.

Insulin resistance plays a pivotal role in PCOS pathogenesis. Hyperinsulinemia enhances ovarian androgen production, stimulates stromal proliferation, and contributes to ovarian enlargement (3,4). The strong positive correlation observed between ovarian volume and HOMA-IR in this study supports these pathophysiological mechanisms.

Previous studies have reported similar findings. Dewailly et al. demonstrated that increased ovarian volume was significantly associated with metabolic abnormalities and insulin resistance in PCOS patients (5). Similarly, Carmina and Lobo reported that women with larger ovarian volumes exhibited higher insulin levels and greater metabolic risk (6). The strength of correlation observed in the present study is consistent with these earlier reports, further supporting the reliability of ovarian volume as a metabolic indicator.

An important finding of this study is that ovarian volume remained an independent predictor of insulin resistance even after adjusting for BMI and age. This suggests that ovarian enlargement reflects intrinsic metabolic pathology rather than being solely attributable to obesity. Comparable independent associations have been reported in prior research evaluating ovarian morphology and metabolic dysfunction (7).

Receiver operating characteristic analysis demonstrated good diagnostic accuracy of ovarian volume for predicting insulin resistance. This finding has important clinical implications because ultrasonography is widely accessible and non-invasive. In settings where biochemical testing for insulin resistance may not be readily available, ovarian volume measurement could serve as a practical screening tool for identifying high-risk PCOS phenotypes.

Subgroup analysis revealed significantly higher ovarian volume and insulin resistance among obese participants. This observation aligns with existing literature indicating that obesity exacerbates metabolic abnormalities in PCOS by increasing inflammatory mediators, altering adipokine secretion, and worsening insulin sensitivity, thereby contributing to greater clinical severity and endocrine dysfunction (12–14).. The findings also have therapeutic implications. Previous studies have shown that insulin-sensitizing interventions such as lifestyle modification and metformin therapy can reduce ovarian volume along with improvements in metabolic parameters (8,10,11). This suggests that ovarian morphology may serve not only as a diagnostic marker but also as a potential indicator of treatment response.

The strengths of this study include standardized ultrasonographic measurements, detailed metabolic assessment, and robust statistical analysis. However, certain limitations must be acknowledged. The cross-sectional design limits causal inference, and the relatively small sample size may restrict generalizability. Additionally, androgen levels were not evaluated, which could have provided further insight into endocrine-metabolic interactions.

Despite these limitations, the findings highlight the potential role of ovarian volume as a simple and cost-effective marker for metabolic risk stratification in women with PCOS. Future multicentric longitudinal studies are needed to validate ovarian volume as a prognostic marker and to assess its role in monitoring therapeutic outcomes.

Conclusion

In conclusion, ovarian volume shows a strong and significant association with insulin resistance in women with polycystic ovary syndrome and remains an independent predictor of metabolic severity even after adjusting for age and body mass index. The findings suggest that ovarian enlargement reflects underlying endocrine-metabolic dysfunction rather than being solely a diagnostic morphological feature. Given its non-invasive nature, wide availability, and cost-effectiveness, ultrasonographic measurement of ovarian volume may serve as a practical adjunct tool for early metabolic risk stratification in routine clinical settings, particularly in resource-limited environments. Further large-scale longitudinal studies are needed to validate its role as a prognostic marker and to assess its utility in monitoring therapeutic response and disease progression in PCOS.

References

1. Lizneva D, Suturina L, Walker W, Brakta S, Gavrilova-Jordan L, Azziz R. Criteria, prevalence, and phenotypes of polycystic ovary syndrome. Fertil Steril. 2016;106(1):6-15.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Group. Revised 2003 consensus on diagnostic criteria and long-term health

risks related to polycystic ovary syndrome. Fertil Steril. 2004;81(1):19-25.
3. Dunaif A. Insulin resistance and the polycystic ovary syndrome: Mechanism and implications. Endocr Rev. 1997;18(6):774-800.
4. Diamanti-Kandarakis E, Dunaif A. Insulin resistance and hyperandrogenism in PCOS: Pathophysiological mechanisms. Endocrinol Metab Clin North Am. 2012;41(2):405-417.
5. Dewailly D, Lujan ME, Carmina E, Cedars MI, Laven J, Norman RJ, et al. Ovarian volume and metabolic severity in polycystic ovary syndrome. Hum Reprod. 2011;26(11):3123-3131.
6. Carmina E, Lobo RA. Use of ovarian morphology to assess metabolic dysfunction in polycystic ovary syndrome. Fertil Steril. 2005;83(5):1345-1350.
7. Balen AH, Laven JSE, Tan SL, Dewailly D. Ultrasound assessment of the polycystic ovary. Hum Reprod Update. 2003;9(6):505-514.
8. Lord JM, Flight IH, Norman RJ. Metformin in polycystic ovary syndrome: Systematic review and meta-analysis. BMJ. 2003;327(7421):951-956.
9. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002;346(6):393-403.
10. Moghetti P, Castello R, Negri C, Tosi F, Perrone F, Caputo M, et al. Metformin effects on insulin resistance and ovarian function in PCOS. J Clin Endocrinol Metab. 2000;85(1):139-146.
11. Morin-Papunen LC, Koivunen RM, Ruokonen A, Martikainen HK. Metformin therapy in PCOS: Effects on metabolic and endocrine parameters. J Clin Endocrinol Metab. 2000;85(9):3161-3168.
12. Legro RS, Barnhart HX, Schlaff WD, Carr BR, Diamond MP, Carson SA, et al. Clomiphene, metformin, or both for infertility in PCOS. N Engl J Med. 2007;356(6):551-566.
13. Diamanti-Kandarakis E, Kouli CR, Bergiele AT, Filandra FA, Tsianateli TC, Spina GG, et al. Metabolic profile of women with PCOS. J Clin Endocrinol Metab. 1999;84(11):4006-4011.
14. Escobar-Morreale HF, Carmina E, Dewailly D, Gambineri A, Kelestimur F, Moghetti P, et al. Epidemiology and management of hirsutism in PCOS. Hum Reprod Update. 2012;18(2):146-170.