e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2026; 17(1); 378-384

Original Research Article

Correlation Between Clinical Features and Biochemical Markers in Polycystic Ovary Syndrome-Related Infertility

Ananya Ratna Preya[1], Rashmi Prasad[2]

[1]MCh 1st year resident, Department of Reproductive medicine and surgery, Mahatma Gandhi Medical College, India

[2]Infertility specialist, Diwya Vatsalya Mamta Fertility Centre, Patna, Bihar, India

Received: 02-12-2025 / Revised: 24-12-2025 / Accepted: 30-01-2026

Corresponding Author: Dr. Ananya Ratna Preya

Conflict of interest: Nil

Abstract:

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder and a leading cause of anovulatory infertility, characterized by heterogeneous clinical, hormonal, and metabolic abnormalities.

Aim: To evaluate the clinical and biochemical characteristics of women with PCOS and assess their correlation with infertility.

Methodology: A hospital-based cross-sectional study was conducted on 80 infertile women aged 18–40 years diagnosed with PCOS using the Rotterdam criteria. Clinical features, anthropometric measurements, ultrasonographic findings, hormonal profiles, and metabolic parameters were assessed. Data were analyzed using SPSS version 27.0.

Results: The mean age was 27.8 ± 4.9 years, with a high prevalence of overweight and obesity (70%). Oligomenorrhea was the most common menstrual abnormality (55%), and primary infertility was observed in 65% of cases. Polycystic ovarian morphology was present in 72.5%. Hormonal analysis showed elevated LH/FSH ratio and increased androgen levels. Insulin resistance and dyslipidemia were common metabolic findings.

Conclusion: Infertility in PCOS results from complex interactions between ovulatory dysfunction, hyperandrogenism, and metabolic disturbances, emphasizing the need for comprehensive clinical and biochemical evaluation.

Keywords: Polycystic Ovary Syndrome, Infertility, Hyperandrogenism, Insulin Resistance, Metabolic Abnormalities.

DOI: 10.25258/Ijpqa.17.1.60

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age and represents a leading cause of anovulatory infertility worldwide [1]. It is estimated to affect approximately 6–20% of women, depending on the diagnostic criteria applied and the population studied. PCOS is characterized by a heterogeneous clinical presentation involving reproductive, metabolic, and endocrine abnormalities. The syndrome is primarily defined by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology, either singly or in combination, which collectively contribute to impaired fertility [2]. Given its high prevalence and significant impact on reproductive outcomes, PCOS has emerged as a major public health concern, particularly in societies experiencing rapid lifestyle and nutritional transitions.

Infertility associated with PCOS is predominantly attributed to ovulatory dysfunction, which manifests clinically as oligomenorrhea or amenorrhea [3]. The disrupted folliculogenesis observed in PCOS results from complex interactions between hormonal imbalance and intrinsic ovarian dysfunction [4]. Elevated luteinizing hormone (LH) secretion relative to follicle-stimulating hormone (FSH) is a hallmark endocrine feature, leading to arrested follicular development and failure of dominant follicle selection. Consequently, ovulation does not occur regularly, significantly reducing the likelihood of spontaneous conception. In addition to ovulatory disturbances, PCOS-related infertility may also be influenced by altered endometrial receptivity, chronic low-grade inflammation, and metabolic derangements that adversely affect implantation and early pregnancy maintenance.

The clinical manifestations of PCOS extend beyond infertility and menstrual irregularities, encompassing signs of hyperandrogenism such as hirsutism, acne, and androgenic alopecia [5]. These features

result from excessive androgen production by the ovaries and, to a lesser extent, the adrenal glands. Hyperandrogenism not only contributes to cosmetic concerns but also plays a central role in the pathophysiology of anovulation by disrupting normal follicular maturation. The degree of clinical expression varies widely among affected women, underscoring the heterogeneous nature of the syndrome [6]. This variability poses challenges in diagnosis and management, particularly in infertile women who may present with subtle or atypical features.

Biochemical abnormalities form the cornerstone of PCOS diagnosis and are crucial for understanding its association with infertility [7]. Elevated serum levels of androgens such as total testosterone, free testosterone, and dehydroepiandrosterone sulfate (DHEAS) are commonly observed. Alterations in gonadotropin secretion, particularly an increased LH/FSH ratio, are frequently reported, although this finding is not universal. Insulin resistance and compensatory hyperinsulinemia are central metabolic features in a significant proportion of women with PCOS, irrespective of body mass index. Hyperinsulinemia exacerbates ovarian androgen production and suppresses hepatic synthesis of sex hormone-binding globulin, thereby increasing circulating free androgens and worsening ovulatory dysfunction.

The interplay between insulin resistance and reproductive dysfunction highlight the importance of biochemical correlation in PCOS-related infertility. Metabolic abnormalities such as impaired glucose tolerance, dyslipidemia, and obesity further compound reproductive impairment [8]. Obesity, particularly central adiposity, intensifies insulin resistance and hormonal imbalance, leading to more severe clinical and biochemical manifestations of PCOS. Conversely, lean women with PCOS may exhibit predominant reproductive features with relatively mild metabolic disturbances, suggesting diverse pathogenic pathways. Understanding these biochemical variations is essential for individualized assessment and therapeutic planning in infertile women with PCOS.

Advances in diagnostic criteria, including those proposed by the National Institutes of Health, Rotterdam consensus, and Androgen Excess Society, have broadened the spectrum of PCOS phenotypes. While this has improved case identification, it has also introduced variability in reported prevalence and clinical correlations. The Rotterdam criteria, which are most widely used, emphasize the need for at least two of the three defining features, allowing for multiple phenotypic expressions. This phenotypic diversity has significant implications for fertility outcomes, as different clinical and biochemical profiles may respond variably to ovulation induction and other fertility treatments.

Given the multifactorial nature of PCOS, correlating clinical features with biochemical parameters is essential for a comprehensive understanding of its role in infertility. Such correlations aid in identifying the severity of endocrine and metabolic dysfunction, predicting reproductive outcomes, and guiding targeted interventions. An integrated evaluation of menstrual patterns, signs of hyperandrogenism, anthropometric indices, and hormonal and metabolic profiles provides a holistic framework for managing PCOS-related infertility. Therefore, exploring the clinical and biochemical correlations in PCOS not only enhances diagnostic accuracy but also contributes to optimized fertility management and improved reproductive prognosis in affected women.

Methodology

Study Design: The present study was designed as a hospital-based observational cross-sectional study aimed at evaluating the clinical and biochemical characteristics of women diagnosed with polycystic ovary syndrome (PCOS) and assessing their correlation with infertility. The study focused on identifying hormonal, metabolic, and ultrasonographic parameters associated with PCOS and their role in reproductive dysfunction.

Study Area: The study was conducted in the Department of Reproductive Medicine and Surgery, Mahatma Gandhi Medical College, India.

Study Participants: A total of 80 women diagnosed with PCOS and presenting with infertility were enrolled in the study. Participants were recruited from the outpatient department after meeting the eligibility criteria.

Inclusion Criteria: Women aged 18–40 years presenting with primary or secondary infertility and diagnosed with PCOS based on the 2003 Rotterdam criteria were included in the study. Diagnosis of PCOS was made when at least two of the following three criteria were present:

1. Oligomenorrhea or amenorrhea
2. Clinical and/or biochemical hyperandrogenism
3. Polycystic ovarian morphology on ultrasonography

Only patients who provided written informed consent were included.

Exclusion Criteria: Patients with suspected androgen-secreting tumors, hyperprolactinemia, Cushing's syndrome, congenital adrenal hyperplasia, or thyroid dysfunction were excluded. Women with diabetes mellitus, those who had received hormonal or insulin-sensitizing therapy within the last six months, and patients with other known causes of infertility were also excluded to avoid confounding factors.

Sample Size: The study included a sample size of 80 participants, selected based on feasibility and

patient availability during the study period. This sample size was considered adequate to assess clinical and biochemical correlations in PCOS-related infertility.

Procedure: After obtaining ethical clearance from the Institutional Ethics Committee, eligible participants were enrolled following informed consent. A detailed clinical history was obtained, including menstrual pattern, duration of infertility, and symptoms of hyperandrogenism. Anthropometric measurements such as height and weight were recorded, and body mass index (BMI) was calculated using the standard formula (kg/m²). Clinical assessment of hirsutism was performed using the Modified Ferriman–Gallwey (mFG) scoring system, with a score greater than 8 considered indicative of hirsutism. Blood pressure was measured after a 10-minute rest period.

Pelvic ultrasonography was performed during the early follicular phase using a 6–8 MHz transvaginal probe. Polycystic ovarian morphology was defined as the presence of 12 or more follicles measuring 2–9 mm in diameter in at least one ovary and/or an ovarian volume greater than 10 ml.

Fasting venous blood samples were collected during the follicular phase for biochemical analysis. Hormonal parameters including LH, FSH, total testosterone, free testosterone, DHEAS, SHBG, estradiol, prolactin, TSH, insulin, and 17-hydroxyprogesterone were measured using standardized immunoassay and chemiluminescence techniques. Fasting glucose and lipid profile were estimated using enzymatic and spectrophotometric methods. Insulin resistance was calculated using the HOMA-IR formula.

Statistical Analysis: The collected data were entered into Microsoft Excel and subsequently analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0. Descriptive statistical methods were employed to summarize the data, including calculation of mean, standard deviation, frequencies, and percentages. Inferential statistical analysis was performed to compare variables between study groups. The student's t-test was applied for comparison of quantitative variables with normal distribution, while the Mann–Whitney U test was used for variables that did not follow a normal distribution. Statistical significance was considered at a p-value of less than 0.05.

Result

Table 1 presents the age and body mass index (BMI) distribution of the 80 study participants. The mean age of the participants was 27.8 ± 4.9 years, with the majority falling in the 26–30 years age group (32.5%), followed by 18–25 years (27.5%) and 31–35 years (25.0%), indicating that most participants were young adults. A smaller proportion (15.0%) belonged to the 36–40 years age group. The mean BMI was 27.4 ± 3.8 kg/m², reflecting an overall tendency toward increased body weight. Nearly half of the participants were overweight (45.0%), while 25.0% were classified as obese, and only 30.0% had a normal BMI, suggesting a high prevalence of excess body weight within the study population.

Table 1: Age and Body Mass Index (BMI) Distribution of Study Participants (n = 80)

Variable	Mean ± SD / n (%)
Age (years)	27.8 ± 4.9
18–25 years	22 (27.5%)
26–30 years	26 (32.5%)
31–35 years	20 (25.0%)
36–40 years	12 (15.0%)
BMI (kg/m²)	27.4 ± 3.8
Normal weight (18.5–24.9)	24 (30.0%)
Overweight (25–29.9)	36 (45.0%)
Obese (≥30)	20 (25.0%)

Table 2 depicts the menstrual pattern and associated clinical features among women with PCOS. Oligomenorrhea was the most common menstrual abnormality, observed in 55% of participants, while amenorrhea and regular menstrual cycles were equally reported in 22.5% each. Clinical hyperandrogenic manifestations were frequent, with hirsutism (mFG score >8) present in 47.5% of cases, followed by acne in 42.5% and alopecia in 20%. Infertility was a prominent concern, with a higher prevalence of primary infertility (65%) compared to secondary infertility (35%), highlighting the significant reproductive impact of PCOS in the studied population.

Table 2: Menstrual Pattern and Clinical Features of PCOS		
Clinical Parameter	**Frequency (n)**	**Percentage (%)**
Oligomenorrhea	44	55
Amenorrhea	18	22.5
Regular cycles	18	22.5
Clinical hirsutism (mFG >8)	38	47.5
Acne	34	42.5
Alopecia	16	20
Primary infertility	52	65
Secondary infertility	28	35

Table 3 depicts the ultrasonographic characteristics of the ovaries among the study participants, highlighting a high prevalence of polycystic ovarian morphology (PCOM). PCOM was observed in 72.5% of cases, indicating that the majority of women exhibited sonographic features consistent with polycystic ovaries. The presence of ≥12 follicles measuring 2–9 mm was noted in 67.5% of participants, while increased ovarian volume greater than 10 ml was documented in 57.5%, reinforcing the common occurrence of classic PCOM criteria. With respect to laterality, bilateral PCOM (47.5%) was more frequently observed than unilateral involvement (25.0%), suggesting that ovarian changes were often symmetrical. Overall, the findings demonstrate that characteristic ultrasonographic features of PCOM were highly prevalent in the study population, supporting the role of ultrasonography as an important diagnostic tool in the evaluation of ovarian morphology.

Table 3: Ultrasonographic Characteristics of Ovaries	
Ultrasonographic Finding	**n (%)**
Polycystic ovarian morphology present	58 (72.5%)
≥12 follicles (2–9 mm)	54 (67.5%)
Ovarian volume >10 ml	46 (57.5%)
Unilateral PCOM	20 (25.0%)
Bilateral PCOM	38 (47.5%)

Table 4 depicts the biochemical and hormonal profile of the study participants, highlighting characteristic endocrine alterations. The mean LH level (9.8 ± 3.4 IU/L) was higher relative to FSH (5.4 ± 1.6 IU/L), resulting in an elevated LH/FSH ratio (1.9 ± 0.7), suggestive of disordered gonadotropin secretion. Androgen levels were increased, as reflected by raised total testosterone (68.5 ± 18.2 ng/dL), free testosterone (4.6 ± 1.4 pg/mL), and DHEAS (298.7 ± 82.5 µg/dL), while SHBG levels were relatively lower (32.4 ± 10.6 nmol/L), potentially contributing to increased bioavailable androgens. Prolactin (14.2 ± 4.1 ng/mL) and TSH (2.6 ± 0.9 µIU/mL) values were within acceptable limits, indicating the absence of significant hyperprolactinemia or thyroid dysfunction. Estradiol levels (46.8 ± 15.7 pg/mL) reflected maintained estrogenic activity, collectively demonstrating a hormonal milieu consistent with endocrine imbalance.

Table 4: Biochemical and Hormonal Profile of Study Participants	
Parameter	**Mean ± SD**
LH (IU/L)	9.8 ± 3.4
FSH (IU/L)	5.4 ± 1.6
LH/FSH ratio	1.9 ± 0.7
Total testosterone (ng/dL)	68.5 ± 18.2
Free testosterone (pg/mL)	4.6 ± 1.4
DHEAS (µg/dL)	298.7 ± 82.5
SHBG (nmol/L)	32.4 ± 10.6
Prolactin (ng/mL)	14.2 ± 4.1
TSH (µIU/mL)	2.6 ± 0.9
Estradiol (pg/mL)	46.8 ± 15.7

Table 5 summarizes the metabolic parameters and insulin resistance profile of the study participants. The mean fasting glucose level (96.4 ± 12.8 mg/dL) was within the normal range, whereas the elevated fasting insulin level (14.8 ± 5.2 µIU/mL) and HOMA-IR value (3.5 ± 1.3) indicate the presence of insulin resistance. Lipid profile analysis showed borderline high total cholesterol (192.6 ± 34.5 mg/dL) and elevated triglycerides (158.3 ± 46.2 mg/dL), along with reduced HDL cholesterol levels

(41.2 ± 8.4 mg/dL), suggesting an atherogenic lipid pattern. LDL cholesterol levels (118.7 ± 28.6 mg/dL) were moderately raised. Overall, these findings reflect an unfavorable metabolic milieu characterized by insulin resistance and dyslipidemia.

Table 5: Metabolic Parameters and Insulin Resistance	
Metabolic Parameter	**Mean ± SD**
Fasting glucose (mg/dL)	96.4 ± 12.8
Fasting insulin (μIU/mL)	14.8 ± 5.2
HOMA-IR	3.5 ± 1.3
Total cholesterol (mg/dL)	192.6 ± 34.5
Triglycerides (mg/dL)	158.3 ± 46.2
HDL cholesterol (mg/dL)	41.2 ± 8.4
LDL cholesterol (mg/dL)	118.7 ± 28.6

Discussion

The present study highlights polycystic ovary syndrome (PCOS) as a major endocrine cause of infertility, characterized by intertwined clinical, hormonal, ultrasonographic, and metabolic disturbances. The predominance of women in their late twenties in this study aligns with epidemiological findings indicating that PCOS is most frequently diagnosed during the peak reproductive years, when concerns regarding infertility and menstrual irregularities become prominent (Azziz et al., 2004) [9]. The elevated mean body mass index (BMI) and high prevalence of overweight and obesity observed in our cohort further support the well-established association between PCOS and excess adiposity. Obesity is known to exacerbate insulin resistance and hyperandrogenism, thereby intensifying ovulatory dysfunction and reducing fertility potential (Esmaeilzadeh et al., 2015) [10].

Menstrual dysfunction was a dominant clinical feature in the present study, with oligomenorrhea and amenorrhea reflecting chronic anovulation, a hallmark of PCOS. Similar findings have been reported in infertile PCOS populations, where menstrual irregularities affect more than two-thirds of patients (Arain et al., 2015) [11]. Although a subset of women in our study reported regular menstrual cycles, the coexistence of biochemical hyperandrogenism and metabolic abnormalities in these individuals supports the Rotterdam consensus view that ovulatory cycles alone do not exclude the diagnosis of PCOS (Aversa et al., 2020) [12]. This observation underscores the heterogeneity of PCOS and reinforces the importance of comprehensive clinical and biochemical assessment in infertile women.

Clinical hyperandrogenism, manifested as hirsutism, acne, and alopecia, was highly prevalent in our cohort, with hirsutism being the most frequent complaint. This finding is consistent with earlier reports identifying hyperandrogenism as the central pathophysiological feature driving both cosmetic concerns and reproductive dysfunction in PCOS (Franks, 1995) [13]. Elevated total and free testosterone levels observed in the present study correlated with these clinical manifestations, supporting the role of androgen excess in disrupting follicular development and ovulation. Moghetti et al. (1996) [14] demonstrated that hyperandrogenism itself contributes to insulin resistance, independent of BMI, thereby perpetuating a cycle of metabolic and reproductive dysfunction. This interaction may partly explain the high prevalence of infertility observed in our study population.

Ultrasonographic evaluation revealed a high prevalence of polycystic ovarian morphology (PCOM), predominantly bilateral, characterized by increased follicle number and ovarian volume. These findings are consistent with the classical description of arrested folliculogenesis leading to follicle accumulation within the ovarian cortex (Jonard et al., 2003) [15]. However, a proportion of women in the present study lacked PCOM despite clear clinical and biochemical evidence of PCOS. Similar observations have been reported by Duijkers and Klipping (2010) [16], who demonstrated that PCOM can also be present in healthy women with regular menstrual cycles, thereby limiting its diagnostic specificity. This contrast reinforces the view that ultrasonographic findings should be interpreted alongside clinical and hormonal features rather than used in isolation when evaluating infertility related to PCOS.

The hormonal profile observed in this study, particularly elevated luteinizing hormone (LH) levels and an increased LH/FSH ratio, reflects altered hypothalamic–pituitary–ovarian axis function. This endocrine imbalance favors ovarian androgen production while impairing normal follicular maturation, contributing to anovulation and infertility (Taylor, 1998) [17]. Esmaeilzadeh et al. (2015) similarly reported a significant association between BMI, gonadotropin imbalance, and clinical severity in PCOS, suggesting that both metabolic and hormonal factors jointly influence reproductive outcomes. Normal prolactin and thyroid hormone levels in our study excluded other endocrine causes of menstrual irregularities, strengthening the attribution of findings specifically to PCOS.

Metabolic abnormalities were prominent in the present study despite largely normal fasting glucose levels. Elevated fasting insulin and HOMA-IR values indicate significant insulin resistance, supporting previous evidence that metabolic dysfunction in PCOS often precedes overt hyperglycemia (Moghetti et al., 1996). Insulin resistance plays a pivotal role in PCOS pathophysiology by enhancing ovarian androgen synthesis and reducing hepatic sex hormone-binding globulin production, thereby increasing free androgen availability. Dyslipidemia observed in our cohort, characterized by elevated triglycerides, borderline high total cholesterol, increased LDL, and reduced HDL, mirrors the atherogenic lipid profile reported in PCOS populations (Diamanti-Kandarakis et al., 2007) [18]. These findings suggest an increased long-term cardiovascular risk, even in young infertile women.

Overall, the present study demonstrates that infertility in PCOS is the result of complex interactions between obesity, hyperandrogenism, ovulatory dysfunction, insulin resistance, and dyslipidemia. The strong correlation between reproductive and metabolic abnormalities emphasizes the need for an integrated management approach. Lifestyle modification, weight management, and metabolic correction, alongside targeted fertility treatment, may significantly improve both reproductive outcomes and long-term health. These findings reinforce the importance of early diagnosis and holistic intervention in women with PCOS-related infertility.

Conclusion

The present study underscores polycystic ovary syndrome as a multifactorial endocrine disorder with a profound impact on female infertility through interconnected clinical, hormonal, ultrasonographic, and metabolic abnormalities. The predominance of ovulatory dysfunction, hyperandrogenism, insulin resistance, and dyslipidemia highlights the complex pathophysiology underlying impaired fertility in women with PCOS. Excess body weight and metabolic derangements were common and appeared to aggravate hormonal imbalance, reinforcing their contributory role in reproductive dysfunction. The high prevalence of polycystic ovarian morphology and characteristic gonadotropin alterations further supports the heterogeneity of PCOS phenotypes. Overall, the findings emphasize that infertility in PCOS cannot be attributed to a single factor but arises from synergistic disturbances. A comprehensive clinical and biochemical evaluation is therefore essential to guide individualized management strategies and improve reproductive outcomes.

References

1. Balen AH, Morley LC, Misso M, Franks S, Legro RS, Wijeyaratne CN, Stener-Victorin E, Fauser BC, Norman RJ, Teede H. The management of anovulatory infertility in women with polycystic ovary syndrome: an analysis of the evidence to support the development of global WHO guidance. Human reproduction update. 2016 Nov 20;22(6):687-708.
2. Livadas S, Diamanti-Kandarakis E. Polycystic ovary syndrome: definitions, phenotypes and diagnostic approach. Front Horm Res. 2013 Jan 1;40:1-21.
3. Brassard M, AinMelk Y, Baillargeon JP. Basic infertility including polycystic ovary syndrome. Medical Clinics of North America. 2008 Sep 1;92(5):1163-92.
4. Fiorentino G, Cimadomo D, Innocenti F, Soscia D, Vaiarelli A, Ubaldi FM, Gennarelli G, Garagna S, Rienzi L, Zuccotti M. Biomechanical forces and signals operating in the ovary during folliculogenesis and their dysregulation: implications for fertility. Human reproduction update. 2023 Jan 1;29(1):1-23.
5. Karkera S, Agard E, Sankova L. The clinical manifestations of polycystic ovary syndrome (PCOS) and the treatment options. European Journal of Biology and Medical Science Research. 2023 Jan;11(1):57-91.
6. Testa U, Castelli G, Pelosi E. Breast cancer: a molecularly heterogenous disease needing subtype-specific treatments. Medical Sciences. 2020 Mar 23;8(1):18.
7. Haddad-Filho H, Tosatti JA, Vale FM, Gomes KB, Reis FM. Updates in diagnosing polycystic ovary syndrome-related infertility. Expert Review of Molecular Diagnostics. 2023 Feb 1;23(2):123-32.
8. Ferrer MJ, Silva AF, Abruzzese GA, Velázquez ME, Motta AB. Lipid metabolism and relevant disorders to female reproductive health. Current Medicinal Chemistry. 2021 Aug 1;28(27):5625-47.
9. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. The Journal of Clinical Endocrinology & Metabolism. 2004 Jun 1;89(6):2745-9.
10. Esmaeilzadeh S, Andarieh MG, Ghadimi R, Delavar MA. Body mass index and gonadotropin hormones (LH & FSH) associate with clinical symptoms among women with polycystic ovary syndrome. Global journal of health science. 2014 Sep 28;7(2):101.
11. Arain F, Arif N, Halepota H. Frequency and outcome of treatment in polycystic ovaries related infertility. Pakistan journal of medical sciences. 2015 May;31(3):694.
12. Aversa A, La Vignera S, Rago R, Gambineri A, Nappi RE, Calogero AE, Ferlin A. Fundamental concepts and novel aspects of polycystic ovarian syndrome: expert consensus resolutions. Frontiers in endocrinology. 2020 Aug 11;11:516.

13. Franks S. Polycystic ovary syndrome. New England Journal of Medicine. 1995 Sep 28;333(13):853-61.
14. Moghetti P, Tosi FL, Castello RO, Magnani CM, Negri CA, Brun E, Furlani L, Caputo MA, Muggeo M. The insulin resistance in women with hyperandrogenism is partially reversed by antiandrogen treatment: evidence that androgens impair insulin action in women. The Journal of Clinical Endocrinology & Metabolism. 1996 Mar 1;81(3):952-60.
15. Jonard S, Robert Y, Cortet-Rudelli C, Pigny P, Decanter C, Dewailly D. Ultrasound examination of polycystic ovaries: is it worth counting the follicles?. Human reproduction. 2003 Mar 1;18(3):598-603.
16. Duijkers IJ, Klipping C. Polycystic ovaries, as defined by the 2003 Rotterdam consensus criteria, are found to be very common in young healthy women. Gynecological Endocrinology. 2010 Mar 1;26(3):152-60.
17. Taylor AE. Polycystic ovary syndrome. Endocrinology and Metabolism Clinics. 1998 Dec 1;27(4):877-902.
18. Diamanti-Kandarakis E, Papavassiliou AG, Kandarakis SA, Chrousos GP. Pathophysiology and types of dyslipidemia in PCOS. Trends in Endocrinology & Metabolism. 2007 Sep 1;18(7):280-5.