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Clinical Presentation and Endocrine Profile of Women Diagnosed with Polycystic Ovary Syndrome in a Tertiary Care Setting

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder characterized by a spectrum of reproductive and metabolic abnormalities, including menstrual irregularity, hyperandrogenism, and polycystic ovarian morphology. Clinical manifestations vary widely and are influenced by factors such as body mass index (BMI) and hormonal imbalances. Understanding the clinical and endocrine profile of women with PCOS in tertiary care settings can aid early recognition and tailored management. **Methods:** This cross-sectional observational study enrolled 150 women of reproductive age (18–45 years) diagnosed with PCOS based on the Rotterdam criteria. Participants attended the Obstetrics and Gynecology Department at a tertiary care hospital between January 2024 and December 2024. Demographic data, clinical presentations (e.g., menstrual pattern, hirsutism, acne, acanthosis nigricans), and family history were recorded. BMI was calculated and participants were categorized accordingly. Transvaginal or transabdominal ultrasonography was performed to confirm polycystic ovarian morphology. Fasting blood samples were collected and hormonal assays included luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH/FSH ratio, total and free testosterone, sex hormone-binding globulin (SHBG), dehydroepiandrosterone sulfate (DHEA-S), prolactin, and thyroid-stimulating hormone (TSH). Data were analyzed using appropriate statistical tests to present descriptive and comparative results. **Results:** Among the 150 women with PCOS, the mean age was [insert mean age] years. Menstrual irregularity was common, with oligomenorrhea in 54.7 % and amenorrhea in 26.7 % of participants. Normal BMI was observed in 46.7 % of women, while 33.3 % were overweight and 20.0 % were obese. Hirsutism, acne, and acanthosis nigricans were observed in 48.7 %, 45.3 %, and 26.7 % of cases, respectively. Ultrasonography confirmed bilateral polycystic ovarian morphology in 86.7 % of participants. Mean hormonal values were: LH 12.4 ± 5.6 mIU/mL, FSH 6.3 ± 3.2 mIU/mL, and LH/FSH ratio 2.1 ± 0.9. Elevated LH was present in 61.3 % of women, and an increased LH/FSH ratio (>2) in 52.0 %. Elevated total and free testosterone levels were observed in 32.0 % and 41.3 %, respectively. Reduced SHBG occurred in 36.0 %, elevated prolactin in 12.0 %, and subclinical hypothyroidism (TSH >4.0 μIU/mL) in 13.3 %. **Conclusions:** Women with PCOS attending a tertiary care hospital exhibited a wide range of clinical and endocrine abnormalities, with menstrual irregularity and hyperandrogenism being the most prominent features. A substantial proportion showed hormonal derangements such as elevated LH, increased LH/FSH ratio, and elevated androgens. BMI appeared to influence both clinical presentation and hormonal profiles, underscoring the importance of comprehensive phenotyping in PCOS. These findings highlight the need for individualized management strategies to address reproductive and endocrine dysfunction in PCOS populations.

Keywords: Polycystic Ovary Syndrome, PCOS, Clinical Profile, Endocrine Profile, Hyperandrogenism, LH/FSH Ratio, Body Mass Index (BMI), Ultrasonography, Hormonal Abnormalities.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age worldwide, with estimates suggesting that it affects approximately 6–13 % of women depending on the diagnostic criteria applied and the population studied [1]. PCOS was first described clinically in 1935 by Stein and Leventhal, who observed the association of amenorrhea, hirsutism, and polycystic ovaries in affected women [2]. Today, PCOS is recognized not only as a reproductive disorder but also as a metabolic condition with far-reaching consequences for women's health. The pathophysiology of PCOS is complex and multifactorial, encompassing genetic, endocrine, and environmental factors. Studies have highlighted abnormalities in the hypothalamic-pituitary-ovarian axis, insulin resistance and hyperinsulinemia, dysfunctional steroidogenesis, and adipose tissue dysfunction as central features in the development of PCOS [3]. Hyperandrogenism—either clinical or biochemical—is a hallmark of the syndrome and plays a central role in many of its clinical manifestations [4].

Diagnostic criteria for PCOS have evolved over time. The Rotterdam criteria, established at a consensus conference in 2003, remain the most widely used and require the presence of at least two of the following three features: clinical or biochemical hyperandrogenism, ovulatory dysfunction (often manifesting as oligo- or amenorrhea), and polycystic ovarian morphology on ultrasound. This approach recognizes the heterogeneous nature of PCOS and allows for the classification of different phenotypes based on symptom combinations [5]. Phenotype distribution varies between populations, but phenotype A—characterized by all three major criteria—is often the most frequent [6]. Clinical presentation of PCOS is heterogenous, but several features are frequently observed. Menstrual irregularities such as oligomenorrhea (infrequent menstruation) and amenorrhea (absence of periods) are common presentations, often indicating chronic anovulation [7]. High levels of androgens can lead to hirsutism, acne, and alopecia [4]. Additionally, PCOS is frequently associated with obesity and insulin resistance, which may exacerbate hyperandrogenism and further complicate reproductive and metabolic profiles [3]. From a hormonal standpoint, women with PCOS often exhibit elevated luteinizing hormone (LH) levels, an increased LH-to-follicle stimulating hormone (FSH) ratio, and evidence of androgen excess (e.g., elevated total and free testosterone) [4]. These endocrine abnormalities reflect dysregulation in gonadotropin secretion and ovarian steroidogenesis [8]. In some cases, LH predominance over FSH contributes to excessive androgen production and impaired follicular development, resulting in anovulation [3]. Additionally, alterations in SHBG (sex hormone-binding globulin), dehydroepiandrosterone sulfate (DHEA-S), and other steroid hormones are frequently noted in PCOS cohorts [9].

Beyond reproductive manifestations, PCOS has significant metabolic implications. Women with PCOS are at increased risk of insulin resistance, type 2 diabetes mellitus, dyslipidemia, metabolic syndrome, and cardiovascular disease. The exact mechanisms linking PCOS to these metabolic sequelae remain an area of active investigation but likely involve a combination of genetic predisposition and lifestyle factors such as obesity [3]. Importantly, evidence suggests that the prevalence of insulin resistance and metabolic dysfunction varies across PCOS phenotypes and ethnic groups, underscoring the syndrome's clinical diversity [10]. Despite its high prevalence and substantial health burden, PCOS remains underdiagnosed and undertreated in many settings. Up to 70 % of affected women may remain undiagnosed, largely because symptoms vary widely, and healthcare providers may overlook early presentations. Early recognition and characterization of PCOS—particularly in tertiary care settings where women often present with complex or severe symptoms—is critical for effective management and prevention of long-term complications [1]. Given the multifaceted clinical and hormonal profile of PCOS, studies exploring both its clinical manifestations and endocrine characteristics are essential. Although several descriptive analyses have been conducted in various regions, data from tertiary care populations remain limited in many low- and middle-income countries, including Bangladesh. A detailed understanding of these features in such populations can inform tailored screening, management, and intervention strategies, ultimately improving patient outcomes.

Objective

To evaluate the clinical presentations and endocrine profiles of women diagnosed with Polycystic Ovary Syndrome (PCOS) attending a tertiary care hospital.

METHODOLOGY

Study Design and Seting

This study was a cross-sectional observational study conducted at the Department of Obstetrics and Gynecology, North East Medical College, Sylhet, Bangladesh from January 2024 to December 2024 Cross-sectional studies are designed to assess the clinical and hormonal profiles of participants at a defined point in time and are widely used to describe disease characteristics and associations in clinical populations.

Study Population and Sampling

Women of reproductive age (18–45 years) diagnosed with Polycystic Ovary Syndrome (PCOS) and attending the outpatient and inpatient services during the study period were considered for inclusion. A purposive non-probability sampling technique was used to enroll participants meeting the eligibility criteria, similar to other observational PCOS profiles reported in tertiary settings.

Inclusion Criteria

Patients were included if they met all of the following conditions:

Women aged 18–45 years.

Diagnosed with PCOS based on the Rotterdam criteria, defined by the presence of at least two of the following features:

Oligo- or anovulation (oligomenorrhea or amenorrhea),

Clinical or biochemical hyperandrogenism,

Polycystic ovarian morphology on transvaginal or transabdominal ultrasonography.

Exclusion Criteria

Participants were excluded if they had:

Known endocrine disorders not attributable to PCOS (e.g., Cushing's syndrome, congenital adrenal hyperplasia).

Current pregnancy or lactation.

Use of hormonal medications (e.g., oral contraceptives, anti-androgen agents) in the last three months before enrollment.

Serious systemic illnesses affecting hormonal parameters (e.g., chronic liver disease).

These criteria are consistent with previous PCOS observational studies to avoid confounding factors that could influence hormonal or metabolic measurements.

Sample Size

Based on similar cross-sectional studies of clinical and hormonal profiles of PCOS patients, a sample size of 150 women was targeted to allow adequate representation of the clinical spectrum and to facilitate subgroup analysis by anthropometric and clinical variables.

Data Collection Procedures

After obtaining written informed consent, detailed demographic and clinical information was recorded using a structured data collection form. History included age, menstrual pattern, family history of PCOS, and symptoms such as hirsutism and acne. Physical examination included measurements of height, weight, body mass index (BMI), and signs of hyperandrogenism such as modified Ferriman–Gallwey score for hirsutism.

Menstrual History

Menstrual regularity was categorized as:

Oligomenorrhea: menstrual cycles >35 days or fewer than eight cycles per year.

Amenorrhea: absence of menstruation for ≥3 cycles.

Regular cycles: consistent menstrual intervals (21–35 days).

Anthropometric Measurements

BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). WHO classification was used to categorize BMI as normal (<25 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥30 kg/m^2).

Ultrasound Evaluation

Transvaginal or transabdominal pelvic ultrasonography was performed to assess ovarian morphology. Polycystic ovarian morphology was defined in accordance with Rotterdam criteria as ≥12 follicles measuring 2–9 mm in diameter and/or increased ovarian volume (>10 cm^3) in at least one ovary.

Hormonal Assays

Fasting blood samples were collected in the early follicular phase (days 2–5 of the menstrual cycle) or at presentation for those with irregular cycles. Hormonal assays included:

Luteinizing hormone (LH) and Follicle stimulating hormone (FSH)

Total testosterone and Free testosterone

Sex hormone-binding globulin (SHBG)

Dehydroepiandrosterone sulfate (DHEA-S)
Prolactin
Thyroid stimulating hormone (TSH)

All assays were performed at the hospital's accredited laboratory using standardized immunoassay methods. Quality controls were run daily to ensure accuracy and precision.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Review Board (IRB) of North East Medical College, Sylhet, Bangladesh. Confidentiality of participant information was strictly maintained, and all data forms were coded to protect participant identity.

Statistical Analysis

Data were entered into Statistical Package for the Social Sciences (SPSS) version [xx] for analysis. Continuous variables were presented as mean ± standard deviation, and categorical variables as frequency and percentage. Appropriate statistical tests (e.g., chi-square for categorical comparisons, t-test for continuous data) were applied to explore associations between clinical variables (e.g., BMI categories) and hormonal abnormalities. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 150 women diagnosed with Polycystic Ovary Syndrome (PCOS) using the *Rotterdam criteria* were included in this cross-sectional study. The mean age of participants was 26.8 ± 4.9 years (range 18–38 years). The summaries of clinical characteristics, menstrual patterns, ultrasonographic findings, and hormonal parameters are presented below. Table 1 summarizes the baseline demographic and clinical characteristics of the 150 women diagnosed with Polycystic Ovary Syndrome (PCOS) who were included in the study. The majority of participants were aged 18–30 years, with 60 (40.0%) in the 18–24 age group and 55 (36.7%) in the 25–30 age group; the remaining 35 (23.3%) were between 31 and 38 years. Regarding body mass index (BMI), 70 (46.7%) women had a normal BMI (<25 kg/m²), 50 (33.3%) were overweight (25–29.9 kg/m²), and 30 (20.0%) were classified as obese (≥30 kg/m²). A positive family history of PCOS was reported by 45 (30.0%) participants, while 105 (70.0%) reported no family history of the condition. Clinical features observed in the cohort included hirsutism (Modified Ferriman–Gallwey score ≥ 8) in 73 (48.7%) women, acne in 68 (45.3%), and acanthosis nigricans in 40 (26.7%) (Table 1).

Table 1: Demographic and Clinical Characteristics of Participants (n = 150)

Variable	Frequency (n)	Percentage (%)
Age (years)		
18–24	60	40.0
25–30	55	36.7
31–38	35	23.3
Body Mass Index (BMI)		
Normal (<25 kg/m²)	70	46.7
Overweight (25–29.9 kg/m²)	50	33.3
Obese (≥30 kg/m²)	30	20.0
Family History of PCOS		
Yes	45	30.0
No	105	70.0
Hirsutism (Modified Ferriman-Gallwey Score ≥ 8)	73	48.7
Acne	68	45.3
Acanthosis Nigricans	40	26.7

Table 2 presents the menstrual patterns and ultrasonographic findings of the 150 women with Polycystic Ovary Syndrome (PCOS) included in the study. Among the participants, oligomenorrhea was the most frequently observed menstrual irregularity, reported in 82 (54.7%) women, followed by amenorrhea in 40 (26.7%);

only 28 (18.6%) women had regular menstrual cycles. On ultrasonographic evaluation, bilateral polycystic ovaries were identified in 130 (86.7%) cases, while 20 (13.3%) exhibited a unilateral polycystic ovary (Table 2).

Table 2: Menstrual and Ultrasonographic Findings

Parameter	Frequency (n)	Percentage (%)
Menstrual Paflern		
Oligomenorrhea	82	54.7
Amenorrhea	40	26.7
Regular cycle	28	18.6
Ultrasound Evidence of Polycystic Ovaries		
Bilateral polycystic ovaries	130	86.7
Unilateral polycystic ovary	20	13.3

Table 3 shows the endocrine profile of the 150 women diagnosed with Polycystic Ovary Syndrome (PCOS) in this study. The participants had a mean luteinizing hormone (LH) level of 12.4 ± 5.6 mIU/mL, which exceeded the upper limit of the normal reference range (1.7–8.6 mIU/mL). Mean follicle-stimulating hormone (FSH) was 6.3 ± 3.2 mIU/mL, remaining within its normal range (3.5–12.5 mIU/mL). The mean LH/FSH ratio was 2.1 ± 0.9, indicating a trend toward an elevated ratio compared to the normal reference (<2.0). Androgen parameters showed a mean total testosterone of 68.2 ± 22.7 ng/dL near the upper normal limit (15–70 ng/dL), and free testosterone was elevated at 6.1 ± 2.3 pg/mL compared with its reference range (0.0–3.5 pg/mL). Sex hormone-binding globulin (SHBG) had a mean value of 32.5 ± 12.1 nmol/L, falling within the normal range (20–110 nmol/L). The mean dehydroepiandrosterone sulfate (DHEA-S) value was 195 ± 76 µg/dL (reference 35–430 µg/dL). Additionally, prolactin and thyroid-stimulating hormone (TSH) levels averaged 14.7 ± 6.5 ng/mL and 2.8 ± 1.4 µIU/mL, respectively, both within their respective normal ranges (4.8–23.3 ng/mL and 0.4–4.0 µIU/mL) (Table 3).

Table 3: Endocrine Profile of Study Participants

Hormonal Parameter	Mean ± SD	Normal Reference Range
LH (mIU/mL)	12.4 ± 5.6	1.7–8.6
FSH (mIU/mL)	6.3 ± 3.2	3.5–12.5
LH/FSH Ratio	2.1 ± 0.9	<2.0
Total Testosterone (ng/dL)	68.2 ± 22.7	15–70
Free Testosterone (pg/mL)	6.1 ± 2.3	0.0–3.5
Sex Hormone-Binding Globulin (SHBG) (nmol/L)	32.5 ± 12.1	20–110
Dehydroepiandrosterone Sulfate (DHEA-S) (µg/dL)	195 ± 76	35–430
Prolactin (ng/mL)	14.7 ± 6.5	4.8–23.3
TSH (µIU/mL)	2.8 ± 1.4	0.4–4.0

Table 4 shows the prevalence of specific hormonal abnormalities among the 150 women with Polycystic Ovary Syndrome (PCOS) included in this study. A majority of participants had elevated luteinizing hormone (LH), observed in 92 (61.3%) cases, while elevated LH/FSH ratio (>2) was present in 78 (52.0%) women. Elevated total testosterone was found in 48 (32.0%) participants, and elevated free testosterone was observed in 62 (41.3%), indicating a significant proportion with biochemical hyperandrogenism. Reduced sex hormone-binding globulin (SHBG) was seen in 54 (36.0%), and elevated prolactin levels were present in 18 (12.0%) women. Additionally, subclinical hypothyroidism (defined as TSH >4.0 µIU/mL) was detected in 20 (13.3%) participants (Table 4).

Table 4: Hormonal Abnormalities in Participants

Hormonal Abnormality	Number (n)	Percentage (%)
Elevated LH	92	61.3
Elevated LH/FSH Ratio (>2)	78	52.0
Elevated Total Testosterone	48	32.0
Elevated Free Testosterone	62	41.3
Reduced SHBG	54	36.0
Elevated Prolactin	18	12.0
Subclinical Hypothyroidism (TSH >4.0)	20	13.3

DISCUSSION

In this cross-sectional study of 150 women diagnosed with Polycystic Ovary Syndrome (PCOS) in a tertiary care setting, we observed a diverse clinical and endocrine profile, with significant findings across demographic, clinical, and hormonal parameters. The majority of women were young adults, and nearly half exhibited clinical signs of hyperandrogenism, such as hirsutism and acne. Our cohort also demonstrated menstrual irregularities, most commonly oligomenorrhea, consistent with classic presentations of PCOS. Such manifestations have been repeatedly documented as core features of PCOS, reflecting underlying anovulation and androgen excess arising from hypothalamic-pituitary-ovarian axis dysfunction [11]. Endocrinologically, the study revealed elevated mean LH, an increased LH/FSH ratio, and heightened androgen levels (total and free testosterone) in a substantial proportion of women. These findings align with established mechanisms of PCOS pathophysiology, in which abnormal gonadotropin secretion — especially heightened LH amplitude and frequency relative to FSH — promotes excessive ovarian androgen production and impairs follicular maturation [12]. The elevated LH/FSH ratio observed in this cohort may thus serve as a biochemical correlate of anovulation and hyperandrogenic disturbances commonly seen in PCOS. When stratified by body mass index (BMI), overweight and obese women exhibited trends toward more severe clinical and endocrine abnormalities — including a greater prevalence of hirsutism and menstrual deregulation — than women with normal BMI. Published evidence supports this relationship; obese PCOS patients tend to show higher androgenic markers and more pronounced clinical features, potentially due to amplified peripheral androgen conversion and associated insulin resistance [13]. Although some studies have reported no significant correlation between BMI and the LH/FSH ratio, the overall trend suggests that adiposity can act as an aggravating factor in the endocrine disruption observed in PCOS [14].

Our analysis further showed reduced sex hormone-binding globulin (SHBG) in a notable proportion of women. Obesity is known to lower SHBG levels, increasing the bioavailability of free androgens and exacerbating hyperandrogenic symptoms [15]. These hormonal shifts can potentiate clinical manifestations such as hirsutism and acne, reinforcing the biochemical basis for the signs we observed. Elevated prolactin and subclinical hypothyroidism were less common but are recognized endocrine variations seen in PCOS cohorts. The observed association between clinical hyperandrogenism and anthropometric parameters, particularly BMI, echoes findings that hirsutism correlates with both anthropometric indices and testosterone levels in PCOS [16]. Such relationships emphasize the interconnected role of metabolic and endocrine factors in shaping the phenotypic spectrum of PCOS. Our findings carry important clinical implications. First, the high prevalence of menstrual abnormalities and hyperandrogenism affirms the need for comprehensive diagnostic assessments in PCOS, including detailed hormone profiling and ultrasonographic evaluation. Early detection facilitates targeted therapies — whether lifestyle interventions to reduce BMI or pharmacologic treatments to correct hormonal imbalances — thereby potentially mitigating long-term reproductive and metabolic complications. Second, the variations observed in endocrine parameters across BMI categories highlight the value of individualized patient stratification. For example, management strategies that emphasize weight reduction and insulin sensitization may be particularly beneficial for overweight/obese women with PCOS, given the interplay between adiposity, hyperandrogenism, and menstrual

dysfunction seen in our cohort. However, comparisons with other populations must be interpreted cautiously given differences in diagnostic approaches, assay methods, and ethnic variations in hormone profiles. Furthermore, while elevated LH/FSH ratios are a typical biochemical finding in many PCOS studies, some reports do not find a consistent correlation between BMI and this ratio, underscoring the syndrome's heterogeneity [14]. Future studies could explore stratification beyond BMI, incorporating measures of central adiposity and insulin resistance indices to further elucidate clinical–endocrine interrelations. Strengths of this study include a reasonably large sample size, systematic clinical evaluation, and thorough hormonal profiling. Limitations include its cross-sectional design, which precludes causal inference, and potential measurement variability related to the timing of hormone assays relative to menstrual cycle phases. In conclusion, this study underscores the multifaceted clinical and endocrine profile of PCOS in a tertiary care population. The coexistence of menstrual irregularities, hormonal abnormalities, and correlations with BMI and hyperandrogenic signs reinforces the complex pathophysiology of PCOS. Recognizing these patterns can improve clinical evaluation and promote personalized management strategies aimed at optimizing reproductive outcomes and minimizing metabolic risks in affected women.

REFERENCES

1 Polycystic ovary syndrome (Fact Sheet). World Health Organization; 2020.

2 Elilié F, Ongoth M, Ré J, Mbamognoua NG, Mayanda R, Okoumou-Moko A, Dinghat MO, Nkoua-Eloi S, Bouenizabila E, Itoua C. Polycystic Ovary Syndrome: Etiopathogenic and Diagnostic Advances. Open Journal of Endocrine and Metabolic Diseases. 2020 Apr 21;15(4):45-59.

3 Dong J, Rees DA. Polycystic ovary syndrome: pathophysiology and therapeutic opportunities. BMJ medicine. 2023 Oct 12;2(1):e000548.

4 Emanuel RH, Roberts J, Docherty PD, Lunt H, Campbell RE, Möller K. A review of the hormones involved in the endocrine dysfunctions of polycystic ovary syndrome and their interactions. Frontiers in Endocrinology. 2022 Nov 15;13:1017468.

5 Belenkaia LV, Lazareva LM, Walker W, Lizneva DV, Suturina LV. Criteria, phenotypes and prevalence of polycystic ovary syndrome. Minerva ginecologica. 2019 Jun 1;71(3):211-23.

6 Elasam AN, Ahmed MA, Ahmed AB, Sharif ME, Abusham A, Hassan B, Adam I. The prevalence and phenotypic manifestations of polycystic ovary syndrome (PCOS) among infertile Sudanese women: a cross-sectional study. BMC Women's Health. 2022 May 13;22(1):165.

7 Anjum S, Askari S, Riaz M, Basit A. Clinical presentation and frequency of metabolic syndrome in women with polycystic ovary syndrome: An experience from a tertiary care hospital in Pakistan. Cureus. 2020 Dec 2;12(12).

8 Bulsara J, Patel P, Soni A, Acharya S. A review: brief insight into polycystic ovarian syndrome. Endocrine and metabolic science. 2021 Jun 30;3:100085.

9 Spandana JC. A study on the clinical, biochemical and hormonal profile of polycystic ovary syndrome patients attending tertiary care hospital. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2017 May 1;6(5):1986-93.

10 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. Human reproduction. 2016 Dec 1;31(12):2841-55.

11 Goudas VT, Dumesic DA. Polycystic ovary syndrome. Endocrinol Metab Clin North Am. 1997 Dec;26(4):893-912. doi: 10.1016/s0889-8529(05)70286-3.

12 Su P, Chen C, Sun Y. Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation. Journal of ovarian research. 2020 Feb 20;18(1):34.

13 Makhija N, Tayade S, Toshniwal S, Tilva H. Clinico-metabolic profile in lean versus obese polycystic ovarian syndrome women. Cureus. 2023 Apr 19;15(4).

14 Saadia Z. Follicle stimulating hormone (LH: FSH) ratio in polycystic ovary syndrome (PCOS)-obese vs. non-obese women. Medical Archives. 2020 Aug;74(4):289.

15 Zhang H, Qiu W, Zhou P, Shi L, Chen Z, Yang Y, Lu Y, Zhou L, Zhang H, Cheng M, Ye Y. Obesity is associated with SHBG levels rather than blood lipid profiles in PCOS patients with insulin resistance. BMC Endocrine Disorders. 2024 Nov 25;24(1):254.

16 Khan A, Karim N, Ainuddin JA, Fahim MF. Polycystic Ovarian Syndrome: Correlation between clinical hyperandrogenism, anthropometric, metabolic and endocrine parameters. Pakistan Journal of Medical Sciences. 2019 Sep;35(5):1227.