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A Study on the Clinical, Hormonal and Gynecological Parameters in Polycystic Ovarian Syndrome

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ABSTRACT

Background: Polycystic ovary syndrome is a common endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Variability in clinical presentation and hormonal patterns complicates timely diagnosis and assessment. This study evaluated clinical features, hormonal alterations, and gynecological ultrasound findings in affected women compared with healthy controls. **Material and Methods:** A comparative cross-sectional study was conducted at the Department of Obstetrics and Gynecology, Sheikh Zayed Medical Complex, Lahore, from March to June 2025, following institutional ethical approval. A total of 104 women aged 18–45 years were enrolled, including 52 with the condition and 52 healthy controls selected through consecutive sampling. Clinical features, including menstrual irregularities, hirsutism, acne, alopecia, and body mass index, were documented. Early-cycle serum luteinizing hormone, follicle-stimulating hormone, testosterone, estradiol, and thyroid-stimulating hormone levels were measured. Transvaginal ultrasonography assessed ovarian volume and follicle count. Data were analyzed using appropriate parametric and non-parametric tests, with $p<0.05$ considered significant. **Results:** Affected women had higher body mass index (26.1 ± 5.0 vs 22.5 ± 3.6 kg/m^2, $p<0.001$) and greater frequency of oligomenorrhea (55.8% vs 11.5%, $p<0.001$), amenorrhea (21.2% vs 1.9%, $p<0.001$), hirsutism (63.5% vs 11.5%, $p<0.001$), acne (57.7% vs 15.4%, $p<0.001$), and androgenic alopecia (34.6% vs 5.8%, $p<0.001$). Luteinizing hormone levels were significantly elevated (9.82 ± 3.15 vs 5.21 ± 2.48 IU/L, $p<0.001$), while follicle-stimulating hormone was lower (4.32 ± 1.04 vs 6.18 ± 1.22 IU/L, $p<0.001$), resulting in a higher LH:FSH ratio. Estradiol concentrations were increased (134.6 ± 32.8 vs 96.4 ± 24.2 pg/mL, $p<0.001$), whereas testosterone and thyroid-stimulating hormone showed no significant differences. Ultrasound revealed larger ovarian volumes and increased follicle counts bilaterally, with polycystic ovarian morphology present in 73.1% of affected women versus 9.6% of controls ($p<0.001$). **Conclusion:** Women with the condition exhibited distinct clinical, hormonal, and ultrasonographic differences from healthy controls. The combined evaluation of clinical manifestations, endocrine abnormalities, and ovarian morphology enhances diagnostic precision and supports more comprehensive assessment in routine gynecological practice.

INTRODUCTION

Polycystic ovarian syndrome (PCOS) represents a prevalent endocrine disorder, afflicting approximately 6–10% of women in their reproductive years [1]. Significantly, the prevalence of PCOS exhibits considerable geographic and racial variability. Notably, studies employing the Rotterdam criteria have indicated that in South Asian populations, particularly among Pakistani women, the prevalence reaches an alarming rate of 52%, which starkly contrasts with the 20-25% observed in Caucasian populations in the United Kingdom [2]. This syndrome is marked by a constellation of symptoms that can severely affect a woman's hormonal balance, reproductive capabilities, and overall health. PCOS is identified through a triad of characteristics: hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, as observed in ultrasound examinations. Hyperandrogenism is particularly noted as the most consistent manifestation of PCOS in both adult and adolescent females [3,4].

The clinical presentation of PCOS is diverse, ranging from hirsutism and acne to obesity, all of which suggest underlying hormonal imbalances. These imbalances include atypical levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), thyroid-stimulating hormone (TSH), estrogen, and testosterone. Furthermore, PCOS is frequently accompanied by various gynecological anomalies, complicating the clinical picture [5,6]. Despite

advancements in the understanding of PCOS, significant gaps remain in the comprehension of its pathophysiology, diagnosis, and optimal management strategies. Adolescents presenting with PCOS symptoms pose a particular diagnostic challenge, as their symptoms often overlap with those of normal puberty, leading to potential under or misdiagnosis [7,8].

A case-control study compared 201 women with PCOS (cases) to 233 healthy controls, finding significant differences in age (cases: 27.12 ± 5.521 years; controls: 25.83 ± 4.638 years, p=.010), BMI (cases: 24.72 ± 4.926 kg/m^2; controls: 22.30 ± 3.794 kg/m^2, p=.001). LH levels were higher (cases: 5.12 ± 0.31 IU/L; controls: 3.83 ± 0.29 IU/L, p=.047) and FSH lower in cases (cases: 4.08 ± 0.36 IU/L; controls: 5.90 ± 0.93 IU/L, p=.00), with elevated estradiol (cases: 105.88 ± 20.54 pmol/l; controls: 78.53 ± 8.81 pmol/l, p=.00). Testosterone levels (cases: 6.65 ± 2.68 nmol/L; controls: 6.44 ± 2.90 nmol/L, p=.057), SHBG (cases: 16.84 ± 15.19 nmol/L; controls: 16.31 ± 14.26 nmol/L, p=.965), and free testosterone index (cases: 4.53 ± 0.07; controls: 4.17 ± 0.07, p=.562) were not significantly different [9]. Another study examined fifty PCOS patients, diagnosed according to the Rotterdam criteria, against twenty healthy females with regular menstrual cycles and no reproductive history issues. Findings revealed the PCOS group had a mean fasting blood sugar level of 79.92 ± 8.55 mg%, a mean BMI of 26.54 ± 5.14 kg/m^2, and significantly higher mean LH levels (10.46 ± 3.02 mIU/ml) compared to controls (5.89 ± 2.12 mIU/ml, $p < 0.001$). The mean LH-FSH ratio also differed significantly between the PCOS group (1.97 ± 0.83) and controls (1.17 ± 0.37, $p < 0.001$). Thyroid-stimulating hormone levels were similar between groups (study: 2.42 ± 0.99 µIU/ml; control: 2.48 ± 0.72 µIU/ml, $p = 0.811$). Ultrasonography showed 72% of the PCOS group with polycystic ovaries, while all controls had normal ovarian morphology [7].

The rationale for this study lies in the need to clarify ongoing inconsistencies in the clinical, hormonal, and gynecological profile of polycystic ovary syndrome, a highly prevalent condition with substantial effects on women's health. Understanding how menstrual irregularities, hyperandrogenic features, and ovarian morphology relate to underlying endocrine disturbances is essential for improving diagnostic precision and management. This study therefore aimed to compare the frequency and severity of key clinical features, quantify variations in luteinizing hormone, follicle-stimulating hormone, testosterone, estrogen, and thyroid-stimulating hormone, and evaluate ovarian volume and follicle count along with their hormonal correlations between affected women and healthy controls.

MATERIAL AND METHOD

The study was designed as a comparative cross-sectional investigation conducted in the Department of Obstetrics and Gynecology at Sheikh Zayed Medical Complex, Lahore. Ethical approval had been obtained from the Institutional Review Board prior to initiation of data collection, and the study period extended from 2 March 2025 to 4 June 2025. A total sample of 104 women, comprising 52 individuals with polycystic ovary syndrome and 52 age-matched healthy controls, had been enrolled through non-probability consecutive sampling. The sample size had been calculated using reported BMI estimates for affected individuals (24.72 ± 4.926 kg/m^2) and controls (22.30 ± 3.794 kg/m^2), incorporating a 95% confidence level and 80% statistical power [9]. Eligibility required women aged 18–45 years. Participants in the affected group satisfied at least two Rotterdam diagnostic criteria, encompassing oligo-ovulation or anovulation, clinical or biochemical hyperandrogenism, or sonologically defined polycystic ovaries. Controls were healthy women with regular menstrual cycles, no evidence of endocrine disturbance, and no history of infertility or hyperandrogenic manifestations. Women were excluded if they had systemic or endocrine disorders that could mimic polycystic ovary syndrome, recent use of hormonal or metabolic medications, significant cardiac, hepatic, renal, or psychiatric disease, pregnancy or lactation, or prior surgical or aesthetic interventions targeting PCOS-related manifestations; matched exclusions applied to the control group to avoid confounding.

After written informed consent had been obtained, eligible participants underwent structured interviews to document demographic and reproductive characteristics, including menstrual patterns, symptoms of hyperandrogenism, and weight-related data. Clinical definitions followed standardized criteria: oligo-ovulation was indicated by fewer than eight cycles annually; hirsutism was assessed by terminal hair scoring across 11 sites with a diagnostic threshold of eight or more points; acne vulgaris was characterized by the presence of comedones, papules, pustules, nodules, or cysts; androgenic alopecia followed the clinical pattern of crown and frontal thinning; amenorrhea represented absence of menses for at least six months; menometrorrhagia denoted prolonged irregular bleeding; and oligomenorrhea corresponded to intervals exceeding 35 days. Obesity classification was based on calculated body mass index, with standardized thresholds applied for underweight, normal weight, overweight, and classes I–III obesity. Hormonal parameters were measured using early-cycle venous samples to obtain luteinizing hormone, follicle-stimulating hormone, testosterone, estradiol, and thyroid-stimulating hormone concentrations within established reference ranges.

All participants underwent transvaginal ultrasonography using a GE Voluson E8 system to document ovarian volume and follicle number, based on diagnostic criteria of ≥12 follicles measuring 2–9 mm or ovarian volume exceeding 10 mL. Additional examinations and laboratory tests were performed to exclude alternative endocrinopathies where clinically indicated. Data collection was completed using a structured proforma.

Statistical analysis was carried out using SPSS version 26. Continuous variables were summarized as means and standard deviations, and categorical data as frequencies and percentages. Between-group comparisons of clinical and hormonal parameters were performed using independent t-tests for normally distributed variables or the Mann–Whitney U test for skewed distributions. Categorical variables were compared using the Chi-square or Fisher's exact test. Correlations between hormonal markers, clinical hyperandrogenic features, and

ultrasonographic parameters were assessed using Pearson or Spearman coefficients as appropriate. A p-value below 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 104 women were enrolled, comprising 52 diagnosed with polycystic ovary syndrome and 52 healthy controls. The mean age was 27.4 ± 5.2 years in the affected group and 25.9 ± 4.8 years in the control group. Body mass index was significantly higher in the affected group (26.1 ± 5.0 kg/m^2) compared with controls (22.5 ± 3.6 kg/m^2, p<0.001). Menstrual irregularities were more common among affected women, with oligomenorrhea observed in 55.8%, amenorrhea in 21.2%, and menometrorrhagia in 15.4%, while the majority of controls reported regular cycles. Hirsutism (63.5%), acne (57.7%), and androgenic alopecia (34.6%) were also significantly more frequent among affected participants. Obesity (BMI ≥30 kg/m^2) was present in 30.8% of the affected group compared with 7.7% of controls.

Table 1

Clinical Characteristics of Participants (n = 104)

Variable	PCOS (n=52)	Control (n=52)	p-value
Age (years), mean ± SD	27.4 ± 5.2	25.9 ± 4.8	0.041
BMI (kg/m^2), mean ± SD	26.1 ± 5.0	22.5 ± 3.6	<0.001
Oligomenorrhea, n (%)	29 (55.8%)	6 (11.5%)	<0.001
Amenorrhea, n (%)	11 (21.2%)	1 (1.9%)	<0.001
Menometrorrhagia, n (%)	8 (15.4%)	2 (3.8%)	0.047
Hirsutism ≥8 score, n (%)	33 (63.5%)	6 (11.5%)	<0.001
Acne, n (%)	30 (57.7%)	8 (15.4%)	<0.001
Androgenic alopecia, n (%)	18 (34.6%)	3 (5.8%)	<0.001
Obesity (BMI ≥30), n (%)	16 (30.8%)	4 (7.7%)	0.004

Hormonal analysis demonstrated significantly higher luteinizing hormone levels in the affected group (9.82 ± 3.15 IU/L) compared with controls (5.21 ± 2.48 IU/L, p<0.001). Follicle-stimulating hormone was lower (4.32 ± 1.04 vs 6.18 ± 1.22 IU/L, p<0.001), resulting in an elevated LH:FSH ratio. Estradiol concentrations were significantly higher among affected participants (134.6 ± 32.8 pg/mL vs 96.4 ± 24.2 pg/mL, p<0.001). Testosterone showed a borderline non-significant elevation, while thyroid-stimulating hormone did not differ between groups.

Table 2

Hormonal Parameters in PCOS and Controls

Marker	PCOS (Mean ± SD)	Controls (Mean ± SD)	p-value
LH (IU/L)	9.82 ± 3.15	5.21 ± 2.48	<0.001
FSH (IU/L)	4.32 ± 1.04	6.18 ± 1.22	<0.001
LH:FSH Ratio	2.27 ± 0.78	0.86 ± 0.32	<0.001
Testosterone (nmol/L)	2.18 ± 0.92	1.84 ± 0.71	0.056
Estradiol (pg/mL)	134.6 ± 32.8	96.4 ± 24.2	<0.001
TSH (μIU/mL)	2.51 ± 0.97	2.46 ± 0.82	0.762

Ultrasound evaluation showed significantly increased ovarian volume and follicle count in the affected group. Right ovarian volume averaged 12.8 ± 3.9 cm^3 compared with 7.4 ± 2.1 cm^3 in controls (p<0.001), while left ovarian volumes were 13.1 ± 4.2 cm^3 and 7.2 ± 2.4 cm^3, respectively (p<0.001). Follicle counts were markedly higher on both sides. Polycystic ovarian morphology was identified in 73.1% of affected women compared with 9.6% of controls (p<0.001).

Table 3

Gynecological Ultrasound Findings

Parameter	PCOS (Mean ± SD / n %)	Controls (Mean ± SD / n %)	p-value
Right ovarian volume (cm^3)	12.8 ± 3.9	7.4 ± 2.1	<0.001
Left ovarian volume (cm^3)	13.1 ± 4.2	7.2 ± 2.4	<0.001
Right follicle count	18.6 ± 4.8	8.9 ± 2.7	<0.001
Left follicle count	19.2 ± 5.1	9.4 ± 2.9	<0.001
Polycystic ovarian morphology, n (%)	38 (73.1%)	5 (9.6%)	<0.001

DISCUSSION

The present case–control analysis demonstrated a consistent pattern of clinical, hormonal, and ultrasonographic differences between women with polycystic ovary syndrome and age-matched healthy controls, aligning with contemporary diagnostic constructs that emphasise ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. The higher frequency of menstrual irregularities, hirsutism and acne, alongside greater ovarian volume and follicle counts, was concordant with the syndrome's cardinal phenotype, while biochemical profiling showed a higher luteinising hormone concentration with a lower follicle-stimulating hormone level and comparable thyroid-stimulating hormone, mirroring recognised endocrine signatures of the disorder [10–12]. The absence of a statistically significant difference in total testosterone with a modest difference in free-androgen surrogates is biologically plausible, given known variability in assay performance and the influence of sex hormone–binding globulin on the free androgen fraction [13,18,19].

The hormonal pattern observed—elevated luteinising hormone with relative suppression of follicle-stimulating hormone—supports a pathophysiological model centred on accelerated gonadotropin-releasing hormone pulsatility and preferential pituitary luteinising hormone secretion, a hallmark disturbance in polycystic ovary syndrome that fosters ovarian theca hyperplasia and androgen excess [10,13]. While some programmes historically relied on the luteinising hormone/follicle-stimulating hormone ratio, contemporary evidence cautions against over-reliance on a numeric threshold because ratios vary across assays and populations; the present data comport with this perspective by demonstrating between-group differences in absolute gonadotropins rather than a uniform ratio cut-off [16,17]. In parallel, the lack of a large between-group separation in total testosterone underscores the importance of evaluating free-androgen indices and sex hormone–binding globulin, since reductions in sex hormone–binding globulin associated with adiposity and insulin resistance amplify biologically active androgens without necessarily elevating total testosterone substantially [18,19].

The clinical profile in the affected group—more frequent oligomenorrhoea/amenorrhoea and higher hirsutism and acne burden—accords with the established spectrum of hyperandrogenic and ovulatory manifestations, and is consistent with guideline summaries and narrative

reviews that highlight cutaneous features as reliable markers of androgen action at the pilosebaceous unit and hair follicles [11,12,15]. These findings are also congruent with regional literature indicating a comparatively high symptomatic burden in South Asian settings, where genetic and lifestyle factors may potentiate insulin resistance and adiposity, thereby exacerbating hyperandrogenism through reduced sex hormone–binding globulin and augmented ovarian thecal steroidogenesis [13,23,24]. The higher body mass index observed in the affected group is therefore expected to intensify the endocrine milieu via hepatic and peripheral mechanisms, further reinforcing the clinical expression of the disorder [15,24].

Ultrasonographic differences observed in ovarian volume and follicle number map onto current morphology thresholds. Earlier Rotterdam statements emphasised ≥12 follicles (2–9 mm) and/or ovarian volume >10 mL, whereas more recent technology-informed guidance proposes follicle number per ovary ≥20–25 with ovarian volume ≥10 mL in adults; the present imaging findings sit within this evolving framework and support a robust morphological component to diagnosis when assessed with appropriate equipment and cycle timing [10–14,21,22]. The alignment with updated international guidance further strengthens the external validity of the sonographic component in this population [12,14,17].

The neutral between-group difference for thyroid-stimulating hormone is consistent with guideline statements that recommend screening for thyroid dysfunction as a comorbidity rather than a defining feature; available evidence suggests thyroid status can modulate gonadotropin dynamics and the luteinising hormone/follicle-stimulating hormone ratio in subsets, but a universal elevation is not anticipated, explaining the similarity observed here [12,20]. In adolescents, where physiological anovulation and acne are common, diagnostic caution is recommended; however, the current adult sample and the requirement for at least two Rotterdam features mitigate the risk of over-classification, and the observed concordance of clinical, hormonal, and morphological markers strengthens case status attribution [12,15].

Taken together, these findings corroborate contemporary models in which disordered folliculogenesis and chronic anovulation arise from an interplay between hypothalamic–pituitary–ovarian signalling, intrinsic thecal hyperandrogenism, and metabolic modifiers, particularly adiposity and insulin resistance. The pattern of higher luteinising hormone and estradiol with lower follicle-stimulating hormone, more pronounced clinical hyperandrogenism, and enlarged, micro-follicle-rich ovaries reflects this integrated pathophysiology and is broadly consistent with international guidance and region-specific reports [10–14,23–25]. The clinical implication is that comprehensive evaluation—integrating symptoms, targeted biochemistry with attention to free-androgen surrogates, and high-quality ultrasound—remains essential for diagnostic accuracy and risk stratification, particularly in settings with high baseline prevalence and heterogeneous phenotypes.

CONCLUSION

The study demonstrated significant differences in clinical, hormonal, and gynecological parameters between women with polycystic ovary syndrome and healthy controls. Menstrual disturbances, hyperandrogenic features, and higher body mass index were markedly more frequent among affected women, accompanied by a distinct hormonal pattern characterized by elevated luteinizing hormone, reduced follicle-stimulating hormone, and increased estradiol levels. Ultrasound findings further confirmed enlarged ovarian volumes and increased follicle counts in the affected group. These results reaffirm the characteristic clinical and endocrine profile of the condition and underscore the value of integrated hormonal and ultrasonographic assessment in improving diagnostic accuracy.

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