Original Article

Dissociation of Clinical and Anthropometric Phenotypes from Severe Endocrine Disruptions in Polycystic Ovary Syndrome: A Regional Cross-Sectional Study from Western Iran

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Abstract

Background: The clinical heterogeneity of Polycystic Ovary Syndrome (PCOS) complicates risk stratification, particularly when phenotypic presentations mismatch endocrine severity. This study investigated clinical, anthropometric, and biochemical variations in Iranian PCOS patients to evaluate if physical traits reliably reflect underlying gonadotropin and androgenic disruptions.

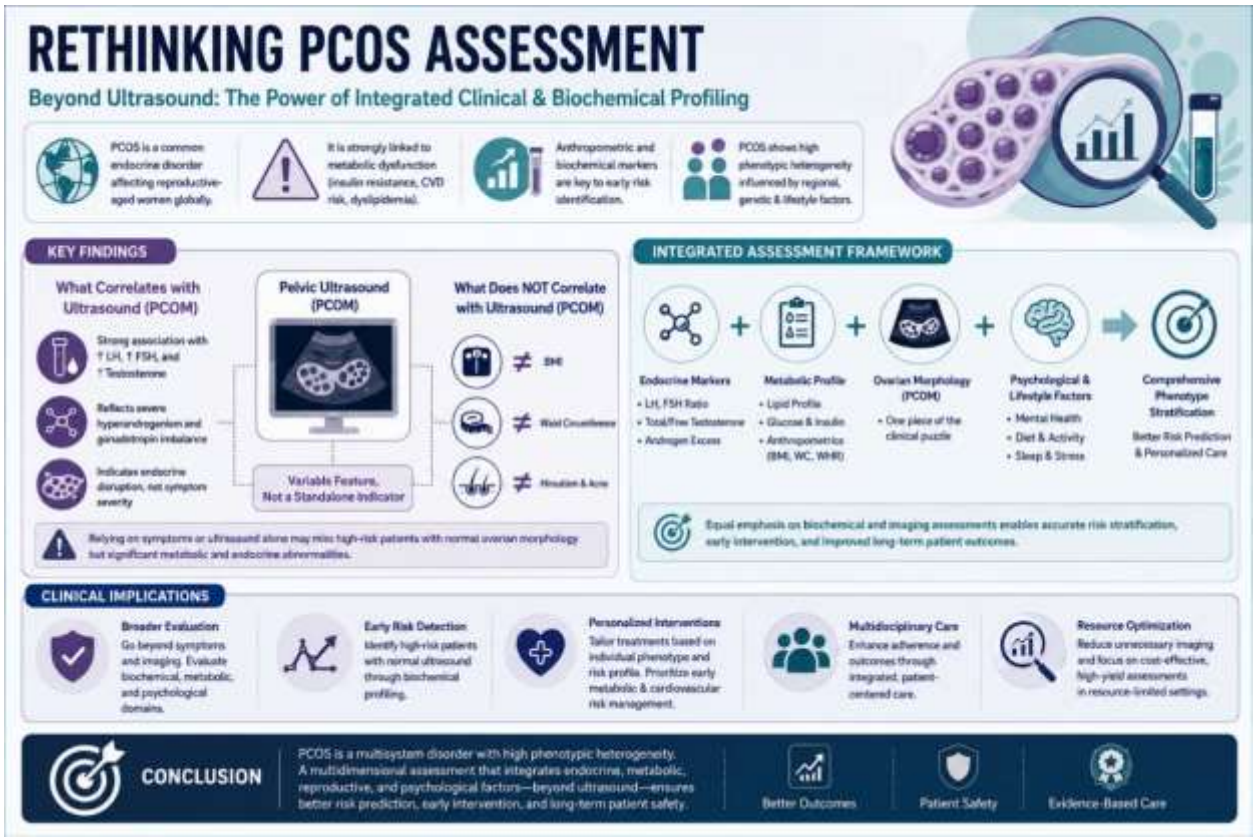
Materials and Methods: In this cross-sectional study, 211 women diagnosed with PCOS (Rotterdam criteria) at a regional referral center were stratified based on ultrasonographic evidence of polycystic ovarian morphology (PCOM) into PCOM-Negative (n = 147) and PCOM-Positive (n = 64) groups. Anthropometric parameters, clinical signs (hirsutism, acne), and serum biochemical profiles were compared.

Results: The mean age was 22.92 ± 2.63 years. Clinical hirsutism and acne affected 73.5% and 47.9% of the total population, respectively. Paradoxically, peripheral clinical features and anthropometric distributions showed structural dissociation; clinical hirsutism ($p = 0.316$), acne ($p = 0.231$), and BMI categories ($p = 0.230$) were uniformly distributed regardless of ovarian morphology. Conversely, the biochemical profile revealed profound divergence; PCOM-Positive patients exhibited significantly higher LH levels (6.79 ± 0.95 vs. 4.93 ± 0.49 mIU/mL, $p < 0.001$) and marked hyperandrogenemia than their PCOM-Negative counterparts.

Conclusion: Peripheral phenotypic traits are poor predictors of internal endocrine severity in PCOS. Normal ovarian morphology can mask severe gonadotropin derangements. Objective biochemical stratification must be prioritized over subjective clinical scoring.

Implications for Patient Care: Clinicians must implement multidimensional screening protocols granting equal weight to biochemical profiling (testosterone spikes and LH/FSH inversions) alongside imaging. This eliminates diagnostic blind spots for high-risk patients with normal morphology, optimizing resource allocation and facilitating earlier targeted metabolic therapies in resource-limited regional settings.

Keywords: Polycystic ovary syndrome; Ovarian morphology; Biochemical hyperandrogenism; Gonadotropin imbalance; Phenotypic heterogeneity



Graphical Abstract. Integrated Risk Stratification in PCOS: Biochemical hyperandrogenism and gonadotropin imbalances, rather than ovarian morphology alone, are primary drivers of metabolic risk. A combined assessment of hormonal and imaging profiles is essential for the early detection and personalized management of high risk PCOS phenotypes.

Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders among reproductive-aged women worldwide (1). PCOS is characterized by oligo-anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology, with diagnosis requiring at least two of these features (2). Beyond its primary reproductive challenges, PCOS is heavily linked to severe metabolic dysfunction (3). Women with this syndrome face a significantly higher risk of developing insulin resistance, type 2 diabetes, dyslipidemia, and cardiovascular diseases later in life (4). These metabolic risks often begin in adolescence and progress into adulthood, making timely evaluation crucial (5). Consequently, early and accurate clinical assessment is vital to mitigate these long-term health complications and reduce the overall healthcare burden (6). The clinical presentation of PCOS varies widely among individual patients, showcasing a diverse range of phenotypes (7). This phenotypic heterogeneity is further compounded by emerging evidence suggesting a bidirectional relationship between obesity and PCOS, where adiposity worsens PCOS via insulin resistance and hyperandrogenism, while PCOS promotes weight gain and metabolic dysfunction (8). Anthropometric indices, such as BMI, waist circumference, and waist-to-hip ratio, are simple, non-invasive, and cost-effective measures commonly used to assess metabolic disorders and insulin resistance (9). When combined with biochemical markers like lipid profiles and

glycemic indices, these physical measurements provide a much clearer picture of a patient's current health status (10). Identifying these connections helps clinicians recognize high-risk patients before advanced metabolic diseases develop (11). Nevertheless, these metabolic profiles in PCOS are not uniform; they are deeply influenced by regional, cultural, genetic, and lifestyle factors (12). While the global burden of PCOS is well-documented, localized clinical data remains essential for tailored healthcare delivery (7). Evaluating the specific demographic, physical, and biochemical characteristics of patients within a given population helps clinicians design more effective, patient-centered interventions (13). In many developing regions, accessing advanced diagnostic tools is challenging, which elevates the clinical value of baseline physical and biochemical tracking (14). Therefore, to address this regional gap, this study aims to investigate the demographic, anthropometric, and biochemical profiles of women with PCOS referring to a major referral gynecology clinic in western Iran. By stratifying clinical and biochemical phenotypes, we look to provide practical insights that support better clinical decision-making, optimize resource allocation, and enhance early metabolic screening strategies in routine clinical practice.

Methods

Study Design, Institutional Setting, and Ethical Compliance

This descriptive-analytical, cross-sectional study evaluated the demographic, anthropometric, and biochemical characteristics of women diagnosed with Polycystic Ovary Syndrome (PCOS). To minimize selection bias and generate robust real-world evidence, participants were consecutively enrolled from the outpatient gynecology clinic of Shahid Rahimi Hospital, a major tertiary referral center affiliated with Lorestan University of Medical Sciences in Khorramabad, Iran. Data collection spanned the first half of 2023.

This investigation strictly complied with the ethical principles outlined in the Declaration of Helsinki. The study protocol received formal review and approval from the Institutional Ethics Committee of Lorestan University of Medical Sciences (No: IR.LUMS.REC.1402.386). Additional administrative clearances were granted by the university's research vice-chancellery and the hospital management team. Patient privacy was carefully protected throughout the project; all extracted clinical data were fully anonymized, stored securely, and utilized exclusively for aggregate statistical reporting.

Participant Selection and Sample Size Estimation

The target population encompassed reproductive-aged women diagnosed with PCOS who visited the clinic during the study period. To ensure the validity of the real-world evidence, a consecutive sampling approach was utilized, wherein all eligible participants who met the strict clinical criteria and visited the clinic during the specified baseline period were systematically enrolled. To determine the necessary sample size, the standard single proportion formula was applied:

$$n = (Z^2 * p * (1 - p)) / d^2$$

The calculation was based on an expected target proportion (p) of clinical manifestations (such as hirsutism/menstrual dysfunction) of approximately 73.5% derived from baseline pilot data and existing regional literature, a 95% confidence interval (Z = 1.96), and a margin of error (d) set at 6%. This statistical framework yielded a final required sample size of approximately 211 participants.

Eligibility Criteria and Diagnostic Confirmation

The study enrolled women with a confirmed diagnosis of PCOS based on the revised

Rotterdam consensus criteria. Under these guidelines, patients were required to meet at least two of the following three definitive features: (1) clinical or biochemical hyperandrogenism (manifested as hirsutism and/or acne), (2) oligoovulation or anovulation (manifested as menstrual irregularities), and (3) polycystic ovarian morphology confirmed via pelvic ultrasound (defined as the presence of 12 or more follicles measuring 2–9 mm in diameter and/or increased ovarian volume >10 mL). All pelvic ultrasound examinations were performed by a single experienced radiologist using the same equipment to eliminate inter-observer variability.

Conversely, patients were excluded from the study if their medical records lacked baseline demographic profiles, essential physical measurements, or primary laboratory results. Out of the initially screened cases, 24 patients were excluded due to incomplete data tracking, resulting in a finalized sample of 211 fully completed cases for statistical evaluation.

Data Acquisition and Clinical Protocols

Data collection commenced immediately after securing institutional and ethical approvals. A structured, investigator-developed checklist served as the primary tool for data extraction. The primary investigator systematically reviewed the medical files and clinical charts of eligible participants at the gynecology clinic. Relevant demographic details, anthropometric measurements (including waist and hip circumferences), and baseline biochemical/hormonal parameters (FSH, LH, total testosterone, free testosterone, prolactin, and TSH) were carefully transcribed into the study database. To maintain dataset integrity, records with substantial data gaps or missing laboratory parameters were systematically excluded from the final analysis.

Statistical Evaluation

Statistical analysis was performed using SPSS software (Version 26.0, IBM Corp., Armonk, NY, USA). Continuous variables, including hormonal levels and anthropometric measurements, were assessed for normality utilizing the Kolmogorov-Smirnov test and are expressed as mean ± standard deviation (SD). Categorical variables, such as clinical phenotypes, age groups, and body mass index (BMI) categories, are presented as absolute frequencies and percentages (n, %). Proportions and categorical associations between the ultrasound-negative and ultrasound-positive groups were compared using Pearson's chi-square (χ^2) test. Mean values of continuous variables between the two independent study groups were compared using the Independent Samples t-test. A two-tailed p-value of less than 0.05 was considered statistically significant for all analyses.

Results

1. General and Clinical Characteristics of the Participants

A total of 211 women diagnosed with Polycystic Ovary Syndrome (PCOS) participated in this study. The mean age of the participants was 22.92 ±2.63 years, with an overall range of 16 to 29 years. The vast majority of the population (80.1%) were older than 20 years. Regarding body composition, only 27.5% of the women exhibited a normal Body Mass Index (BMI). Overweight was documented in 46.0% of the patients, while 26.5% were classified as obese. Active smoking was rare, with only 5.7% of the women reporting regular tobacco use. In terms of clinical features, menstrual irregularities represented the most prevalent clinical manifestation, affecting 78.2% of the cohort. Additionally, hirsutism was detected in 73.5% of the cases, and acne was reported by 47.9% of the participants. Overall, while all patients fulfilled the clinical diagnostic requirements under the Rotterdam consensus criteria, a positive

polycystic ovarian morphology via pelvic ultrasound was confirmed in 30.3% of the total study population (Table 1).

2. Baseline Anthropometric and Hormonal Profiles

The detailed baseline physical and endocrine parameters of the cohort are summarized in Table 2. Laboratory evaluations revealed marked hyperandrogenemia and a classic gonadotropin inversion across the study population. Specifically, the mean concentrations of free testosterone and total testosterone were 32.54 ± 40.66 pg/mL and 32.35 ± 42.95 ng/dL, respectively, underscoring significant biochemical androgen excess. Furthermore, a characteristically elevated Luteinizing Hormone (LH) to Follicle-Stimulating Hormone (FSH) profile was observed, with the mean LH level being substantially higher than the mean FSH level (13.50 ± 2.37 mIU/mL versus 5.49 ± 1.08 mIU/mL). This inverted LH/FSH ratio aligns precisely with the neuroendocrine alterations hallmark of PCOS. Regarding the anthropometric profile, physical measurements indicated an increased tendency toward central adiposity, evidenced by a mean waist circumference of 93.14 ± 9.30 cm and a mean hip circumference of 106.69 ± 5.89 cm (Table 2).

3. Association Between Ovarian Ultrasonographic Morphology and Hirsutism

The relationship between pelvic ultrasonographic findings and the presence of clinical hirsutism was evaluated. Among the 64 women presenting with positive polycystic ovarian morphology, 44 (68.8%) exhibited hirsutism, while 20 (31.2%) did not. Within the ultrasound-negative group (n = 147), clinical hirsutism was documented in 111 patients (75.5%) and was absent in 36 patients (24.5%). Pearson's chi-square test demonstrated that this distribution was not statistically significant (p = 0.316). These findings indicate that the presence of sonographic polycystic features does not significantly correlate with the manifestation of clinical hirsutism within this cohort, highlighting the heterogenous phenotype of the syndrome (Table 3).

Variable	Category	Frequency (n)	Percentage (%)
Age Group	≤20 years	42	19.9
	>20 years	169	80.1
Body Mass Index (BMI)	Normal (18.5–24.9 kg/m²)	58	27.5
	Overweight (25.0–29.9 kg/m²)	97	46.0
	Obese (≥30.0 kg/m²)	56	26.5
Smoking Status	Yes	12	5.7
	No	199	94.3
Acne	Present	101	47.9
	Absent	110	52.1
Menstrual Irregularities	Present	165	78.2
	Absent	46	21.8
PCO Ultrasound Appearance	Positive	64	30.3
	Negative	147	69.7
Hirsutism	Present	155	73.5
	Absent	56	26.5
Total	–	**211**	**100.0**

Table 1. Table 1. General and Clinical Characteristics of the Study Population. Data are presented as frequencies and percentages n (%) for categorical variables. The mean age of the population is 22.92 ± 2.63 years (range: 16–29 years). PCOS = Polycystic Ovary Syndrome; BMI = Body Mass Index.

Profile / Parameter	Unit	Minimum	Maximum	Mean	Standard Deviation (±SD)
Endocrine Profile					
FSH	mIU/mL	3.60	8.50	5.49	1.08
LH	mIU/mL	9.60	20.20	13.50	2.37
Total Testosterone	ng/dL	1.40	115.00	32.35	42.95
Free Testosterone	pg/mL	2.10	108.30	32.54	40.66
Prolactin	ng/mL	5.00	52.00	28.21	12.76
TSH	mIU/L	0.70	5.70	3.08	1.07
Anthropometric Profile					
Waist Circumference	cm	71.00	116.00	93.14	9.30
Hip Circumference	cm	90.00	122.00	106.69	5.89

Table 2. Baseline Anthropometric and Endocrine Profiles of the Participants. Data are expressed as mean ± standard deviation (SD) along with the absolute minimum and maximum ranges. LH = Luteinizing Hormone; FSH = Follicle-Stimulating Hormone; TSH = Thyroid-Stimulating Hormone; mIU = milli-international units; ng = nanograms; pg = picograms; mL = milliliter; dL = deciliter; L = liter; cm = centimeters.

Clinical Feature	Category	Ultrasound Negative (n = 147)	Ultrasound Positive (n = 64)	Total Cohort (n = 211)	P-value
Hirsutism	Absent, n (%)	36 (24.5%)	20 (31.2%)	56 (26.5%)	0.316*
	Present, n (%)	111 (75.5%)	44 (68.8%)	155 (73.5%)	
Total	n (%)	**147 (100.0%)**	**64 (100.0%)**	**211 (100.0%)**	

Table 3. Correlation Between Pelvic Ultrasound Morphology and Clinical Hirsutism. Data are expressed as absolute frequencies and percentages n (%). *P-value calculated using Pearson's Chi-Square test (significance level set at $p < 0.05$).

4. Association Between Ovarian Ultrasonographic Morphology and Acne

The study further evaluated the potential association between sonographic ovarian morphology and the presence of clinical acne. Among the 64 patients with confirmed positive ultrasound findings, acne was present in 35 women (54.7%) and absent in 29 women (45.3%). Within the ultrasound-negative cohort (n = 147), clinical acne was documented in 66 individuals (44.9%), while 81 patients (55.1%) did not present with this symptom. Statistical analysis using Pearson's chi-square test revealed no significant association between these two variables ($p = 0.231$). These results indicate that ultrasound-confirmed polycystic ovarian morphology is not significantly associated with the clinical presentation or prevalence of acne in this study population (Table 4).

Variable	Category	Ultrasound Negative (n = 147)	Ultrasound Positive (n = 64)	Total (n = 211)	P-value*
Acne	Absent, n (%)	81 (55.1%)	29 (45.3%)	110 (52.1%)	0.231
	Present, n (%)	66 (44.9%)	35 (54.7%)	101 (47.9%)	
Total	n (%)	147 (100.0%)	64 (100.0%)	211 (100.0%)	

Table 4. Association Between Pelvic Ultrasound Findings and Acne Presentation. Data are reported as counts and column percentages n (%). *P-value calculated using Pearson's Chi-Square test (significance level set at $p < 0.05$). The p-value ($p = 0.231$) shows that the occurrence of acne does not differ significantly between patients with positive and negative ultrasound findings.

5. Age Distribution Across Ovarian Ultrasonographic Groups

The distribution of age categories was evaluated in relation to pelvic ultrasonographic findings. Within the positive polycystic ovarian morphology group (n = 64), 11 women (17.2%) were aged 20 years or younger, whereas 53 patients (82.8%) were older than 20 years. Conversely, among the ultrasound-negative participants (n = 147), 31 individuals (21.1%) were 20 years or younger, and 116 women (78.9%) fell into the older than 20 categories. Statistical comparison using Pearson's chi-square test demonstrated that the age distribution did not differ significantly between the two sonographic groups (p = 0.519). These results indicate that age is not a significant determinant or confounding factor of ovarian ultrasound morphology in this study population (Table 5).

Variable	Category	Ultrasound Negative (n = 147)	Ultrasound Positive (n = 64)	Total (n = 211)	P-value*
Age	≤20 years, n (%)	31 (21.1%)	11 (17.2%)	42 (19.9%)	0.519
	>20 years, n (%)	116 (78.9%)	53 (82.8%)	169 (80.1%)	
Total	n (%)	147 (100.0%)	64 (100.0%)	211 (100.0%)	

Table 5. Distribution of Ovarian Ultrasound Findings Across Age Groups. Data are reported as frequencies and column percentages n (%). *P-value calculated using Pearson's Chi-Square test (significance level set at $p < 0.05$).

6. Distribution of Body Mass Index Relative to Ultrasonographic Status

The distribution of body weight categories was examined across the different pelvic ultrasonographic phenotypes. Among the 64 women presenting with positive polycystic ovarian morphology, 22 (34.4%) had a normal BMI, 29 (45.3%) were overweight, and 13 (20.3%) were classified as obese. Within the ultrasound-negative group (n = 147), a normal BMI was recorded in 36 women (24.5%), 68 (46.3%) were overweight, and 43 (29.3%) were obese. The Pearson chi-square test demonstrated no statistically significant difference in BMI distribution between the two sonographic groups (p = 0.230). These statistical outcomes indicate that body weight and BMI categories are not significantly associated with ultrasound-confirmed polycystic ovarian morphology within this study population (Table 6).

BMI Category (kg/m²)	Category Detail	Ultrasound Negative (n = 147)	Ultrasound Positive (n = 64)	Total (n = 211)	P-value*
Normal	18.6–24.9, n (%)	36 (24.5%)	22 (34.4%)	58 (27.5%)	0.230
Overweight	25.0–29.9, n (%)	68 (46.3%)	29 (45.3%)	97 (46.0%)	
Obese	≥30.0, n (%)	43 (29.3%)	13 (20.3%)	56 (26.5%)	
Total	n (%)	147 (100.0%)	64 (100.0%)	211 (100.0%)	

Table 6. Distribution of Body Mass Index Categories Relative to Ultrasound Status. Data are reported as counts and column percentages n (%). BMI = Body Mass Index. *P-value evaluated using the Chi-square test of independence (significance level set at $p < 0.05$).

7. Comparative Analysis of Hormonal and Anthropometric Markers

Hormonal and physical markers were compared directly between the ultrasound-negative and ultrasound-positive phenotypes. As detailed in Table 7, statistically highly significant differences were observed in key endocrine parameters. Women with positive polycystic ovarian morphology via ultrasound demonstrated significantly elevated gonadotropin and androgen levels. Specifically, the mean LH level was 15.27 ± 2.62 mIU/mL in the ultrasound-positive group, compared to 12.73 ± 1.78 mIU/mL in the ultrasound-negative group ($p < 0.001$). Similarly, FSH levels were significantly higher in the ultrasound-positive cohort (6.79 ± 0.95 versus 4.93 ± 0.49 mIU/mL, $p < 0.001$). Total testosterone and free testosterone were also profoundly elevated in the ultrasound-positive patients ($p < 0.001$ for both markers). In sharp contrast, metabolic and anthropometric profiles showed no statistically significant variations between the two groups. Parameters such as TSH ($p = 0.926$) and prolactin ($p = 0.997$) remained nearly identical. Finally, waist and hip circumferences did not present any meaningful statistical discrepancies relative to the ultrasound categories, indicating that overall central adiposity was uniformly distributed across both phenotypes (Table 7).

Discussion

Our findings demonstrate that while hormonal imbalances and elevated androgen markers are markedly pronounced in patients with positive ultrasound findings, typical clinical symptoms like hirsutism and acne do not strictly correlate with ovarian morphology. This clear dissociation suggests that peripheral tissue sensitivity to circulating androgens plays a more decisive role in expressing these symptoms than the structural appearance of the ovaries themselves. Furthermore, the lack of a significant link between body mass index and ultrasound outcomes implies that weight status alone does not dictate the physical manifestation of polycystic ovaries in this population. These patterns emphasize that ovarian appearance via ultrasound is a variable feature rather than a reliable indicator of overall metabolic or clinical severity. Consequently, reliance on imaging alone may lead to underestimating the metabolic risks in patients who present with normal ovarian structures but severe endocrine disruption.

Profile	Parameter	Ultrasound Negative (n = 147)	Ultrasound Positive (n = 64)	P-value*
Hormonal Profile (Mean ± SD)	FSH (mIU/mL)	4.93 ± 0.49	6.79 ± 0.95	< 0.001
	LH (mIU/mL)	12.73 ± 1.78	15.27 ± 2.62	< 0.001
	Total Testosterone (ng/dL)	4.20 ± 1.28	97.02 ± 6.90	< 0.001
	Free Testosterone (pg/mL)	5.86 ± 4.46	93.82 ± 2.09	< 0.001
	Prolactin (ng/mL)	28.21 ± 12.60	28.20 ± 13.22	0.997
	TSH (mIU/L)	3.08 ± 1.07	3.07 ± 1.08	0.926
Anthropometric Profile (Mean ± SD)	Waist Circumference (cm)	93.06 ± 8.93	93.31 ± 10.16	0.861
	Hip Circumference (cm)	106.52 ± 5.88	107.07 ± 5.95	0.532

Table 7. Comparative Analysis of Hormonal and Anthropometric Markers Based on Ultrasound Status. Data are expressed as mean ± standard deviation (SD). LH = Luteinizing Hormone; FSH = Follicle-Stimulating Hormone; TSH = Thyroid-Stimulating Hormone. *P-value evaluated using the Independent Samples t-test (statistically significant at $p < 0.05$).

This striking contrast highlights the critical necessity for an integrated clinical approach that moves beyond traditional diagnostic boundaries to ensure patient safety and optimize care outcomes. Pelvic ultrasonography alone is not recommended for diagnosing PCOS during adolescence because of low specificity and overlap with normal pubertal physiology (15). Clinical evidence shows that metabolic risk aligns more closely with hyperandrogenism and ovulatory dysfunction than with ovarian morphology (16). Consequently, patients who do not exhibit classic polycystic ovarian morphology may still present with significant underlying metabolic and endocrine abnormalities that could be under-recognized if diagnostic emphasis is placed predominantly on imaging findings (16). These findings support the need for a broader evaluative model capable of capturing the multisystem nature of PCOS (17). Under this framework, endocrine markers such as gonadotropin dynamics and total or free testosterone levels are considered clinically as important as ultrasound-based ovarian morphology in the evaluation of PCOS (18). By integrating intelligent detection frameworks and standardized feature taxonomies, clinicians can enhance the accuracy and efficiency of PCOS diagnosis, thereby supporting earlier identification of patients at risk (19). Furthermore, a comprehensive assessment of PCOS, encompassing metabolic, reproductive, and psychological domains improves clinical decision-making through structured screening and evaluation tools within an integrated care framework (20). Our results specifically revealed that patients with positive ultrasound findings exhibit significantly higher LH, FSH, and testosterone levels compared to the ultrasound-negative cohort (Table 7), independent of body weight or dermatological status. Standardized longitudinal assessment in PCOS is effective for tracking temporal changes and treatment response, but baseline or dynamic clinical features do not reliably predict individual phenotypic transitions (21). For instance, patients presenting with marked biochemical hyperandrogenism despite normal ovarian morphology may warrant earlier metabolic and cardiovascular risk evaluation (22). This is consistent with guidelines recommending lifestyle interventions as first-line therapy for PCOS, reflecting its metabolic and psychological dimensions beyond reproduction (23). Such a targeted approach facilitates the identification and longitudinal follow-up of individuals at increased risk, while reducing unnecessary investigations through adherence to evidence-based diagnostic criteria and the avoidance of non-recommended imaging in adolescent populations (24).

The heterogeneous nature of polycystic ovary syndrome is reflected in the variable expression of clinical symptoms, metabolic phenotype across different body mass index categories, and ovarian morphological features, highlighting its complex endocrine and multifactorial pathophysiology (2). Given this high heterogeneity and multisystem involvement, clinical management should take into account multiple reproductive, metabolic, and cardiovascular risk factors rather than relying on single diagnostic features (25). The evidence strongly supports evidence-based therapeutic strategies for managing PCOS, with particular emphasis on lifestyle interventions and pharmacological treatments (26). In addition, the literature highlights the importance of multidisciplinary care in improving adherence to lifestyle modification and optimizing overall clinical outcomes (26). Future quality improvement efforts should prioritize the development of standardized and validated screening tools, including risk prediction models, to support more effective management of diverse patient populations (13).

Conclusion

In conclusion, this study demonstrates a pronounced divergence among the structural, clinical, and biochemical features of Polycystic Ovary Syndrome (PCOS). While pelvic ultrasound findings show no significant correlation with BMI, waist circumference, hirsutism, or acne, they are systematically coupled with severe hyperandrogenism and gonadotropin imbalances. These findings reveal a critical diagnostic blind spot in clinical practice. Relying solely on peripheral tissue responses or pelvic imaging may inadvertently mask severe underlying metabolic disruptions in patients with normal ovarian morphology. To improve healthcare delivery, practitioners must transition from symptom-driven approaches toward a multidimensional screening framework. Implementing a dual assessment protocol is essential. Within this model, comprehensive biochemical profiling, specifically tracking testosterone spikes and LH/FSH inversions, must receive equal diagnostic weight to imaging. Within resource-limited regional centers, this phenotype stratification serves as an objective framework to optimize institutional resource allocation, ensuring that high-risk patients are immediately accelerated into early preventive metabolic therapies.

Implications for Patient Care

Integrating these insights into clinical workflows enables healthcare providers to deliver personalized and targeted medicine. Instead of utilizing uniform treatment plans, patients should be precisely stratified based on their endocrine profiles rather than outward cutaneous markers. Individuals identified with severe biochemical vulnerabilities must be immediately integrated into early preventive care models, including standardized metabolic screenings and evidence-based lifestyle interventions to mitigate long-term cardiovascular risks. Furthermore, in regional referral settings, this objective risk stratification optimizes institutional resource allocation by reducing reliance on repeated, unnecessary pelvic imaging for stable phenotypes, thereby minimizing the financial burden on patients and improving patient-clinician communication.

Conflict of Interest

The authors declare that they have no conflict of interest.

Author Contributions

All authors contributed to the conception and design of the study, literature search, data interpretation, drafting of the manuscript, and critical revision of the article. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Ethics Statement

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Data Availability Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Reference

1. Dokras A. Polycystic ovary syndrome in 2025—insights and innovations. Fertility and Sterility. 2025;124(5, Part 2):907-9. https://doi.org/10.1016/j.fertnstert.2025.09.025.

2. Lentscher JA, Decherney AH. Clinical Presentation and Diagnosis of Polycystic Ovarian Syndrome. Clin Obstet Gynecol. 2021;64(1):3-11.

https://doi.org/10.1097/GRF.0000000000000563
.

3. Naeem I, Zehra A, Rehman F, Hussain A, Hussain A, Hussain N, et al. polycystic ovarian syndrome a risk factor for non-communicable diseases: insights into recent research and prevention approaches. J Ovarian Res. 2025;18(1):219. https://doi.org/10.1186/s13048-025-01741-z.

4. Prosperi S, Chiarelli F. Insulin resistance, metabolic syndrome and polycystic ovaries: an intriguing conundrum. Front Endocrinol (Lausanne). 2025;16:1669716. https://doi.org/10.3389/fendo.2025.1669716.

5. Naveed A, Shah R, Bernier A, Muradova V, Savgan Gurol E, Cree M, et al. Metabolic Implications of PCOS. Curr Pediatr Rep. 2025;13. https://doi.org/10.1007/s40124-025-00364-8.

6. Louwers YV, Laven JSE. Characteristics of polycystic ovary syndrome throughout life. Ther Adv Reprod Health. 2020;14:2633494120911038. https://doi.org/10.1177/2633494120911038.

7. Gao X, Zhao S, Du Y, Yang Z, Tian Y, Zhao J, et al. Data-driven subtypes of polycystic ovary syndrome and their association with clinical outcomes. Nature Medicine. 2025;31(12):4214-24. https://doi.org/10.1038/s41591-025-03984-1.

8. Teede HJ, Tay CT. Polycystic ovary syndrome: A female endocrine metabolic reproductive syndrome in the context of rising global obesity. Global Reproductive Health. 2025;10(4):e0122. https://doi.org/10.1097/GRH.0000000000000122.

9. Liu T, Wang Q, Huang W, Tan J, Liu D, Pei T, et al. Anthropometric indices to predict insulin resistance in women with polycystic ovary syndrome in China. Reproductive BioMedicine Online. 2019;38(1):101-7. https://doi.org/10.1016/j.rbmo.2018.10.001.

10. Babar B, Farooq G, Manzoor M, Khan H. Clinical and biochemical profile in adolescent and adult polycystic ovary syndrome patients. Pak J Med Sci. 2025;41(9):2518-22. https://doi.org/10.1016/j.rbmo.2018.10.00110.12669/pjms.41.9.12228.

11. Parker J, O'Brien C, Hawrelak J, Gersh FL. Polycystic Ovary Syndrome: An Evolutionary Adaptation to Lifestyle and the Environment. Int J Environ Res Public Health. 2022;19(3). https://doi.org/10.3390/ijerph19031336.

12. VanHise K, Wang ET, Norris K, Azziz R, Pisarska MD, Chan JL. Racial and ethnic disparities in polycystic ovary syndrome. Fertil Steril. 2023;119(3):348-54. https://doi.org/10.1016/j.fertnstert.2023.01.031.

13. Sacca L, Lobaina D, Knopf E, Burgoa S, Jimenez S, Okwaraji G, et al. Assessment of the Validity and Quality of Polycystic Ovarian Syndrome (PCOS) Screening Tools Available for Women Globally: A Systematic Review. Clin Pract. 2024;14(5):1625-49. https://doi.org/10.3390/clinpract14050131.

14. Agapova SE, Cameo T, Sopher AB, Oberfield SE. Diagnosis and challenges of polycystic ovary syndrome in adolescence. Semin Reprod Med. 2014;32(3):194-201. https://doi.org/10.1055/s-0034-1371091.

15. Peña AS, Witchel SF, Hoeger KM, Oberfield SE, Vogiatzi MG, Misso M, et al. Adolescent polycystic ovary syndrome according to the international evidence-based guideline. BMC Med. 2020;18(1):72. https://doi.org/10.1186/s12916-020-01516-x.

16. Christ JP, Cedars MI. Current Guidelines for Diagnosing PCOS. Diagnostics (Basel). 2023;13(6). https://doi.org/10.3390/diagnostics13061113.

17. Teede HJ, Moran LJ, Morman R, Gibson M, Dokras A, Berry L, et al. Polycystic ovary syndrome perspectives from patients and health professionals on clinical features, current name, and renaming: a longitudinal international online survey. EClinicalMedicine. 2025;84:103287. https://doi.org/10.1016/j.eclinm.2025.103287.

18. Witchel SF, Oberfield SE, Peña AS. Polycystic Ovary Syndrome: Pathophysiology,

Presentation, and Treatment With Emphasis on Adolescent Girls. J Endocr Soc. 2019;3(8):1545-73. https://doi.org/10.1210/js.2019-00078.
19. Li M, He Z, Shi L, Lin M, Li M, Cheng Y, et al. Intelligent detection for Polycystic Ovary Syndrome (PCOS): Taxonomy, datasets and detection tools. Computational and Structural Biotechnology Journal. 2025;27:1578-99. https://doi.org/10.1016/j.csbj.2025.04.011.
20. Almhmoud H, Alatassi L, Baddoura M, Sandouk J, Alkayali MZ, Najjar H, et al. Polycystic ovary syndrome and its multidimensional impacts on women's mental health: A narrative review. Medicine (Baltimore). 2024;103(25):e38647. https://doi.org/10.1097/MD.0000000000038647.
21. Carter FE, Jarrett BY, Oldfield AL, Vanden Brink H, Kim JY, Lujan ME. Impact of Hypocaloric Dietary Intervention on Phenotypic Presentations of Polycystic Ovary Syndrome (PCOS). Nutrients. 2025;17(13):2223. https://doi.org/10.3390/nu17132223.
22. Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2013;98(12):4565-92. https://doi.org/10.1210/jc.2013-2350.
23. Cowan S, Lim S, Alycia C, Pirotta S, Thomson R, Gibson-Helm M, et al. Lifestyle management in polycystic ovary syndrome - beyond diet and physical activity. BMC Endocr Disord. 2023;23(1):14. https://doi.org/10.1186/s12902-022-01208-y.
24. Peña AS, Witchel SF, Boivin J, Burgert TS, Ee C, Hoeger KM, et al. International evidence-based recommendations for polycystic ovary syndrome in adolescents. BMC Med. 2025;23(1):151. https://doi.org/10.1186/s12916-025-03901-w.
25. Palomba S, Santagni S, Falbo A, La Sala GB. Complications and challenges associated with polycystic ovary syndrome: current perspectives. Int J Womens Health. 2015;7:745-63. https://doi.org/10.2147/IJWH.S70314.
26. Akre S, Sharma K, Chakole S, Wanjari MB. Recent Advances in the Management of Polycystic Ovary Syndrome: A Review Article. Cureus. 2022;14(8):e27689. https://doi.org/10.7759/cureus.27689.