

Clinical and Diagnostic Features of Polycystic Ovary Syndrome in Reproductive-Age Women: A Comprehensive Assessment of Hormonal and Metabolic Factors

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Abstract

Background: Polycystic ovary syndrome (PCOS) combines reproductive dysfunction with heterogeneous androgenic and metabolic abnormalities. Diagnostic assessment must distinguish defining features from associated risks while excluding disorders that produce similar presentations. This illustrative investigation examined how clinical findings, hormonal measurements, and metabolic indicators contribute to comprehensive evaluation.

Methods: A synthetic cross-sectional dataset represented 120 reproductive-age women, comprising 80 PCOS cases and 40 age-matched controls. Case classification followed Rotterdam criteria after simulated exclusion of alternative diagnoses. Variables included menstrual patterns, hirsutism, ovarian morphology, total testosterone, sex hormone-binding globulin, body mass index, glucose tolerance, and lipid measurements. Analyses were descriptive; no actual participants, recruitment, or laboratory testing were involved.

Results: Among simulated cases, 64 women had ovulatory dysfunction, 60 had clinical or biochemical hyperandrogenism, and 56 had polycystic ovarian morphology. Median total testosterone was 1.9 nmol/L in cases and 1.1 nmol/L in controls. Median body mass index was 28.4 versus 24.6 kg/m². Impaired glucose tolerance occurred in 20 cases and four controls; metabolic syndrome occurred in 24 and four, respectively. These contrasts were not adjusted for adiposity.

Conclusions: The constructed findings illustrate why PCOS evaluation should integrate reproductive and androgenic features with independent cardiometabolic screening. They cannot establish prevalence, effect sizes, or causal relationships. Prospective research with standardized measurements and appropriate confounder adjustment is required to validate comparable patterns in clinical populations.

Keywords: polycystic ovary syndrome, hyperandrogenism, ovulatory dysfunction, insulin resistance, glucose tolerance, metabolic syndrome, diagnostic assessment

1. Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting reproductive-age women,

with manifestations that extend beyond menstrual irregularity and androgen excess. Its clinical presentation varies substantially across individuals and populations, reflecting differences in age, adiposity, genetic susceptibility, and environmental influences. Women may present with oligomenorrhea, anovulatory infertility, hirsutism, acne, or concerns related to weight and metabolic health. This heterogeneity can complicate recognition, particularly when symptoms are mild or when reproductive and metabolic abnormalities do not occur together. A comprehensive assessment is therefore important for characterizing disease expression and identifying clinically relevant comorbidities.

In adults, PCOS is generally diagnosed by the presence of at least two of three features: ovulatory dysfunction, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology, after exclusion of alternative explanations. Depending on the diagnostic framework and clinical context, appropriately validated anti-Müllerian hormone measurements may provide an alternative to ultrasonography for defining polycystic ovarian morphology in adults. Neither ovarian morphology nor an isolated hormonal abnormality is sufficient to establish the diagnosis. Thyroid dysfunction, hyperprolactinemia, and nonclassic congenital adrenal hyperplasia are among the conditions requiring consideration, while rapidly progressive androgenic symptoms warrant evaluation for less common causes of androgen excess. Diagnostic interpretation also requires attention to medication use, menstrual history, and the limitations of laboratory assays.

Hormonal disturbances in PCOS commonly involve increased ovarian androgen production and altered hypothalamic–pituitary–ovarian signaling. Insulin resistance and compensatory hyperinsulinemia may contribute to androgen excess and reduced sex hormone-binding globulin concentrations, thereby increasing circulating free androgen availability. However, these abnormalities are not uniformly present, and their severity cannot be inferred reliably from appearance or body mass index alone. Total and free testosterone assessment can support biochemical characterization, whereas gonadotropin concentrations and the luteinizing hormone-to-follicle-stimulating hormone ratio are not independently diagnostic. Variation in assay performance, sampling conditions, and reference intervals further complicates comparisons between patients and studies.

Metabolic features associated with PCOS include central adiposity, dysglycemia, and adverse lipid profiles, although their frequency and magnitude differ across phenotypes. Excess adiposity may amplify these disturbances, but women without overweight can also exhibit metabolic abnormalities. Concurrent evaluation of anthropometric measures, blood pressure, glycemic status, and lipid parameters may therefore provide information not captured by reproductive symptoms alone. Understanding how these measures relate to clinical and hormonal findings could support more individualized risk assessment without assuming that all affected women share the same metabolic profile.

Against this background, the present study aims to characterize the clinical and diagnostic features of PCOS in reproductive-age women through an integrated assessment of reproductive symptoms, androgen-related findings, hormonal measurements, and metabolic parameters. It also seeks to examine associations among these domains and describe variation across diagnostic phenotypes. This approach is intended to clarify patterns of presentation and inform comprehensive clinical evaluation, while recognizing that observed associations do not establish causal relationships.

2. Materials and Methods

This analytical cross-sectional study will recruit reproductive-age women attending gynecology and endocrinology outpatient clinics at a tertiary-care hospital over 12 months. The target sample will comprise 240 participants aged 18–40 years: 120 women with polycystic ovary syndrome (PCOS) and 120 comparison participants without PCOS. Consecutive eligible patients will be approached, and comparison participants will be recruited from the same clinical population without matching on body mass index (BMI), allowing adiposity-related differences to be examined. The study will assess clinical, hormonal, and metabolic characteristics and their associations with PCOS phenotype.

PCOS will be diagnosed using modified Rotterdam criteria, requiring at least two of the following after exclusion of alternative causes: oligo-anovulation, clinical or biochemical hyperandrogenism, and

polycystic ovarian morphology. Oligo-anovulation will be defined by menstrual cycles shorter than 21 or longer than 35 days, or fewer than eight cycles annually. Comparison participants must have regular cycles and no clinical or biochemical hyperandrogenism. Isolated polycystic ovarian morphology will not independently exclude them, but will be documented for sensitivity analysis.

Exclusion criteria will include pregnancy, breastfeeding, menopause, known diabetes mellitus, significant hepatic or renal disease, and use of hormonal contraception, antiandrogens, glucocorticoids, or insulin-sensitizing medication within the preceding three months. Thyroid dysfunction, hyperprolactinemia, and nonclassic congenital adrenal hyperplasia will be evaluated using thyroid-stimulating hormone, prolactin, and early-morning 17-hydroxyprogesterone measurements, with confirmatory testing when indicated. Rapidly progressive virilization or features suggesting Cushing syndrome will prompt targeted assessment before eligibility is determined. Prescribed treatment will not be withdrawn solely for research participation.

A standardized questionnaire will capture menstrual history, reproductive history, medication exposure, smoking, physical activity, and family history of diabetes. Trained investigators will measure height, weight, waist circumference, and blood pressure using standardized procedures. BMI will be calculated as weight divided by squared height. Hirsutism will be graded using the modified Ferriman–Gallwey score, applying a prespecified ethnicity-appropriate threshold. Acne, androgen-related hair loss, and acanthosis nigricans will also be recorded.

Blood samples will be obtained between 08:00 and 10:00 after an overnight fast, preferably during cycle days 2–5. Women with prolonged amenorrhea may undergo sampling on any day after pregnancy exclusion; cycle timing will be recorded. Measurements will include total testosterone, sex hormone-binding globulin, dehydroepiandrosterone sulfate, luteinizing hormone, follicle-stimulating hormone, estradiol, fasting glucose, insulin, and lipids. Total testosterone will be measured using liquid chromatography–tandem mass spectrometry, and free testosterone will be calculated using testosterone, sex hormone-binding globulin, and albumin. Biochemical hyperandrogenism will be defined against validated laboratory reference intervals. Internal quality controls will accompany each assay batch.

All participants will undergo a 75-g oral glucose tolerance test with fasting and two-hour plasma glucose measurements. Insulin resistance will be estimated using the homeostasis model assessment, calculated as fasting insulin multiplied by fasting glucose in mmol/L and divided by 22.5; this index will be treated as a surrogate rather than a diagnostic standard. Transvaginal ultrasonography, when acceptable, will assess follicle number and ovarian volume using a transducer of at least 8 MHz. Polycystic morphology will be defined as at least 20 follicles per ovary or ovarian volume of at least 10 mL, excluding dominant follicles and cysts. Transabdominal examinations will rely on ovarian volume.

The sample provides approximately 80% power to detect a standardized between-group difference of 0.4 with two-sided alpha of 0.05 and modest incomplete data. The primary comparison will concern insulin resistance; multivariable regression will adjust for age and BMI. Secondary analyses will examine hormonal profiles and PCOS phenotypes, with multiplicity addressed using false-discovery-rate control. Missingness will be described, with multiple imputation considered when appropriate. Institutional ethics approval and written informed consent will precede recruitment; data will be coded and stored securely.

3. Results

The following results describe an illustrative, simulated case–control dataset rather than observed patient data. The analysis included 240 reproductive-age women: 120 meeting Rotterdam criteria for polycystic ovary syndrome (PCOS) and 120 controls without PCOS. Mean age was similar between groups (27.8 ± 5.4 versus 28.3 ± 5.7 years). Women with PCOS had higher mean body mass index (BMI; 28.6 ± 6.1 versus 25.2 ± 4.8 kg/m²) and waist circumference (91.4 ± 13.8 versus 82.6 ± 11.5 cm). Obesity, defined as BMI ≥ 30 kg/m², was present in 45 women with PCOS (37.5%) and 22 controls (18.3%). Current hormonal contraceptive use, pregnancy, and alternative endocrine causes of ovulatory dysfunction or androgen excess were exclusion criteria.

Ovulatory dysfunction was identified in 98 women with PCOS (81.7%), predominantly as infrequent

menstrual cycles. Seventy-six participants (63.3%) reported cycles longer than 35 days, while 22 (18.3%) reported amenorrhea lasting at least 90 days. Hirsutism, assessed using a prespecified modified Ferriman–Gallwey score threshold of 8, was observed in 74 women with PCOS (61.7%) and 11 controls (9.2%). Acne was recorded in 48 (40.0%) and 21 (17.5%), respectively, while female-pattern hair loss was less frequent, affecting 19 (15.8%) and eight (6.7%). These findings overlapped within participants and were not treated as mutually exclusive categories.

Clinical or biochemical hyperandrogenism was present in 108 women with PCOS (90.0%). Median total testosterone was higher in the PCOS group than in controls (1.9 [interquartile range, 1.4–2.6] versus 1.1 [0.8–1.5] nmol/L). Sex hormone-binding globulin concentrations were lower (32 [23–45] versus 51 [38–67] nmol/L), accompanied by a higher free androgen index (5.9 [3.6–9.2] versus 2.2 [1.4–3.5]). Total testosterone was measured using liquid chromatography–tandem mass spectrometry, and biochemical hyperandrogenism was classified using laboratory-specific reference limits. Elevated total testosterone or free androgen index was identified in 91 cases (75.8%); 57 also had hirsutism. Dehydroepiandrosterone sulfate showed substantial between-group overlap, indicating that adrenal androgen elevation was not a consistent feature.

Median luteinizing hormone concentration was 8.7 [5.6–12.4] IU/L in women with PCOS and 5.3 [3.8–7.2] IU/L in controls. Follicle-stimulating hormone concentrations were broadly comparable. The median luteinizing hormone-to-follicle-stimulating hormone ratio was 1.6 [1.0–2.4] versus 0.9 [0.7–1.3], but considerable overlap limited its discriminatory value. Sampling occurred during the early follicular phase where feasible; cycle timing could not be standardized fully in participants with prolonged amenorrhea. Thyroid-stimulating hormone and prolactin concentrations remained within the prespecified eligibility ranges in both groups.

Standardized transvaginal ultrasonography identified polycystic ovarian morphology in 92 women with PCOS (76.7%) and 17 controls (14.2%). Morphology was defined by a follicle number per ovary 20 or ovarian volume 10 mL in either ovary, excluding dominant follicles and cysts. Median ovarian volume was 11.8 [9.2–14.7] mL in cases and 7.1 [5.8–8.9] mL in controls. Phenotype A, comprising hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, accounted for 58 cases (48.3%). Phenotypes B, C, and D accounted for 28 (23.3%), 22 (18.3%), and 12 (10.0%), respectively.

Metabolic differences were apparent despite relatively modest differences in fasting glucose. Median fasting insulin was 14.8 [9.7–21.6] versus 8.6 [5.9–12.4] μ IU/mL, and median homeostasis model assessment of insulin resistance was 3.3 [2.1–5.0] versus 1.8 [1.2–2.8]. During a 75-g oral glucose tolerance test, 22 women with PCOS (18.3%) and six controls (5.0%) met criteria for prediabetes. Four cases (3.3%) and one control (0.8%) had glucose values in the diabetes range; these single-visit findings required confirmation. Triglycerides were higher and high-density lipoprotein cholesterol lower in the PCOS group, whereas low-density lipoprotein cholesterol showed greater overlap. Metabolic syndrome was identified in 29 cases (24.2%) and 12 controls (10.0%).

In logistic regression adjusted for age and BMI, PCOS status remained associated with dysglycemia, combining prediabetes and diabetes-range results (adjusted odds ratio, 2.8; 95% confidence interval, 1.1–7.1). Within the PCOS group, waist circumference correlated positively with insulin resistance (Spearman $r = 0.49$), while sex hormone-binding globulin correlated inversely ($r = -0.41$). Women with hyperandrogenic phenotypes generally had less favorable metabolic profiles than those with phenotype D, although the small nonhyperandrogenic subgroup limited precision. Phenotype comparisons were exploratory and did not establish independent differences after accounting for adiposity. Overall, abnormalities varied substantially across individuals, and neither ovarian morphology nor any single hormonal measurement consistently identified those with concurrent metabolic impairment.

4. Discussion

Polycystic ovary syndrome (PCOS) is best understood as a heterogeneous reproductive and metabolic disorder rather than a condition defined by a single hormonal abnormality or imaging feature. A comprehensive assessment of reproductive-age women therefore requires integration of menstrual history, clinical androgen excess, biochemical measurements, and metabolic risk indicators. These domains provide complementary information, but none independently captures the full clinical

spectrum. The principal implication of this integrated approach is that diagnostic classification and risk assessment should be considered related but distinct tasks: establishing PCOS does not, by itself, indicate the severity of metabolic dysfunction or determine an individual patient's reproductive prognosis.

Menstrual irregularity remains an important clinical entry point because it can indicate persistent ovulatory dysfunction. Nevertheless, cycle history requires careful interpretation in relation to age, years since menarche, medication exposure, and recent changes in weight or health. Apparently regular bleeding does not invariably establish ovulation, while irregular cycles can arise from conditions unrelated to PCOS. When ovulatory status is clinically relevant and uncertain, appropriately timed progesterone measurement may provide additional information. Diagnostic assessment should also exclude alternative explanations, including pregnancy when applicable, thyroid dysfunction, hyperprolactinemia, and nonclassic congenital adrenal hyperplasia. Rapidly progressive androgenic symptoms or marked biochemical androgen excess warrant investigation for less common causes rather than automatic attribution to PCOS.

Hyperandrogenism represents another major diagnostic domain, although clinical and biochemical manifestations may not align closely. Hirsutism reflects cumulative androgen exposure and tissue sensitivity, whereas circulating androgen concentrations describe a more immediate biochemical state. Ethnic variation, cosmetic hair removal, and observer-dependent scoring can further complicate clinical assessment. Acne and female-pattern hair loss may support evaluation but are less specific when present without other features. Biochemical interpretation should account for assay performance, laboratory reference intervals, and sex hormone-binding globulin concentrations. Reliable total testosterone measurement, preferably using a validated mass spectrometry method, together with an appropriate assessment of free testosterone, is more informative than reliance on an isolated, potentially imprecise androgen result.

Gonadotropin patterns can provide physiological context but should not be treated as diagnostic requirements. Although altered luteinizing hormone secretion may contribute to ovarian androgen production in some women, the luteinizing hormone-to-follicle-stimulating hormone ratio varies with cycle timing, sampling conditions, and phenotype. A normal ratio does not exclude PCOS, and an elevated ratio does not establish it. Similarly, ovarian morphology requires interpretation within the wider clinical picture. Follicle counts and ovarian volume depend on imaging technology, examination route, and operator expertise. Polycystic ovarian morphology can occur in otherwise healthy women, while its absence does not exclude PCOS when other diagnostic criteria are fulfilled.

Anti-Müllerian hormone may add information about follicular excess in adults, but its interpretation is constrained by assay differences, age, and other biological influences. Within an appropriate adult diagnostic algorithm, it may serve as an alternative to ultrasound for defining polycystic ovarian morphology, rather than as a standalone test for PCOS. Using both measurements routinely for the same purpose may increase the risk of overdiagnosis. When menstrual irregularity and hyperandrogenism are already established, additional morphology testing may not be necessary. These considerations reinforce the importance of applying explicit diagnostic criteria rather than allowing the availability of a particular test to drive classification.

Metabolic assessment is essential because reproductive presentation alone may underestimate broader health risk. Adiposity can aggravate insulin resistance and androgen excess, yet women without overweight may also have impaired glucose regulation or adverse lipid profiles. Body mass index should therefore not function as the sole gatekeeper for metabolic evaluation. Waist circumference can provide complementary information about central adiposity, although neither measure directly quantifies insulin sensitivity. An oral glucose tolerance test generally offers greater sensitivity for detecting dysglycemia in PCOS than fasting glucose alone. Fasting insulin concentrations and derived insulin resistance indices may support research analyses, but assay variability and the absence of universally applicable thresholds limit their routine diagnostic value.

The relationship between hormonal and metabolic abnormalities is biologically plausible but should not be interpreted as unidirectional. Hyperinsulinemia can promote ovarian androgen production and suppress hepatic sex hormone-binding globulin synthesis, potentially increasing free androgen

availability. Conversely, androgen excess may influence adipose tissue distribution and insulin action. Cross-sectional associations cannot establish the temporal sequence or relative contribution of these pathways. Age, adiposity, family history, ethnicity, physical activity, and medication use may also influence both endocrine and metabolic measurements. Multivariable adjustment can reduce measured confounding, but residual confounding remains possible, particularly when lifestyle exposures or treatment histories are incompletely characterized.

Several methodological considerations are important when interpreting a comprehensive clinical assessment. Clinic-based recruitment may overrepresent women with troublesome menstrual symptoms, infertility, or visible androgen excess and may not reflect community presentations. Hormonal measurements obtained at inconsistent cycle stages, or during hormonal treatment, can introduce additional variability. Phenotype comparisons require adequate subgroup sizes and attention to multiple testing; otherwise, apparent differences may be unstable. Associations should therefore be interpreted through effect sizes and uncertainty, not statistical significance alone. Without longitudinal follow-up, diagnostic characteristics cannot establish subsequent diabetes, cardiovascular events, fertility outcomes, or responses to treatment.

Overall, an integrated evaluation supports individualized care while avoiding the assumption that all women with PCOS share the same risk profile. Counseling should address menstrual health, fertility intentions, androgen-related symptoms, and metabolic prevention without making weight the exclusive focus. Psychological well-being and patient priorities also deserve attention because symptom burden may not parallel laboratory abnormalities. Future longitudinal studies using standardized assays, consistent diagnostic definitions, and diverse populations could clarify which combinations of clinical, hormonal, and metabolic features best predict meaningful outcomes and justify tailored surveillance.

5. Conclusion

This comprehensive assessment highlights the clinical and diagnostic heterogeneity of polycystic ovary syndrome in reproductive-age women and supports an integrated approach to evaluating reproductive, androgenic, and metabolic features. Menstrual irregularity, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology remain important diagnostic considerations; however, no single finding adequately captures the full spectrum of the syndrome. Diagnostic interpretation should therefore incorporate clinical history, standardized examination, appropriately selected laboratory testing, and exclusion of alternative causes of ovulatory dysfunction or androgen excess.

Hormonal and metabolic assessments provide complementary information rather than interchangeable evidence of disease. Androgen measurements require careful interpretation in relation to assay performance, medication exposure, and relevant clinical characteristics. Similarly, ovarian morphology should not be interpreted in isolation, particularly when other diagnostic features are absent. Metabolic abnormalities, including dysglycemia and adverse lipid profiles, may coexist with reproductive manifestations and warrant attention even when overt symptoms are limited. Although adiposity can influence metabolic risk, body size alone should not determine whether cardiometabolic assessment is undertaken. Measures of insulin resistance may contribute to research characterization but should not be treated as mandatory diagnostic criteria.

The clinical implications favor individualized assessment and management guided by symptom burden, reproductive goals, and established metabolic risk factors. Clear communication about diagnostic uncertainty and variation in phenotype may improve shared decision-making and support appropriate follow-up without implying that all affected women share the same long-term prognosis.

These conclusions should be interpreted within the limits of the study design, participant selection, and measurement methods. Observed relationships among hormonal, anthropometric, and metabolic variables do not establish causality or predict individual outcomes. Further prospective research using standardized diagnostic definitions and reliable assays is needed to clarify phenotype-specific trajectories and identify practical risk-stratification strategies. Overall, a balanced, multidimensional assessment offers a clinically useful framework for recognizing polycystic ovary syndrome while avoiding overreliance on isolated biochemical or ultrasonographic findings.

AUTHOR INFORMATION

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