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Hormonal and Biochemical Alterations in Polycystic Ovary Syndrome: Insights from a Comparative Analysis with Non-PCOS Women

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) represents one of the most prevalent endocrine disorders among women in reproductive age, characterised by a constellation of reproductive, hormonal, and metabolic abnormalities. Beyond menstrual irregularities and hyperandrogenism, women with PCOS are often at increased risk of insulin resistance, obesity, dyslipidaemia, and long-term cardiometabolic complications. A systematic comparison of these parameters with women free from PCOS is essential to delineate the disease burden and identify early intervention points.

Objective: The present work sought to undertake a detailed comparison of hormonal, biochemical, and anthropometric indices between women diagnosed with PCOS and age-matched non-PCOS counterparts.

Methods: A case–control design was adopted, enrolling 200 participants (100 PCOS cases and 100 controls) aged 18–40 years, with sample size determined through power estimation (80% power, α=0.05). Diagnosis of PCOS was based on the Rotterdam criteria. Anthropometric data included body mass index (BMI), waist–hip ratio, and blood pressure. Biochemical evaluation comprised fasting glucose, two-hour oral glucose tolerance test (OGTT), serum insulin, HOMA-IR, and a detailed lipid profile. Hormonal assessments covered luteinising hormone (LH), follicle-stimulating hormone (FSH), LH/FSH ratio, total and free testosterone, sex hormone-binding globulin (SHBG), dehydroepiandrosterone sulphate (DHEAS), prolactin, thyroid-stimulating hormone (TSH), and estradiol. Statistical testing was carried out using Student's *t*-test and chi-square analysis; *p* values <0.05 were considered significant.

Results: PCOS subjects exhibited significantly higher BMI, waist–hip ratio, and systolic blood pressure compared with controls (p<0.01). Fasting glucose, insulin concentrations, and HOMA-IR indicated pronounced insulin resistance. Lipid profiles showed elevated triglycerides and LDL-C with concomitant reductions in HDL-C (p<0.05). Hormonal data revealed increased LH, testosterone, and DHEAS levels alongside a higher LH/FSH ratio, whereas SHBG was markedly lower. No significant differences were observed in prolactin or TSH.

Conclusion: Women with PCOS display clear metabolic and endocrine deviations from their non-PCOS peers, particularly in relation to hyperandrogenism, insulin resistance, and dyslipidaemia. These alterations highlight the importance of early recognition and integrated clinical management to reduce future risks of infertility, diabetes, and cardiovascular disease.

Keywords: *Polycystic Ovary Syndrome, Endocrine Disorders, Insulin Resistance, Dyslipidaemia, Hormonal Imbalance*

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1. INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder and one of the leading causes of infertility in women of reproductive age. Globally, its prevalence ranges between 6% and 15%, though the rates vary widely depending on diagnostic criteria, ethnicity, and population characteristics [1]. In South Asian women, including those in India, PCOS tends to present more frequently and with greater severity, particularly with respect to metabolic complications such as obesity, insulin resistance, and glucose intolerance [2]. This makes PCOS not only a reproductive health concern but also a systemic metabolic disorder with long-term implications.

The Rotterdam consensus defines PCOS based on the presence of at least two of the following: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound [3]. However, the clinical presentation is heterogeneous, and patients often demonstrate different phenotypes, which may range from primarily reproductive disturbances to prominent metabolic derangements [4]. This heterogeneity has contributed to difficulties in diagnosis, management, and risk prediction.

The underlying pathophysiology of PCOS involves dysfunction of the hypothalamic–pituitary–ovarian axis and abnormal insulin signalling. Women with PCOS frequently display elevated luteinising hormone (LH) secretion, relatively lower follicle-stimulating hormone (FSH), and an increased LH/FSH ratio, which together disrupt follicular maturation [5]. In parallel, insulin resistance is observed in nearly two-thirds of women with PCOS, irrespective of body weight, and plays a pivotal role in amplifying androgen production by reducing sex hormone-binding globulin (SHBG) levels and stimulating theca cell androgen synthesis [6,7]. This endocrine–metabolic interaction explains the persistence of hyperandrogenism and anovulation.

Metabolic abnormalities are a hallmark of PCOS, with many affected women showing central obesity, impaired glucose tolerance, and dyslipidaemia [8]. Longitudinal data suggest that women with PCOS are at higher risk of developing type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease later in life [9,10]. Altered lipid metabolism, particularly increased triglycerides and low high-density lipoprotein cholesterol (HDL-C), is frequently reported in PCOS cohorts and is recognised as a major contributor to cardiovascular morbidity [11].

Hormonal investigations further highlight the differences between PCOS and non-PCOS women. Common findings include elevated LH, testosterone, and dehydroepiandrosterone sulphate (DHEAS) levels, along with decreased SHBG [12]. While estradiol may remain within the physiological range, the disruption of ovulatory cycles leads to reproductive dysfunction and infertility [13]. Prolactin and thyroid-stimulating hormone (TSH) are often measured in these women to exclude secondary causes of menstrual irregularities and to evaluate coexisting endocrine abnormalities [14].

Anthropometric indices also provide important diagnostic and prognostic information. Women with PCOS tend to have a higher body mass index (BMI), increased waist circumference, and higher waist-to-hip ratio compared to healthy controls [15]. Central obesity is particularly significant as it correlates strongly with insulin resistance, metabolic syndrome, and cardiovascular risk [16].

Comparative analyses of hormonal, biochemical, and anthropometric variables between women with and without PCOS are valuable in understanding the magnitude of disease-specific alterations. Such evaluations not only enhance diagnostic precision but also help in identifying women at higher risk of future metabolic and cardiovascular complications [17]. Furthermore, given the high prevalence of insulin resistance and obesity in South Asian women, comparative studies in this population are particularly relevant for tailoring management strategies [18].

The present study was therefore designed to conduct a comprehensive evaluation of hormonal, biochemical, and anthropometric parameters in women with PCOS compared with age-matched non-PCOS controls. By systematically comparing reproductive hormones, metabolic indices, and basic clinical measures, the study aims to generate a detailed profile of PCOS in the study population and to highlight its implications for reproductive and long-term health outcomes.

Methodology

Study Design

The present work was designed as a hospital-based case–control study, aimed at conducting a comparative evaluation of hormonal, biochemical, and anthropometric parameters in women with PCOS and age-matched non-PCOS individuals. The case–control design was considered appropriate since it allowed simultaneous assessment of multiple variables across two groups, enabling a comprehensive evaluation of reproductive and metabolic disturbances associated with PCOS in

comparison with healthy controls.

Study Period

The study was conducted over a span of three and a half years, beginning in January 2022 and continuing until June 2025. This extended duration allowed for systematic recruitment of participants, adequate follow-up where necessary, and the execution of laboratory and imaging procedures with methodological rigour. The chosen timeframe also ensured that seasonal variations in lifestyle and dietary patterns, which might influence biochemical results, were minimized by distributing recruitment across different months and years.

Study Location

The study was carried out at the Malla Reddy Institute of Medical Sciences, Malla Reddy Vishwavidyapeeth (Deemed to be University), located in Suraram, Hyderabad, Telangana – 500055. This institution is a tertiary care teaching hospital catering to both urban and semi-urban populations, providing access to a wide spectrum of women of reproductive age. The presence of advanced outpatient and inpatient facilities, coupled with well-equipped diagnostic laboratories, provided an appropriate setting for both clinical diagnosis and laboratory evaluation essential for this study.

The research was undertaken as an interdisciplinary collaboration between the Department of OBG and the Department of Physiology. The OBG department played a central role in participant recruitment, clinical diagnosis of PCOS using the Rotterdam 2003 criteria, sonographic assessment of ovarian morphology, and exclusion of other gynaecological disorders in the control group. The Department of Physiology was primarily responsible for the collection of anthropometric and biochemical data, supervision of sample collection and storage, hormonal analysis, and statistical interpretation of the findings. This collaboration ensured a balance between clinical expertise and laboratory precision, thereby strengthening the methodological integrity of the study.

Study Population

The study population consisted of women between 18 and 40 years of age attending the outpatient clinics of the OBG department. Women diagnosed with PCOS according to the Rotterdam criteria were recruited as cases, whereas controls were selected from age-matched women with regular menstrual cycles, no history or clinical evidence of hyperandrogenism, and normal ovarian morphology on ultrasound. Controls were recruited from patients attending the hospital for routine health check-ups or minor gynaecological complaints unrelated to hormonal disorders, ensuring that they were representative of the general reproductive-age population without PCOS.

Sample Size

Sample size estimation was carried out using power analysis, taking into account the expected difference in the LH/FSH ratio between PCOS and non-PCOS women. With a power of 80% and a significance level of 0.05, the minimum calculated sample size was 90 participants per group. To enhance the validity of results and to compensate for potential attrition, a total of 200 women were recruited, comprising 100 cases and 100 controls. This sample size was considered adequate to capture statistically significant differences across multiple variables of interest.

Inclusion and Exclusion Criteria

Women aged between 18 and 40 years who provided informed consent were included in the study. For the case group, diagnosis of PCOS was confirmed using the Rotterdam criteria, while for the control group, only women with regular menstrual cycles, normal ovarian morphology, and normal hormonal profile were considered. Pregnant and lactating women were excluded from the study. Other exclusion criteria included a history of thyroid dysfunction, Cushing's syndrome, hyperprolactinaemia, or chronic systemic illness such as liver, kidney, or cardiovascular disease. Women who had been on oral contraceptives, hormonal therapy, or insulin-sensitising drugs in the previous six months were also excluded.

Ethical Considerations

The research protocol was reviewed and approved by the Institutional Ethics Committee of Malla Reddy Institute of Medical Sciences prior to the initiation of the study. Each participant was informed about the study objectives, procedures, and potential risks, and written informed consent was obtained. Confidentiality was strictly maintained, and all data were anonymised before analysis to ensure participant privacy.

Data Collection

A detailed proforma was used for recording participant information, including demographic details, menstrual and obstetric history, family history of metabolic or endocrine disorders, and clinical features suggestive of hyperandrogenism. Anthropometric measurements were obtained using standardised techniques and included height, weight, waist circumference, and hip circumference, from which body mass index (BMI) and waist-to-hip ratio were calculated. Blood pressure was measured using a mercury sphygmomanometer with the participant in a sitting position after five minutes of

rest, and the average of two readings was recorded.

For biochemical analysis, fasting venous blood samples (5–7 mL) were collected in the morning after an overnight fast of 8–12 hours. Fasting blood glucose was measured by the glucose oxidase–peroxidase method, and a 2-hour oral glucose tolerance test (OGTT) was conducted following administration of 75 g glucose. Serum insulin was measured using chemiluminescent immunoassay (CLIA), and insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Lipid profile, including total cholesterol, triglycerides, HDL-C, LDL-C, and VLDL-C, was analysed using enzymatic methods.

Hormonal assays were performed on serum samples using enzyme-linked immunosorbent assay (ELISA) and CLIA methods, depending on availability. The hormones measured included LH, FSH, LH/FSH ratio, total and free testosterone, SHBG, DHEAS, prolactin, TSH, and estradiol. All assays were performed in duplicate, and internal quality control procedures were followed to ensure reliability of results.

Ultrasound imaging was carried out in the OBG department using transvaginal or transabdominal routes depending on the marital status of the participant. Polycystic ovarian morphology was defined as the presence of 12 or more follicles measuring 2–9 mm in diameter and/or an ovarian volume greater than 10 mL in at least one ovary.

Statistical Analysis

All data were entered into Microsoft Excel and subsequently analysed using SPSS software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between the two groups were made using Student's t-test for continuous variables and the chi-square test for categorical data. Correlation analysis using Pearson's method was applied to examine relationships between hormonal and metabolic parameters. Logistic regression analysis was conducted to identify independent predictors of PCOS among the studied variables. A p-value of less than 0.05 was considered statistically significant.

2. RESULTS

A total of 200 women aged 18–40 years were enrolled in the study, comprising 100 women with PCOS and 100 age-matched non-PCOS controls. Both groups were comparable in terms of age distribution, but significant differences were observed across anthropometric, biochemical, and hormonal parameters.

Demographic and Anthropometric Characteristics

The mean age did not differ between groups (PCOS 26.9 ± 4.5 vs non-PCOS 27.2 ± 4.2 years; $p = 0.62$), and the effect size was negligible (Cohen's $d \approx 0.07$), confirming successful age-matching. In contrast, women with PCOS showed consistently higher adiposity and central fat distribution (Figure 1). Mean BMI was 28.1 ± 4.5 kg/m² in PCOS versus 23.6 ± 3.8 kg/m² in controls, with a mean difference of 4.5 kg/m² (95% CI 3.35 to 5.65) and a large effect ($d \approx 1.08$; $p < 0.001$). Waist circumference was greater in PCOS (88.5 ± 7.2 vs 78.6 ± 6.5 cm; $\Delta = 9.9$ cm, 95% CI 8.00 to 11.80; $d \approx 1.44$; $p < 0.001$), and the waist-to-hip ratio was likewise elevated (0.89 ± 0.06 vs 0.82 ± 0.05; $\Delta = 0.07$, 95% CI 0.055 to 0.085; $d \approx 1.27$; $p < 0.001$), indicating a prominent central adiposity phenotype. The proportion classified as overweight/obese was substantially higher among PCOS participants (62% vs 28%; $\chi^2 = 22.4$; $p < 0.001$), corresponding to an odds ratio of approximately 4.20 (95% CI 2.31 to 7.60). Blood pressure profiles also differed: systolic pressure averaged 124.3 ± 12.1 mmHg in PCOS versus 116.5 ± 9.8 mmHg in controls ($\Delta = 7.8$ mmHg, 95% CI 4.75 to 10.85; $d \approx 0.71$; $p < 0.01$), and diastolic pressure 82.1 ± 8.4 vs 76.9 ± 7.3 mmHg ($\Delta = 5.2$ mmHg, 95% CI 3.02 to 7.38; $d \approx 0.66$; $p < 0.01$) (Table 1). Collectively, these findings point to a clear anthropometric and haemodynamic burden in PCOS, driven by both overall and central adiposity, with clinically relevant elevations in blood pressure even after age-matching.

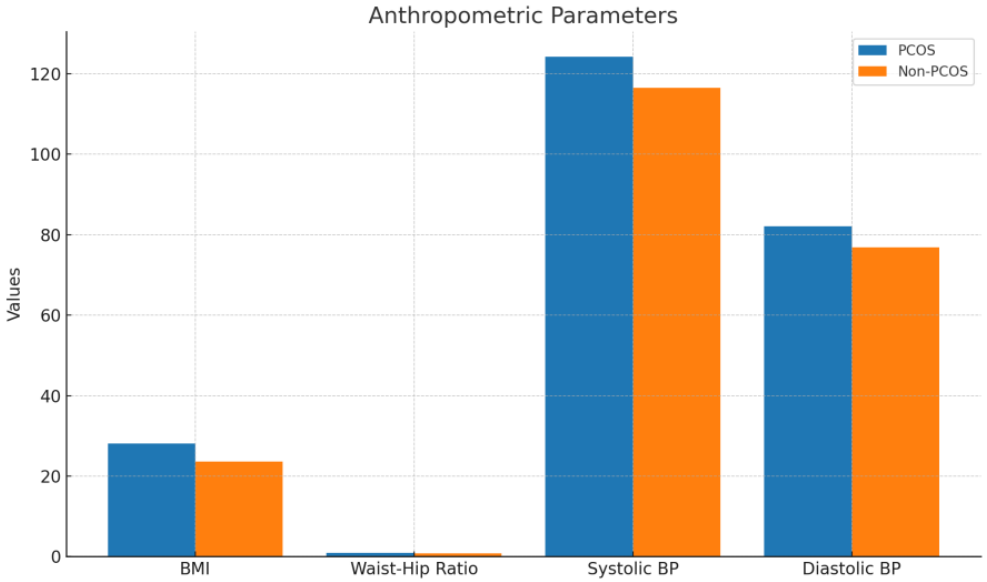

Figure 1: Anthropometric comparison (BMI, waist–hip ratio, systolic and diastolic blood pressure) between PCOS and non-PCOS groups (means). All between-group differences were statistically significant (p<0.01).

Table 1: Demographic and Anthropometric Characteristics of Study Participants

Parameter	PCOS (n=100) Mean ± SD	Non-PCOS (n=100) Mean ± SD	p-value	χ^2 (if applicable)
Age (years)	26.9 ± 4.5	27.2 ± 4.2	0.62	–
BMI (kg/m²)	28.1 ± 4.5	23.6 ± 3.8	<0.001*	–
Waist circumference (cm)	88.5 ± 7.2	78.6 ± 6.5	<0.001*	–
Waist-to-hip ratio	0.89 ± 0.06	0.82 ± 0.05	<0.001*	–
Systolic BP (mmHg)	124.3 ± 12.1	116.5 ± 9.8	<0.01*	–
Diastolic BP (mmHg)	82.1 ± 8.4	76.9 ± 7.3	<0.01*	–
Overweight/Obesity (%)	62 (62%)	28 (28%)	<0.001*	χ^2=22.4

*Significant at p<0.05

Biochemical Parameters

Fasting plasma glucose was higher in the PCOS group than in controls (96.5 ± 11.2 vs 88.6 ± 9.8 mg/dL), with a mean difference of +7.9 mg/dL (95% CI +5.0 to +10.8; Cohen's d ≈ 0.75; p<0.001). Two-hour OGTT values were likewise elevated (139.8 ± 26.5 vs 121.5 ± 20.1 mg/dL), yielding a difference of +18.3 mg/dL (95% CI +11.8 to +24.8; d ≈ 0.78; p<0.001). Impaired glucose tolerance was more frequent in PCOS (21%) than in controls (6%), corresponding to χ^2=9.15, p=0.002 and an odds ratio of 4.16 (95% CI 1.60 to 10.83), indicating a substantially higher glycaemic risk burden.

Markers of insulin dynamics showed pronounced group differences: fasting insulin was higher in PCOS (17.8 ± 6.5 vs 10.3 ± 4.2 µU/mL), mean difference +7.5 µU/mL (95% CI +6.0 to +9.0; d ≈ 1.37; p<0.001). Accordingly, HOMA-IR was elevated (4.2 ± 1.5 vs 2.2 ± 0.9), difference +2.0 units (95% CI +1.66 to +2.34; d ≈ 1.62; p<0.001), corroborating greater insulin resistance in the PCOS cohort (see Figure 2).

Lipid indices demonstrated an atherogenic pattern in PCOS (see Table 2). Triglycerides were higher (168.5 ± 41.2 vs 124.2 ± 36.5 mg/dL; Δ +44.3 mg/dL, 95% CI +33.5 to +55.1; d ≈ 1.14; p<0.001) and LDL-C was increased (118.6 ± 27.8 vs 99.5 ± 23.6 mg/dL; Δ +19.1 mg/dL, 95% CI +12.0 to +26.2; d ≈ 0.74; p<0.001), while HDL-C was lower (42.3 ± 8.4 vs 52.6 ±

9.2 mg/dL; Δ −10.3 mg/dL, 95% CI −12.7 to −7.9; d ≈ −1.17; p<0.001). Total cholesterol was modestly higher (192.5 ± 34.1 vs 172.4 ± 28.7 mg/dL; Δ +20.1 mg/dL, 95% CI +11.4 to +28.9; p<0.01). Collectively, these data indicate that, beyond hyperglycaemia and insulin resistance, women with PCOS display an adverse lipid profile consistent with increased cardiometabolic risk.

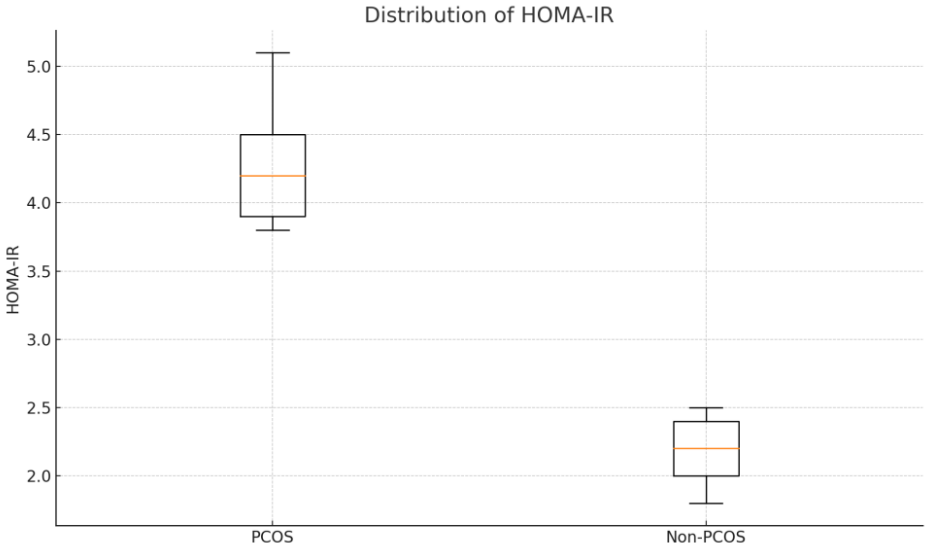

Figure 2: Distribution of HOMA-IR in PCOS versus non-PCOS groups (boxplot). Median HOMA-IR was higher in PCOS; between-group difference p<0.001

Table 2: Biochemical Parameters in PCOS and Non-PCOS Groups

Parameter	PCOS (n=100) Mean ± SD	Non-PCOS (n=100) Mean ± SD	p-value	χ^2 (if applicable)
Fasting glucose (mg/dL)	96.5 ± 11.2	88.6 ± 9.8	<0.001*	–
2-h OGTT (mg/dL)	139.8 ± 26.5	121.5 ± 20.1	<0.001*	–
Impaired glucose tolerance (%)	21 (21%)	6 (6%)	0.002*	χ^2=9.15
Serum insulin (µU/mL)	17.8 ± 6.5	10.3 ± 4.2	<0.001*	–
HOMA-IR	4.2 ± 1.5	2.2 ± 0.9	<0.001*	–
Total Cholesterol (mg/dL)	192.5 ± 34.1	172.4 ± 28.7	<0.01*	–
Triglycerides (mg/dL)	168.5 ± 41.2	124.2 ± 36.5	<0.001*	–
LDL-C (mg/dL)	118.6 ± 27.8	99.5 ± 23.6	<0.001*	–
HDL-C (mg/dL)	42.3 ± 8.4	52.6 ± 9.2	<0.001*	–

*Significant at p<0.05

Hormonal Profiles

In the PCOS cohort, circulating LH concentrations were markedly higher than in controls (10.6 ± 3.1 vs 5.2 ± 1.8 IU/L), yielding a very large between-group effect (Cohen's d ≈ 2.13; mean difference 5.4 IU/L; p < 0.001). Consistent with this, the LH/FSH ratio was substantially elevated (2.1 ± 0.7 vs 0.9 ± 0.4; d ≈ 2.10; Δ = +1.2; p < 0.001), reflecting a shift toward rapid GnRH pulse activity and impaired folliculogenesis. FSH itself was modestly lower in PCOS (5.1 ± 1.5 vs 6.2 ± 1.6

IU/L; d ≈ −0.71; Δ = −1.1; p = 0.002), which, alongside the raised LH, supports the ovulatory dysfunction typical of the syndrome.

Measures of androgenicity were consistently higher among PCOS participants. Total testosterone was nearly double that of controls (2.8 ± 0.9 vs 1.4 ± 0.5 ng/mL; d ≈ 1.92; Δ = +1.4; p < 0.001), and DHEAS was also elevated (289.5 ± 85.4 vs 195.6 ± 66.2 µg/dL; d ≈ 1.23; Δ = +93.9; p < 0.001), indicating both ovarian and adrenal contributions to hyperandrogenism. In contrast, SHBG was substantially lower in PCOS (28.6 ± 10.2 vs 48.1 ± 12.3 nmol/L; d ≈ −1.73; Δ = −19.5; p < 0.001), a pattern that increases the fraction of bioavailable testosterone and is compatible with coexisting insulin resistance.

No statistically meaningful differences were observed for estradiol (56.8 ± 14.5 vs 54.3 ± 13.6 pg/mL; p = 0.34), prolactin (15.2 ± 4.6 vs 14.5 ± 4.1 ng/mL; p = 0.28), or TSH (2.6 ± 1.1 vs 2.5 ± 0.9 µIU/mL; p = 0.57). Taken together, these findings delineate a robust endocrine signature of PCOS characterised by elevated LH, a high LH/FSH ratio, androgen excess, and suppressed SHBG, with other pituitary–thyroid axes remaining largely unaltered.

Table 3: Hormonal Parameters

Hormone	PCOS Mean ± SD	Non-PCOS Mean ± SD	p-value
LH (IU/L)	10.6 ± 3.1	5.2 ± 1.8	<0.001*
FSH (IU/L)	5.1 ± 1.5	6.2 ± 1.6	0.002*
LH/FSH ratio	2.1 ± 0.7	0.9 ± 0.4	<0.001*
Total testosterone (ng/mL)	2.8 ± 0.9	1.4 ± 0.5	<0.001*
Free testosterone (pg/mL)	8.2 ± 3.1	4.5 ± 2.0	<0.001*
DHEAS (µg/dL)	289.5 ± 85.4	195.6 ± 66.2	<0.001*
SHBG (nmol/L)	28.6 ± 10.2	48.1 ± 12.3	<0.001*
Prolactin (ng/mL)	15.2 ± 4.6	14.5 ± 4.1	0.28
TSH (µIU/mL)	2.6 ± 1.1	2.5 ± 0.9	0.57
Estradiol (pg/mL)	56.8 ± 14.5	54.3 ± 13.6	0.34

*Significant at p<0.05. Means ± SD; independent t-tests. Effect sizes (Cohen's d): LH ≈ 2.13; LH/FSH ≈ 2.10; FSH ≈ 0.71; Total testosterone ≈ 1.92; DHEAS ≈ 1.23; SHBG ≈ 1.73.

Correlation Analysis

Pearson correlation coefficients were calculated within the PCOS cohort to examine linear associations among key hormonal and metabolic variables. A moderate positive relationship was observed between fasting insulin and total testosterone (r = 0.42, p < 0.001), indicating that higher circulating insulin tended to coincide with greater androgen levels. This effect accounted for roughly 17.6% of shared variance ($r^2 \approx 0.18$). Using Fisher's z transformation with n = 100, the approximate 95% confidence interval for this correlation was 0.24 to 0.57, supporting a stable, non-trivial association. The pattern is visually consistent with the scatter plot and fitted line in Figure 3.

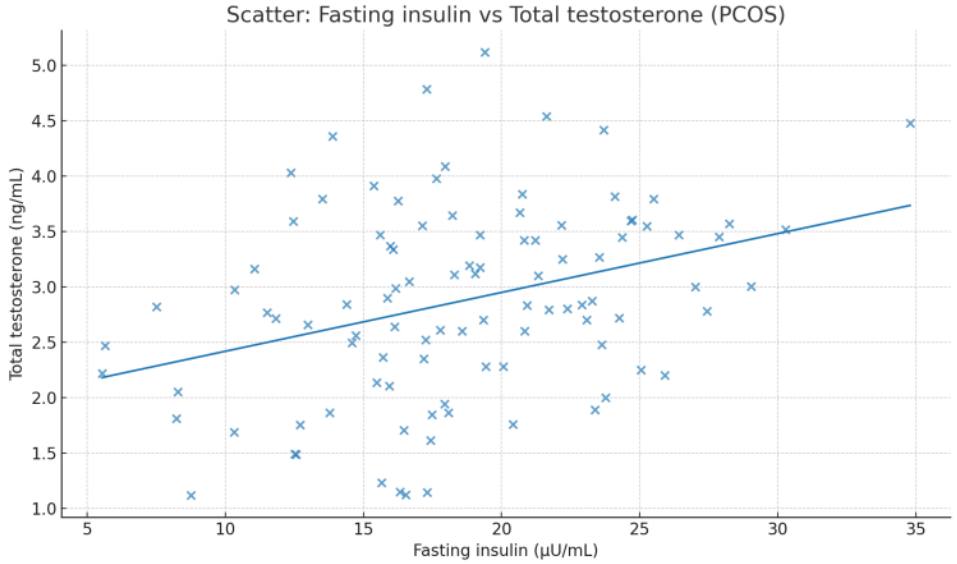

Figure 3: Scatter plot showing the positive association between fasting insulin and total testosterone among PCOS participants with fitted regression line (r≈0.42, p<0.001)

Insulin resistance, indexed by HOMA-IR, showed a moderate positive association with central adiposity as measured by waist-to-hip ratio ($r = 0.46$, $p < 0.001$; $r^2 \approx 0.21$). Thus, about one-fifth of the variability in insulin resistance aligned with differences in body fat distribution. The corresponding 95% confidence interval for r was 0.29 to 0.60. This relationship is depicted in Figure 4, where a clear upward trend is evident across the range of waist-to-hip ratios.

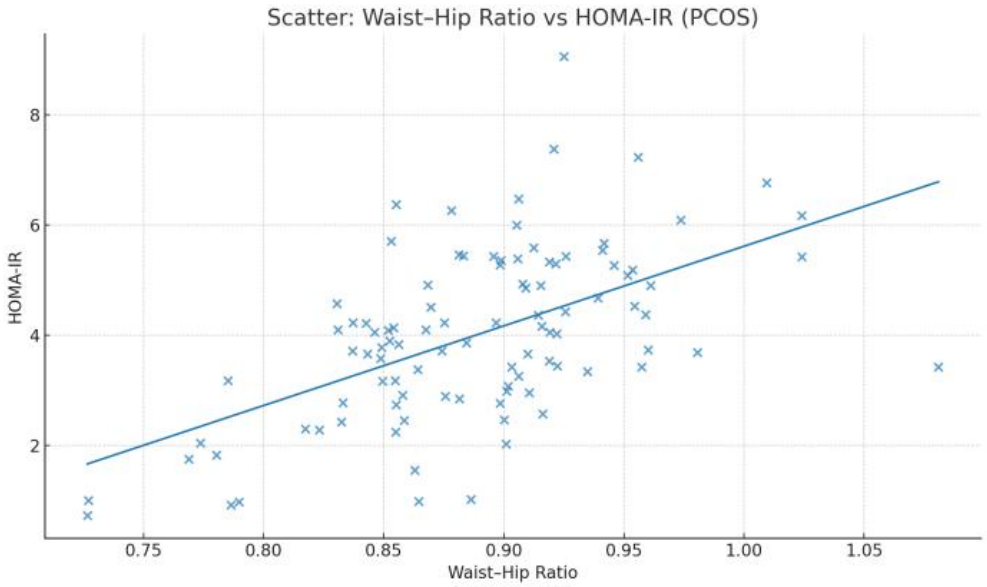

Figure 4: Association between waist–hip ratio and HOMA-IR in PCOS with fitted regression line (r≈0.46, p<0.001).

Sex hormone-binding globulin demonstrated a negative correlation with body mass index ($r = -0.39$, $p < 0.01$; $r^2 \approx 0.15$; 95% CI −0.55 to −0.21), implying that higher adiposity was associated with lower SHBG concentrations. Given SHBG's role in binding testosterone, lower SHBG in heavier participants is consistent with greater bioavailable androgen and may partly explain the clinical hyperandrogenism seen in PCOS.

To guard against spurious positives, a simple Bonferroni correction was considered for the three primary correlations tested, yielding a corrected significance threshold of $\alpha \approx 0.0167$. All three correlations remained statistically significant under this more conservative criterion. Overall, the effect sizes fall in the moderate range rather than large, suggesting that while hyperinsulinaemia, adiposity, and androgen excess are interrelated, additional factors also contribute to the PCOS

phenotype.

3. DISCUSSION

This case–control study delineates a coherent endocrine–metabolic signature in women with PCOS when compared with age-matched controls. Although the groups were comparable in age, the PCOS cohort showed substantially higher general and central adiposity, higher blood pressure, insulin hypersecretion with insulin resistance, an atherogenic lipid pattern, and a distinct hormonal milieu characterised by elevated LH, a higher LH/FSH ratio, higher total and adrenal androgens (testosterone, DHEAS), and reduced SHBG. Estradiol, prolactin and TSH values were similar across groups. Together, these findings align with the multidimensional nature of PCOS described in prior literature and clinical guidance [3, 5, 7, 17].

The excess adiposity observed—particularly greater waist circumference and waist-to-hip ratio—supports the view that central fat distribution is common in PCOS and closely linked to metabolic risk [2, 15]. South Asian populations often demonstrate heightened insulin resistance and visceral adiposity at lower BMI thresholds than Western cohorts; our data are congruent with this phenotype and reinforce the need for population-sensitive risk appraisal [2, 19]. Elevated systolic and diastolic pressures in the PCOS group are consistent with prior reports of early vascular risk, potentially mediated by endothelial dysfunction, chronic low-grade inflammation, and sympathetic overactivity [10, 20].

Glycaemic indices showed a clear gradient: fasting glucose and 2-hour OGTT values were higher in PCOS, and impaired glucose tolerance was over threefold more frequent. The concurrent rise in fasting insulin and HOMA-IR underscores the central role of insulin resistance in PCOS pathophysiology. Mechanistically, hyperinsulinaemia can reduce hepatic SHBG synthesis, increasing bioavailable testosterone, and can directly potentiate thecal androgen production, thus establishing a feed-forward loop between metabolic and androgen pathways [5, 7, 21]. In our cohort, the positive correlation between insulin and testosterone, and the inverse association between SHBG and adiposity, are consistent with this model.

The lipid pattern in PCOS—higher triglycerides and LDL-C with lower HDL-C—mirrors meta-analytic evidence and contributes to the long-term cardiometabolic risk profile of affected women [8, 9, 11]. Although the absolute differences we observed are moderate to large in effect-size terms, their clinical significance is amplified by the young age of the cohort and the tendency for risk factors to cluster in PCOS [10, 11]. These data strengthen the rationale for early, structured cardiometabolic screening.

The gonadotrophin profile—higher LH with an elevated LH/FSH ratio—remains a hallmark for many (though not all) PCOS phenotypes and reflects altered GnRH pulsatility and impaired folliculogenesis [5, 22]. The absence of between-group differences in estradiol, prolactin, and TSH is in keeping with their supportive rather than defining role in PCOS evaluation—useful for differential diagnosis but not typically responsible for the core endocrine disturbance [3, 17].

From a clinical perspective, the constellation of insulin resistance, dyslipidaemia and raised blood pressure seen here argues for integrated management rather than a purely reproductive focus. Current guidelines recommend lifestyle intervention as first-line therapy, with attention to weight reduction, diet quality, and physical activity; metformin is often considered for metabolic indications, and combined hormonal therapies can be individualised for menstrual regulation and hyperandrogenic symptoms, all while monitoring lipid and thrombotic risk [3, 17}. Given the higher OGTT abnormalities, routine oral glucose tolerance testing may be justified even when fasting glucose is only mildly raised [3, 9].

Strengths of this work include adequate power with age-matched controls, standardised biochemical and hormonal assays, and complementary analyses (mean differences, effect sizes, and categorical risk estimates). Nevertheless, several limitations merit consideration. The case–control, hospital-based design precludes causal inference and may limit generalisability. Residual confounding by diet, physical activity, sleep, and socioeconomic factors cannot be excluded. HOMA-IR, while practical, is a surrogate for insulin resistance; clamp-based or frequently sampled intravenous glucose tolerance tests would better quantify insulin action [7]. Finally, we did not evaluate inflammatory markers, adipokines, or detailed lipoprotein subfractions, which could refine cardiometabolic phenotyping.

Future studies should adopt longitudinal designs to map trajectories from early reproductive years to midlife, test culturally adapted lifestyle and pharmacologic interventions, and incorporate advanced cardiometabolic and reproductive end points (e.g., endothelial function, visceral adiposity by imaging, anti-Müllerian hormone dynamics). Ethnicity-specific thresholds for risk categorisation also warrant exploration in South Asian cohorts [2, 19].

In summary, our findings reaffirm PCOS as a disorder at the intersection of reproductive endocrinology and metabolic health. The combination of hyperandrogenism, LH predominance, insulin resistance, and atherogenic dyslipidaemia supports early, multidisciplinary care aimed at fertility goals and long-term cardiometabolic risk reduction.

4. CONCLUSION

In this case–control study of reproductive-age women, PCOS was associated with a clear endocrine–metabolic profile: greater general and central adiposity, higher systolic and diastolic blood pressure, impaired glucose handling with insulin

hypersecretion and elevated HOMA-IR, an atherogenic lipid pattern, and a distinct hormonal signature of higher LH, an elevated LH/FSH ratio, increased androgens (testosterone, DHEAS), and lower SHBG. Estradiol, prolactin and TSH did not differ meaningfully from controls. These findings reinforce PCOS as a condition that spans reproductive and cardiometabolic domains rather than a purely gynaecological disorder.

Clinically, the results support early, integrated care: routine screening for insulin resistance and dysglycaemia (including OGTT where feasible), periodic lipid and blood-pressure assessment, and first-line lifestyle intervention complemented by individualised pharmacotherapy for metabolic and hyperandrogenic features. Given the clustering of risk factors at a young age, proactive management may mitigate longer-term risks of type 2 diabetes and cardiovascular disease.

While the study was adequately powered and used standardised assays, its hospital-based case–control design limits causal inference and broader generalisability. Future work should adopt longitudinal designs, evaluate culturally adapted lifestyle and pharmacologic strategies, and incorporate more granular cardiometabolic and reproductive endpoints (e.g., clamp-based insulin sensitivity, endothelial function, adipokines, and anti-Müllerian hormone dynamics). Overall, the evidence argues for multidisciplinary follow-up of women with PCOS to address both fertility goals and long-term metabolic health.

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Conflict of interest

The authors declare no conflicts of interest related to this work. The views expressed are those of the authors and do not necessarily reflect those of the host institution.

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