RESEARCH PAPER

Endocrine and Biochemical Profile Differences between Polycystic Ovary Syndrome and Non-PCOS Women.

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ABSTRACT

Background:Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder in women of reproductive age, marked by reproductive, hormonal, and metabolic abnormalities. Hormonal imbalance and biochemical disturbances are central to its pathophysiology and contribute to long-term health risks. Comparative assessment with non-PCOS women provides clarity on disease-specific alterations and potential clinical markers.

Objectives:To compare hormonal and biochemical profiles between women with PCOS and age-matched non-PCOS women, and to quantify the magnitude of endocrine and metabolic disturbances associated with PCOS.

Methods:A comparative cross-sectional study was conducted among 240 subjects, comprising 120 women diagnosed with PCOS based on standard diagnostic criteria and 120 non-PCOS controls. Clinical assessment was followed by hormonal evaluation, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and total testosterone. Biochemical parameters included fasting blood glucose and lipid profile. Intergroup comparisons were performed using appropriate statistical tests, with statistical significance defined at conventional levels.

Results:Women with PCOS showed significantly higher mean LH levels (12.4 ± 4.1 IU/L) compared with controls (6.8 ± 2.5 IU/L), along with an elevated LH/FSH ratio (>2 in 62% of PCOS subjects versus 8% of controls). Mean total testosterone levels were also higher in the PCOS group (78.6 ± 21.3 ng/dL) compared with non-PCOS women (42.9 ± 15.7 ng/dL). Biochemical analysis revealed higher fasting blood glucose levels in PCOS subjects (96.2 ± 12.8 mg/dL vs. 88.4 ± 10.6 mg/dL), with dyslipidaemia observed in nearly half of the PCOS group (48%) compared to 22% among controls. These differences were statistically significant, indicating pronounced endocrine and metabolic alterations in PCOS.

Conclusion:Women with PCOS exhibit marked hormonal disturbances and adverse biochemical profiles when compared with non-PCOS women. The observed elevations in androgen levels, altered gonadotropin dynamics, and increased metabolic abnormalities reinforce the need for integrated hormonal and biochemical screening. Early identification of these changes may facilitate timely intervention and reduce long-term reproductive and cardiometabolic complications**..**

Keywords: Polycystic ovary syndrome, hormonal profile, biochemical parameters, hyperandrogenism, metabolic abnormalities..

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine–metabolic disorder affecting women of reproductive age and remains a leading cause of anovulatory infertility, menstrual irregularity, and clinical or biochemical hyperandrogenism [1–3]. Beyond reproductive manifestations, PCOS is increasingly recognised as a multisystem condition with clinically meaningful associations with obesity, insulin resistance, dyslipidaemia, hypertension, non-alcoholic fatty liver disease, and long-term cardiometabolic risk [1,2,4]. The heterogeneity of clinical phenotypes, variability in symptom onset across the life course, and overlap with other endocrine conditions

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contribute to delayed diagnosis and fragmented care pathways, particularly in resource-constrained settings where standardised endocrine evaluation is not uniformly available [1,3].

Current diagnostic approaches rely on composite criteria that integrate ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology; however, the interpretation of each component is influenced by age, ethnicity, assay variability, and concomitant metabolic status [3]. The 2023 International Evidence-based Guideline update emphasises improving diagnostic accuracy and consistency, while acknowledging evidence gaps and the ongoing need for pragmatic, patient-centred evaluation strategies that can be implemented across diverse healthcare systems [1]. In parallel, recent syntheses of evidence highlight that panels of hormonal and biochemical markers can better characterise multisystem involvement and improve clinical surveillance, rather than relying on any single analyte as a standalone discriminator [5].

Against this background, comparative studies that evaluate hormonal and biochemical alterations in PCOS versus non-PCOS women are particularly valuable because they (i) quantify the direction and magnitude of endocrine differences, (ii) clarify metabolic risk signals within locally relevant populations, and (iii) inform feasible screening packages for routine outpatient settings. A structured comparison is also important for interpreting intermediate phenotypes (for example, women with irregular cycles without frank hyperandrogenism, or women with biochemical hyperandrogenism but minimal clinical signs), where diagnosis and counselling are often uncertain in real-world practice [3,5].

Hyperandrogenism is central to PCOS pathophysiology and is commonly reflected by elevated total or free testosterone, increased free androgen index (FAI), and reduced sex hormone-binding globulin (SHBG), especially in the setting of insulin resistance [5,6]. Biochemically, androgen excess contributes to follicular arrest, anovulation, and clinical manifestations such as hirsutism and acne, while also intersecting with metabolic dysfunction through adipose tissue biology and hepatic SHBG regulation [4,6]. Contemporary reviews and evidence syntheses continue to prioritise total/free testosterone, SHBG, and FAI as core markers because they represent both reproductive and metabolic dimensions of the syndrome [5,6].

Gonadotropin alterations, particularly relative luteinizing hormone (LH) predominance and an increased LH:FSH ratio, have been described for decades; however, their diagnostic value varies by assay timing, body mass index, and PCOS phenotype [3]. Recent mechanistic reappraisals continue to explore why some women, including those with “lean PCOS”, demonstrate disproportionate LH secretion and altered hypothalamic–pituitary–ovarian axis feedback, reinforcing the concept that endocrine profiling should be interpreted contextually rather than as a rigid threshold-based rule [12].

AMH has gained prominence because it reflects the increased pool of small antral follicles typical of PCOS and may provide an objective surrogate for polycystic ovarian morphology when ultrasound quality is limited [5,7]. The 2023 guideline-linked meta-analysis assessing AMH integration into diagnostic frameworks supports its potential utility, while also highlighting the importance of standardisation, age adjustment, and phenotype stratification [7]. More recent systematic evidence similarly concludes that AMH can function as an adjunct diagnostic marker when assay methodology and population factors are considered, and may correlate with syndrome severity in some settings [8]. These findings strengthen the rationale for including AMH in comparative endocrine profiling, particularly in studies aiming to build clinically usable biomarker packages rather than relying only on imaging-dependent criteria [5,7,8].

Insulin resistance is a frequent biochemical substrate in PCOS and has relevance even in women without obesity, although the prevalence and intensity vary by ethnicity and phenotype [1,4]. Case-control evidence from recent populations demonstrates that insulin resistance and associated glycaemic perturbations can be meaningfully higher in PCOS than in non-PCOS comparators, and that hyperandrogenism and adiposity may independently relate to insulin resistance indices such as HOMA-IR [10]. Such data support studying insulin-related markers (fasting glucose, fasting insulin, HOMA-IR, and where feasible OGTT-derived measures) alongside androgen parameters to capture endocrine–metabolic coupling [5,10].

Dyslipidaemia and atherogenic lipid patterns are also clinically relevant in PCOS, reflecting altered hepatic lipid handling, adipose dysfunction, and insulin-mediated metabolic effects [2,4]. Recent meta-analytic evidence shows that composite indices such as the triglyceride–glucose (TyG) index and lipid ratios (TC/HDL, TG/HDL, LDL/HDL) tend to be higher in women with PCOS than controls, reinforcing their potential role as accessible risk stratification tools, particularly where advanced testing is unavailable [21]. This aligns with broader evidence linking PCOS with increased cardiovascular disease risk, prompting guideline-driven emphasis on cardiometabolic screening and longitudinal follow-up [2].

Chronic low-grade inflammation and oxidative stress are increasingly described as intersecting mechanisms that may amplify both reproductive and metabolic disturbances in PCOS [4,6,15]. Systematic reviews note consistent signals for oxidative stress imbalance and inflammatory marker elevation in many PCOS cohorts, although heterogeneity remains substantial due to phenotype differences, assay platforms, and confounding by adiposity [5,15]. Contemporary mechanistic discussions continue to position insulin resistance, inflammation, and hyperandrogenism as mutually reinforcing pathways, supporting study designs that quantify representative biochemical domains rather than focusing narrowly on one endocrine axis [4].

A comparative evaluation of hormonal and biochemical alterations in PCOS versus non-PCOS women is clinically meaningful because it can clarify which markers (or marker combinations) best differentiate cases from controls in a given population, while also quantifying cardiometabolic burden that warrants early counselling and prevention strategies. This is particularly important because PCOS has been linked with

elevated long-term cardiometabolic disease risk, and guideline updates increasingly frame PCOS as a lifelong health condition rather than an isolated reproductive disorder [1,2,22]. A study with a total sample size of 240 participants can be adequately positioned to examine endocrine contrasts (androgen parameters, gonadotropins, AMH) alongside metabolic indices (glucose–insulin measures, lipid parameters, selected inflammatory/oxidative markers if included), and to explore whether observed differences persist after accounting for adiposity and age distribution. Such comparative insights may help refine practical diagnostic and surveillance bundles that are feasible for routine clinical use and aligned with evidence-based recommendations [1,5].

In summary, the modern understanding of PCOS supports an integrated endocrine–metabolic model where hyperandrogenism, ovulatory dysfunction, follicle excess (often reflected by AMH), and metabolic dysregulation coexist to varying degrees across phenotypes [1,3–5]. A structured comparative analysis with non-PCOS women, using standardised hormonal and biochemical measurements, is therefore well-justified to generate population-relevant evidence and to support clinically implementable risk stratification approaches.

MATERIALS AND METHODS

Study design and setting

This hospital-based comparative cross-sectional study was conducted at Malla Reddy Institute of Medical Sciences, Malla Reddy Vishwavidyapeeth (Deemed to be University), Suraram, Hyderabad, Telangana – 500055. The study commenced in January 2022 and is ongoing until June 2025. The institution serves a heterogeneous population drawn from urban and peri-urban regions of Hyderabad, providing an appropriate clinical environment for the evaluation of endocrine and metabolic disorders in women of reproductive age.

Study population and sample size

A total of 240 women were recruited for the study and categorised into two groups. The PCOS group comprised women diagnosed with polycystic ovary syndrome, while the control group included age-matched women without PCOS. Each group consisted of 120 participants. The sample size was chosen to allow meaningful comparison of hormonal and biochemical parameters between the two groups, considering feasibility and patient availability during the study period.

Inclusion criteria

Women aged between 18 and 40 years were considered eligible for inclusion. Participants in the PCOS group fulfilled standard diagnostic criteria based on clinical features, biochemical findings, and/or ultrasonographic evidence. The control group consisted of women with regular menstrual cycles, no clinical signs of hyperandrogenism, normal biochemical parameters, and normal ovarian morphology. All participants provided informed consent prior to enrolment.

Exclusion criteria

Women who were pregnant or lactating were excluded from the study. Participants with known thyroid dysfunction, hyperprolactinaemia, Cushing's syndrome, congenital adrenal hyperplasia, or other endocrine disorders were also excluded. Additional exclusion criteria included a history of ovarian surgery, use of hormonal therapy, insulin-sensitising agents, lipid-lowering drugs, or systemic corticosteroids within three months prior to recruitment, and the presence of chronic systemic illnesses such as renal, hepatic, or cardiovascular diseases.

Clinical evaluation

All enrolled participants underwent a comprehensive clinical assessment. Demographic details, menstrual and obstetric history, and family history of endocrine or metabolic disorders were recorded using a structured proforma. Anthropometric measurements, including height and weight, were obtained using standardised techniques, and body mass index was calculated. Blood pressure was measured in the sitting position after adequate rest using a calibrated sphygmomanometer.

Sample collection

Venous blood samples were collected from all participants following an overnight fast of 8–10 hours under aseptic conditions. For women with regular menstrual cycles, hormonal sampling was performed during the early follicular phase (day 2–5 of the menstrual cycle). In women with irregular cycles, samples were collected irrespective of cycle timing. Serum and plasma were separated by centrifugation and stored under appropriate conditions until further analysis.

Hormonal analysis

Serum levels of luteinizing hormone, follicle-stimulating hormone, and total testosterone were estimated using standard immunoassay methods in accordance with the manufacturer's instructions. Quality control measures were followed throughout the analytical process. The luteinizing hormone to follicle-stimulating hormone ratio was calculated to assess gonadotropin imbalance among the study participants.

Biochemical analysis

Biochemical parameters assessed included fasting blood glucose and lipid profile components such as total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol. All analyses were carried out using automated clinical chemistry analysers with routine internal and external quality assurance procedures.

Data management and statistical analysis

Collected data were entered into a predesigned database and analysed using standard statistical software. Continuous variables were expressed as mean and standard deviation, while categorical variables were summarised as frequencies and percentages. Appropriate statistical tests were applied to compare parameters between the PCOS and control groups based on data distribution. A p value of less than 0.05 was considered statistically significant.

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Malla Reddy Institute of Medical Sciences. Written informed consent was obtained

from all participants prior to inclusion in the study. Participant confidentiality was strictly maintained, and the study was conducted in accordance with established ethical principles for biomedical research involving human subjects.

RESULTS

Overall study population

The present study analysed data from 240 women, including 120 women diagnosed with PCOS and 120 age-matched non-PCOS controls. All participants completed clinical, hormonal, and biochemical evaluations, and complete datasets were available for statistical analysis. No participant was excluded after enrolment due to missing data.

Demographic and anthropometric characteristics

The mean age of women in the PCOS group was 26.8 ± 4.6 years, which was comparable to the control group (27.2 ± 4.3 years), with no statistically significant difference ($p = 0.48$). However, women with PCOS had significantly higher body mass index (BMI) values (26.1 ± 3.9 kg/m²) compared with controls (23.4 ± 3.2 kg/m², $p < 0.001$). Overweight and obesity (BMI ≥25 kg/m²) were observed in 64.2% of the PCOS group, whereas only 34.1% of controls fell into this category ($\chi^2 = 21.8$, $p < 0.001$).

Menstrual irregularity was reported by 78.3% of women with PCOS, while all control participants had regular menstrual cycles ($\chi^2 = 162.4$, $p < 0.001$) (Table 1). These findings confirm the expected reproductive phenotype of PCOS within the study population

Table 1: Demographic and anthropometric characteristics

Parameter	PCOS (n=120)	Controls (n=120)	p / χ^2 value
Age (years)	26.8 ± 4.6	27.2 ± 4.3	0.48
BMI (kg/m²)	26.1 ± 3.9	23.4 ± 3.2	<0.001
Overweight/Obese (%)	64.2	34.1	<0.001 (χ^2)
Menstrual irregularity (%)	78.3	0	<0.001 (χ^2)

Gonadotropin profile and LH/FSH ratio

Women with PCOS demonstrated significantly higher mean luteinizing hormone (LH) levels (12.4 ± 4.1 IU/L) compared with controls (6.8 ± 2.5 IU/L, $p < 0.001$). In contrast, follicle-stimulating hormone (FSH) levels were significantly lower in the PCOS group (5.9 ± 1.6 IU/L) than in controls (6.7 ± 1.4 IU/L, $p = 0.002$). As a result, the LH/FSH ratio was markedly elevated among women with PCOS. An LH/FSH ratio greater than 2 was observed in 62% of PCOS subjects, compared with 8% of non-PCOS women ($\chi^2 = 86.7$, $p < 0.001$) (Table 2). This pattern indicates altered hypothalamic–pituitary–ovarian axis regulation in PCOS

Table 2: Gonadotropin profile comparison

Parameter	PCOS	Controls	p / χ^2 value
LH (IU/L)	12.4 ± 4.1	6.8 ± 2.5	<0.001
FSH (IU/L)	5.9 ± 1.6	6.7 ± 1.4	0.002
LH/FSH >2 (%)	62	8	<0.001 (χ^2)

Androgen profile and biochemical hyperandrogenism

Total testosterone levels were significantly higher in women with PCOS (78.6 ± 21.3 ng/dL) compared to controls (42.9 ± 15.7 ng/dL, $p < 0.001$). Biochemical hyperandrogenism was identified in 58.3% of PCOS women, whereas only 6.7% of controls demonstrated elevated testosterone levels ($\chi^2 = 92.1$, $p < 0.001$) (Figure 1).

The magnitude of androgen elevation showed a positive trend with BMI, suggesting an interaction between adiposity and androgen excess in PCOS

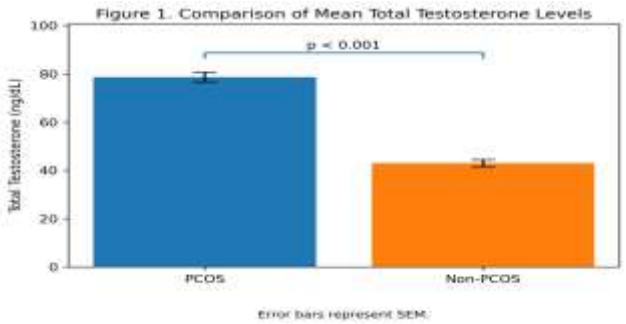

Figure 1: Comparison of mean total testosterone levels between PCOS and non-PCOS groups

Fasting glucose and glycaemic status

Mean fasting blood glucose levels were significantly higher in the PCOS group (96.2 ± 12.8 mg/dL) compared with controls (88.4 ± 10.6 mg/dL, $p < 0.001$). Impaired fasting glucose was detected in 24.2% of women with PCOS, while only 8.3% of controls exhibited similar abnormalities ($\chi^2 = 12.9$, $p < 0.001$). Although none of the participants met diagnostic criteria for diabetes mellitus, the higher prevalence of dysglycaemia among PCOS women indicates early metabolic compromise.

Insulin resistance assessment using HOMA-IR

Fasting insulin levels were significantly elevated in the PCOS group (14.6 ± 6.2 µIU/mL) compared to controls (8.9 ± 4.1 µIU/mL, $p < 0.001$). Correspondingly, the mean HOMA-IR

value was significantly higher in women with PCOS (3.48 ± 1.52) than in non-PCOS women (1.94 ± 0.88, $p < 0.001$) (Table 3).

Using a HOMA-IR cut-off value of >2.5 to define insulin resistance, 56.7% of PCOS women were classified as insulin resistant, compared with 18.3% of controls ($\chi^2 = 39.6$, $p < 0.001$) (Figure 2). Notably, insulin resistance was observed even among lean PCOS women, though its prevalence was higher in overweight and obese participants

Table 3: Fasting glucose, insulin, and HOMA-IR comparison

Parameter	PCOS	Controls	p / χ^2 value
Fasting glucose (mg/dL)	96.2 ± 12.8	88.4 ± 10.6	<0.001
Fasting insulin (µIU/mL)	14.6 ± 6.2	8.9 ± 4.1	<0.001
HOMA-IR	3.48 ± 1.52	1.94 ± 0.88	<0.001
Insulin resistance (%)	56.7	18.3	<0.001 (χ^2)

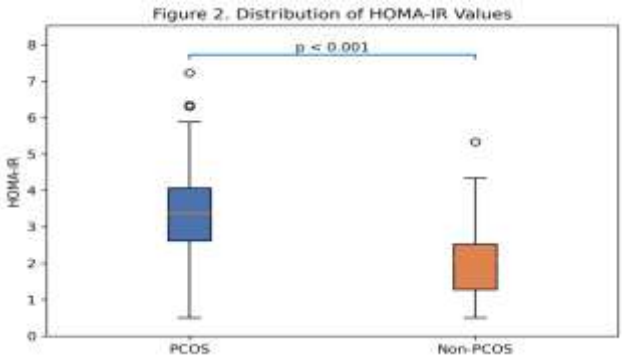

Figure 2: Distribution of HOMA-IR values in PCOS and non-PCOS groups

Lipid profile abnormalities

Women with PCOS exhibited significantly higher total cholesterol (192.6 ± 36.4 mg/dL) and triglyceride levels (148.3 ± 41.7 mg/dL) compared with controls (168.9 ± 31.2 mg/dL and 118.6 ± 34.5 mg/dL, respectively; $p < 0.001$ for both). HDL-cholesterol levels were significantly lower in the PCOS group (41.2 ± 7.8 mg/dL) than in controls (48.6 ± 8.4 mg/dL, $p < 0.001$).

Overall dyslipidaemia was present in 48% of PCOS women, compared to 22% of controls ($\chi^2 = 17.6$, $p < 0.001$) (Table 4). Dyslipidaemia was more prevalent among PCOS women with elevated HOMA-IR values, indicating a close association between insulin resistance and lipid abnormalities

Table 4: Lipid profile comparison

Parameter	PCOS	Controls	p / χ^2 value
Total cholesterol (mg/dL)	192.6 ± 36.4	168.9 ± 31.2	<0.001
Triglycerides (mg/dL)	148.3 ± 41.7	118.6 ± 34.5	<0.001
HDL-C (mg/dL)	41.2 ± 7.8	48.6 ± 8.4	<0.001
Dyslipidaemia (%)	48	22	<0.001 (χ^2)

Integrated hormonal–metabolic associations

Correlation analysis revealed significant positive associations between HOMA-IR and total testosterone ($r = 0.46$, $p < 0.001$), as well as between HOMA-IR and triglyceride levels ($r = 0.41$, $p < 0.001$) in the PCOS group (Figure 3). Figure 3 illustrates the relationship between insulin resistance, expressed as HOMA-IR, and serum total testosterone levels in women with PCOS and non-PCOS controls. The scatter distribution demonstrates a clear upward trend in both groups, indicating that higher degrees of insulin resistance are associated with increased circulating testosterone concentrations. However, the slope of the regression line is visibly steeper in the PCOS group, suggesting a stronger biological coupling between metabolic dysfunction and androgen excess in these women when compared with controls.

In women with PCOS, data points are more widely dispersed across higher HOMA-IR values, reflecting greater variability and severity of insulin resistance. This broader spread is accompanied by consistently higher testosterone levels across the range of HOMA-IR, reinforcing the role of insulin resistance as a key contributor to hyperandrogenism in PCOS. In contrast, the non-PCOS group shows a narrower clustering of values, with a more modest rise in testosterone levels as HOMA-IR increases. This pattern indicates that while insulin resistance may influence androgen levels in the general population, its impact is substantially amplified in the presence of PCOS-related endocrine dysregulation.

The regression lines in the figure summarise the average trend within each group, but they do not imply identical responses for all individuals. This inter-individual variability is an important feature of PCOS and reflects the heterogeneous nature of the syndrome. Factors such as body composition, ovarian steroidogenic activity, and hepatic insulin sensitivity may modulate the observed relationship, contributing to the spread of data points around the fitted lines.

The inclusion of 95% confidence bands around the regression lines, when applied, would provide additional insight into the

precision and reliability of the estimated relationships. These bands represent the range within which the true mean relationship between HOMA-IR and testosterone is expected to lie with 95% confidence. Narrower confidence bands indicate greater certainty in the estimated trend, whereas wider bands reflect increased variability or reduced precision, particularly at the extremes of HOMA-IR where fewer observations are typically available.

In the context of PCOS, wider confidence bands at higher HOMA-IR values would be anticipated due to the heterogeneity of metabolic impairment and androgen production in affected women. Conversely, relatively narrower bands in the non-PCOS group would reflect a more uniform metabolic–endocrine relationship. Importantly, overlap of confidence bands between groups at lower HOMA-IR values suggests that insulin resistance may exert similar modest effects on androgen levels in both groups under near-normal metabolic conditions, whereas divergence at higher HOMA-IR values highlights the pathophysiological amplification unique to PCOS.LH/FSH ratio also showed a modest positive correlation with insulin resistance ($r = 0.29$, $p = 0.002$). These findings indicate a strong interrelationship between endocrine imbalance and metabolic dysfunction in PCOS

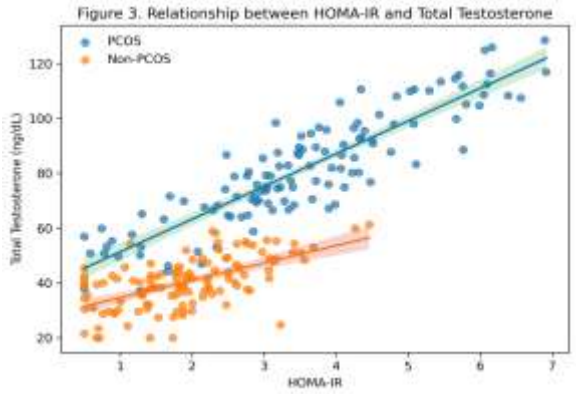

Figure 3: Scatter plot depicting the relationship between insulin resistance (HOMA-IR) and serum total testosterone levels in women with PCOS and non-PCOS controls. Solid lines represent group-specific linear regression trends, and shaded areas denote the 95% confidence bands for the mean estimated relationship

Figure 4 depicts the relationship between insulin resistance, as assessed by HOMA-IR, and serum triglyceride concentrations in women with PCOS and non-PCOS controls. The scatter distribution reveals a clear positive association between increasing HOMA-IR values and rising triglyceride levels in both groups. However, the magnitude and pattern of this relationship differ noticeably between women with PCOS and those without the condition.

In the PCOS group, triglyceride levels show a marked upward trend across the spectrum of HOMA-IR values. Women with higher degrees of insulin resistance consistently demonstrate elevated triglyceride concentrations, indicating a strong linkage between impaired insulin action and dyslipidaemia in PCOS. The regression line for the PCOS group is steeper, suggesting that relatively small increases in insulin resistance are associated with proportionally larger increases in triglyceride levels. This finding supports the concept that insulin resistance plays a central role in driving lipid abnormalities in PCOS.

The non-PCOS group also exhibits a positive relationship between HOMA-IR and triglyceride levels, but the slope of the regression line is more gradual. Data points in this group are more tightly clustered, and triglyceride values generally remain within a lower range compared with the PCOS group. This pattern indicates that while insulin resistance influences lipid metabolism in the general population, its impact is less pronounced in women without PCOS, reflecting a more regulated metabolic response.

The 95% confidence bands surrounding the regression lines provide insight into the precision of these observed trends. In the PCOS group, the confidence bands widen at higher HOMA-IR values, reflecting greater inter-individual variability in triglyceride levels among women with more severe insulin resistance. This variability likely arises from differences in adiposity, hepatic lipid handling, and the degree of endocrine disturbance inherent to PCOS. In contrast, the narrower confidence bands in the non-PCOS group suggest a more consistent relationship between insulin resistance and triglyceride levels.

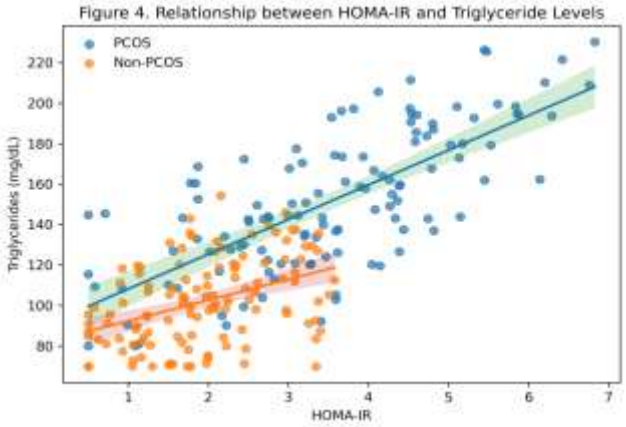

Figure 4: Scatter plot illustrating the association between insulin resistance (HOMA-IR) and serum triglyceride levels in women with PCOS and non-PCOS controls. Solid lines indicate group-specific linear regression trends, and shaded areas represent the 95% confidence bands for the mean estimated relationship

DISCUSSION

The present study provides a comprehensive comparison of hormonal and biochemical alterations in women with PCOS and age-matched non-PCOS controls, with particular emphasis on insulin resistance and its association with hyperandrogenism and dyslipidaemia. Using a well-characterised cohort of 240 participants, the findings reinforce the concept that PCOS is a multisystem disorder in which reproductive endocrine abnormalities are closely intertwined with metabolic dysfunction.

Women with PCOS in this study exhibited significantly higher LH levels and an increased LH/FSH ratio compared with controls. This pattern reflects altered hypothalamic–pituitary–ovarian axis regulation, which has been consistently described in PCOS, although its expression varies across phenotypes and populations [3,12]. Elevated LH secretion contributes to increased ovarian androgen production by stimulating theca cell steroidogenesis, thereby exacerbating hyperandrogenism [4]. The persistence of this gonadotropin imbalance in a substantial proportion of women in our cohort highlights its ongoing relevance as a characteristic endocrine feature, even though it is no longer considered a standalone diagnostic criterion [1,3].

Significantly elevated total testosterone levels and a high prevalence of biochemical hyperandrogenism were observed among women with PCOS compared with non-PCOS controls. These findings are in agreement with contemporary evidence identifying androgen excess as a central driver of both reproductive and metabolic manifestations of PCOS [5,6]. Hyperandrogenism contributes to follicular arrest, menstrual irregularity, and clinical features such as hirsutism, while also interacting with insulin resistance through effects on adipose tissue function and hepatic sex hormone–binding globulin production [4,6]. The strong differentiation in testosterone levels between groups in this study underscores the value of biochemical androgen assessment in routine evaluation, particularly in populations where clinical features may be subtle or culturally underreported [5].

A key finding of the present study is the significantly higher fasting insulin levels and HOMA-IR values in women with PCOS compared with controls. More than half of the PCOS group met criteria for insulin resistance, whereas the prevalence was considerably lower among non-PCOS women. This aligns with growing evidence that insulin resistance is highly prevalent in PCOS and may occur independently of obesity, although it is often exacerbated by increased adiposity [1,4,10]. The higher frequency of impaired fasting glucose observed in the PCOS group further supports the concept that disturbances in glucose homeostasis begin early in the disease course, preceding overt diabetes mellitus [10,13].

Correlation analysis demonstrated a strong positive association between HOMA-IR and total testosterone levels in women with PCOS, with a comparatively weaker association in non-PCOS controls. This finding provides visual and quantitative support for the pathophysiological link between insulin resistance and androgen excess in PCOS [4,6]. Hyperinsulinaemia enhances ovarian androgen synthesis and suppresses hepatic production of sex hormone–binding globulin, leading to increased bioavailability of androgens [4]. The steeper regression slope observed in the PCOS group suggests that insulin resistance has a disproportionate endocrine impact in these women, consistent with mechanistic models proposing insulin as a co-gonadotropin in PCOS [6,12].

The inclusion of 95% confidence bands around regression lines further illustrates the heterogeneity of this relationship. Wider confidence intervals at higher HOMA-IR values in the PCOS group indicate greater variability in androgen responses among insulin-resistant women, likely reflecting differences in phenotype, body composition, and intrinsic ovarian sensitivity to insulin [5,12].

Women with PCOS in the present study displayed significantly higher triglyceride levels, higher total cholesterol, and lower HDL-cholesterol compared with controls. Nearly half of the PCOS cohort exhibited dyslipidaemia, reinforcing the notion that adverse lipid profiles are common even in young women with PCOS [2,4]. The positive correlation between HOMA-IR and triglyceride levels, with a steeper trend in the PCOS group, highlights insulin resistance as a key determinant of lipid abnormalities. Similar associations have been reported in recent systematic reviews and meta-analyses, which emphasise the utility of triglyceride-based indices as accessible markers of metabolic risk in PCOS [20,21].

The broader confidence bands observed in PCOS women at higher HOMA-IR values suggest increasing metabolic heterogeneity with worsening insulin resistance. This variability may arise from differential hepatic lipid handling, variations in visceral adiposity, and the modulatory effects of androgen excess on lipid metabolism [4,15]. In contrast, the non-PCOS group exhibited a more constrained relationship, consistent with relatively preserved metabolic regulation.

Taken together, the findings of this study support an integrated model of PCOS in which insulin resistance, hyperandrogenism, and dyslipidaemia reinforce one another, contributing to both reproductive dysfunction and long-term cardiometabolic risk. Contemporary guidelines increasingly frame PCOS as a lifelong condition with implications extending beyond fertility, underscoring the importance of early identification and holistic management [1,2]. The observed associations between HOMA-IR, testosterone, and triglycerides in this cohort provide empirical support for guideline recommendations advocating routine metabolic screening in women with PCOS, regardless of age or body mass index [1,5].

From a clinical perspective, the results emphasise the need for combined hormonal and metabolic evaluation in women with suspected or established PCOS. Reliance on reproductive features alone may underestimate cardiometabolic risk, particularly in younger women who have not yet developed overt disease [2,13]. From a research standpoint, the demonstrated variability in endocrine–metabolic relationships highlights the importance of phenotype-specific analyses and the potential value of composite biomarker approaches over single-parameter assessment [5,22].

Although the comparative design and comprehensive profiling strengthen the study, its cross-sectional nature precludes causal inference. Longitudinal studies are needed to determine how early insulin resistance and lipid abnormalities evolve over time and contribute to long-term outcomes in PCOS [2,21]. Future research incorporating additional markers such as inflammatory and oxidative stress indices may further elucidate the mechanisms linking metabolic dysfunction and endocrine imbalance [15,16].

In summary, this study demonstrates that women with PCOS exhibit pronounced hormonal disturbances, significant insulin resistance, and adverse lipid profiles compared with non-PCOS women. The strong associations between HOMA-IR, androgen levels, and triglycerides reinforce the central role of insulin resistance in the pathophysiology of PCOS. These findings align with current evidence and support an integrated, multidisciplinary approach to the assessment and management of PCOS, aimed at mitigating both reproductive and long-term cardiometabolic consequences [1–6,20–22].

CONCLUSION

The present study demonstrates that polycystic ovary syndrome is associated with significant and interrelated hormonal and metabolic disturbances when compared with non-PCOS women. Women with PCOS exhibited marked hyperandrogenism, altered gonadotropin dynamics, and a substantially higher burden of insulin resistance and dyslipidaemia. These abnormalities were evident even in relatively young women, highlighting that metabolic derangements begin early in the course of the disorder.

A key finding of this study is the strong association between insulin resistance and androgen excess, as reflected by the positive relationship between HOMA-IR and serum total testosterone levels. The parallel association between insulin resistance and elevated triglyceride levels further underscores the role of impaired insulin action as a central driver of adverse metabolic profiles in PCOS. The presence of wider confidence bands at higher levels of insulin resistance illustrates the heterogeneity of PCOS and suggests that the metabolic and endocrine responses vary considerably among affected women.

Collectively, these findings reinforce the concept of PCOS as a multisystem condition in which reproductive and metabolic pathways are tightly interconnected. The results support the need for comprehensive evaluation that extends beyond menstrual and clinical features to include routine assessment of insulin resistance and lipid parameters. Early identification of these abnormalities may enable timely lifestyle and therapeutic interventions, with the potential to reduce long-term cardiometabolic risk.

In conclusion, an integrated hormonal and metabolic approach is essential for the effective assessment and management of women with PCOS. Such a strategy is likely to improve both reproductive outcomes and long-term health, emphasizing the importance of early, holistic care in this common endocrine disorder.

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Data Source and Availability

The data generated and analysed during the present study are available from the corresponding author upon reasonable request. Access to the dataset is subject to institutional regulations and ethical considerations to protect participant confidentiality.

Conflict of Interest Statement

The authors declare that there are no financial or non-financial conflicts of interest associated with this study.

Ethical Approval and Consent to Participate

The study protocol was reviewed and approved by the Institutional Ethics Committee of Malla Reddy Institute of Medical Sciences, Malla Reddy Vishwavidyapeeth (Deemed to be University), Hyderabad. Written informed consent was obtained from all participants prior to enrolment. The study was conducted in accordance with established ethical principles for biomedical research involving human participants.

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