CORRELATION OF HORMONAL PROFILES IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME (PCOS)

Harsharan Singh,[1] Gagandeep Singh,[2] Musaib Shameem, [3] Parveen Bansal[4]

[1]Department of Allied health sciences Desh Bhagat University, Fatehgarh sahib,India

[2]Assistant professor, Allied health sciences Desh Bhagat University, Fatehgarh sahib,India

[3]Assistant Professor, Faculty of Allied HealthCare and Sciences, Desh Bhagat University, India

[4]Director, Director, Faculty of Allied HealthCare and Sciences, Desh Bhagat University, Punjab, India

ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder characterized by oligo/anovulation, hyperandrogenism, and polycystic ovarian morphology. Hormonal disturbances, such as elevated luteinizing hormone (LH), increased testosterone, and anti-Müllerian hormone (AMH), along with insulin resistance, contribute to the metabolic and reproductive challenges of PCOS. This study explores the correlation between various hormonal parameters in PCOS to better understand the hormonal interplay in this disorder.A cross-sectional study was conducted from January to May 2025 at Life Line Ludhiana, involving 500 women aged 18-40 years diagnosed with PCOS according to the Rotterdam criteria. Exclusion criteria included pregnancy, thyroid dysfunction, hyperprolactinemia, adrenal disorders, and recent use of hormonal medications. Hormonal assays were performed to measure LH, FSH, testosterone, dehydroepiandrosteronesulfate (DHEAS), AMH, fasting insulin, and glucose. Insulin resistance was calculated using HOMA-IR. Pearson and Spearman correlation coefficients were calculated to assess relationships between hormonal parameters. The study included 500 women with a mean age of 27.8 ± 5.4 years and a BMI of 28.3 ± 4.6 kg/m². 82% of participants had oligo/anovulation, and 75% exhibited biochemical hyperandrogenism.Hormonal assays revealed elevated LH (12.4 IU/L), testosterone (78.5 ng/dL), AMH (7.2 ng/mL), and fasting insulin (18.6 µIU/mL), indicating significant metabolic dysfunction.Significant positive correlations were observed between LH, total testosterone, and AMH ($r = 0.38–0.45$, $p < 0.001$). Fasting insulin strongly correlated with HOMA-IR ($r = 0.98$, $p < 0.001$), and a moderate correlation was found between insulin and testosterone ($r = 0.35$, $p < 0.001$).Subgroup analysis by BMI showed that overweight/obese women had significantly higher insulin and HOMA-IR levels compared to normal-weight women, with a stronger correlation between insulin and testosterone in the overweight/obese group.This study demonstrates significant positive correlations between LH, total testosterone, AMH, and insulin resistance in women with PCOS. These associations were notably amplified in overweight/obese individuals, emphasizing the role of excess adiposity in worsening insulin resistance and hyperandrogenism. The findings support the importance of integrated management strategies that address both metabolic and reproductive aspects of PCOS.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with a global prevalence estimated between 6% and 21% depending on the diagnostic criteria used [1,2]. Characterized by oligo- or anovulation, clinical and/or biochemical hyperandrogenism, and polycystic ovarian morphology, PCOS is a multifaceted condition with significant reproductive, metabolic, and psychological consequences [3].

The hormonal milieu in PCOS is notably disrupted. Key hormonal alterations include increased luteinizing hormone (LH) secretion, elevated androgen levels (such as testosterone and dehydroepiandrosteronesulfate), and often, increased anti-Müllerian hormone (AMH) [4,5]. Additionally, many patients exhibit hyperinsulinemia and insulin resistance, which not only contribute to metabolic complications but also exacerbate the reproductive and hyperandrogenic features of the syndrome [6]. The interrelationships among these hormonal disturbances are complex and have been the subject of ongoing research.

Understanding the correlations between various hormonal parameters in PCOS patients is crucial for improving diagnostic accuracy, risk stratification, and individualized management strategies. However, the exact patterns and clinical implications of these correlations remain incompletely understood [7]. Therefore, this study aims to analyse the correlation of key hormonal profiles—including LH, follicle-stimulating hormone (FSH), testosterone, insulin, AMH, and others—in patients diagnosed with PCOS, to better elucidate the hormonal interplay underlying this prevalent disorder.

REVIEW OF LITERATURE

1. **Xu et al. (2024)** conducted a prospective cohort

study investigating the relationship between anti-Müllerian hormone (AMH) and hyperandrogenism in women with PCOS. Their results demonstrated a significant positive correlation between AMH levels and serum testosterone, as well as ovarian volume, suggesting that AMH serves as both a marker of follicular excess and androgenic activity in PCOS [8]. This study supports the role of AMH as a valuable biomarker in understanding PCOS pathophysiology and aligns with our findings of strong AMH-testosterone associations.

2. **Chen et al. (2023)** performed a cross-sectional analysis examining the impact of body mass index (BMI) on insulin resistance and hyperandrogenism in PCOS patients. They found that overweight and obese women exhibited higher fasting insulin and HOMA-IR levels, which correlated strongly with androgen excess compared to normal-weight counterparts [9]. Their research highlights obesity as a key modifier of metabolic and hormonal disturbances in PCOS, consistent with our subgroup analysis showing amplified correlations in overweight/obese patients.

3. **Al-Shami et al. (2023)** explored the regulation of adrenal androgens and gonadotropins in PCOS, reporting that dehydroepiandrosteronesulfate (DHEAS) levels did not significantly correlate with follicle-stimulating hormone (FSH), implying independent regulatory mechanisms [10]. This finding mirrors our observation of no significant FSH-DHEAS correlation, underscoring the heterogeneity of androgen sources in PCOS and the need for comprehensive hormonal assessment

MATERIALS AND METHODS

Study Design and Participants

This cross-sectional study was conducted at life line Ludhiana from January 2025 to May 2025. A total of 500 women aged 18–40 years diagnosed with PCOS according to the Rotterdam criteria were recruited. Exclusion criteria included pregnancy, thyroid dysfunction, hyperprolactinemia, adrenal disorders, and use of hormonal medications within the last three months.

Data Collection

Demographic and clinical data including age, body mass index (BMI), menstrual history, and signs of hyperandrogenism were recorded. Blood samples were collected during the early follicular phase (days 2–5 of the menstrual cycle) after an overnight fast.

Hormonal Assays

Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone, dehydroepiandrosterone sulphate (DHEAS), anti-Müllerian hormone (AMH), fasting insulin, and glucose were measured using standardized immunoassays at the [Name of Laboratory]. Insulin resistance was estimated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) calculated as fasting insulin (μIU/mL) × fasting glucose (mg/dL) / 405.

Statistical Analysis

Data were analysed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation or median (interquartile range) depending on distribution. Pearson or Spearman correlation coefficients were calculated to assess relationships between hormonal parameters. A p-value < 0.05 was considered statistically significant.

RESULTS

The study included 500 women diagnosed with PCOS, with a mean age of 27.8 ± 5.4 years and a mean body mass index (BMI) of 28.3 ± 4.6 kg/m². Clinical features included oligo/anovulation in 82% of patients and biochemical hyperandrogenism in 75%.

Parameter	Mean ± SD
Age (years)	27.8 ± 5.4
Body Mass Index (BMI, kg/m²)	28.3 ± 4.6
Oligo/Anovulation (%)	82%
Biochemical Hyperandrogenism (%)	75%

Table 1: Baseline Characteristics of PCOS Patients (n=500)

Table 1 illustrates that PCOS patients (n=500) were predominantly young (27.8 ± 5.4 years) and overweight (BMI 28.3 ± 4.6 kg/m²). Oligo/anovulation was highly prevalent (82%), and 75% exhibited biochemical hyperandrogenism. This confirms that menstrual irregularities and androgen excess are core clinical features, while elevated BMI indicates potential metabolic risk, typical of PCOS populations.

Hormone/Parameter	Mean ± SD	Units
Luteinizing Hormone (LH)	12.4 ± 6.1	IU/L
Follicle Stimulating Hormone (FSH)	5.8 ± 2.3	IU/L
Total Testosterone	78.5 ± 32.4	ng/dL
DehydroepiandrosteroneSulfate (DHEAS)	235 ± 110	µg/dL
Anti-Müllerian Hormone (AMH)	7.2 ± 3.5	ng/mL
Fasting Insulin	18.6 ± 9.2	µIU/mL
Fasting Glucose	92.4 ± 11.3	mg/dL
HOMA-IR	4.3 ± 2.1	(unitless)

Table 2: Hormonal Profile Summary

Table 2 illustrates that the hormonal profile reveals elevated LH (12.4 IU/L) and total testosterone (78.5 ng/dL), with a high AMH (7.2 ng/mL), all typical of PCOS. Fasting insulin (18.6 µIU/mL) and HOMA-IR (4.3) are increased, indicating significant insulin resistance. Fasting glucose remains within normal limits, suggesting compensatory hyperinsulinemia. These data highlight the interplay between hyperandrogenism and metabolic dysfunction in PCOS, emphasizing the syndrome's dual endocrine and metabolic nature.

Parameter 1	Parameter 2	Correlation Coefficient (r)	p-value
LH	Total Testosterone	0.42	< 0.001
LH	AMH	0.38	< 0.001
AMH	Total Testosterone	0.45	< 0.001
AMH	LH/FSH Ratio	0.40	< 0.001
Fasting Insulin	Total Testosterone	0.35	< 0.001
Fasting Insulin	HOMA-IR	0.98	< 0.001
FSH	DHEAS	0.08	0.12 (NS)

Table 3: Correlations Between Hormonal Parameters

Table 3 reveals that the significant positive correlations exist between LH, AMH, and total testosterone ($r = 0.38–0.45$, $p < 0.001$), indicating that higher gonadotropin and ovarian reserve markers are associated with greater androgen excess. Fasting insulin strongly correlates with HOMA-IR ($r = 0.98$), confirming HOMA-IR as a robust surrogate for insulin resistance. The moderate correlation between insulin and testosterone ($r = 0.35$) suggests metabolic factors contribute to hyperandrogenism in PCOS.

Parameter	Normal Weight (BMI	Overweight/Obese	p-value
Fasting Insulin (μIU/mL)	14.2 ± 6.5	21.3 ± 9.8	< 0.001
HOMA-IR	3.1 ± 1.4	5.2 ± 2.3	< 0.001
Correlation (Insulin & Testosterone)	r = 0.25 (moderate)	r = 0.40 (stronger)	—

Table 4: Subgroup Analysis by BMI Category

Table 4 illustrates that Overweight/obese PCOS patients exhibit significantly higher fasting insulin (21.3 vs. 14.2 μIU/mL) and HOMA-IR (5.2 vs. 3.1) compared to normal-weight counterparts ($p < 0.001$), indicating greater insulin resistance. The correlation between insulin and testosterone is stronger in the overweight/obese group ($r = 0.40$ vs. 0.25), suggesting that excess adiposity amplifies the link between metabolic dysfunction and hyperandrogenism, reinforcing the importance of weight management in PCOS care

DISCUSSION

Our study among 500 women with PCOS demonstrated significant positive correlations between LH, total testosterone, AMH, and insulin resistance, with these associations being especially notable in overweight and obese individuals. These findings are consistent with and expand upon recent research investigating the hormonal and metabolic interplay characteristic of PCOS.

The positive correlation identified between LH and total testosterone aligns with recent multicentre studies, which confirmed that elevated LH remains a hallmark of the classic PCOS phenotype, contributing to increased ovarian androgen production and follicular arrest [11]. Similarly, our observation of heightened AMH levels correlating with both LH and testosterone supports recent evidence that AMH is not only a marker of follicular excess but also reflects the degree of hyperandrogenism and disrupted folliculogenesis in PCOS [12]. A 2024 prospective cohort study by Xu et al. demonstrated that AMH is significantly associated with serum testosterone and ovarian volume, reinforcing its diagnostic and prognostic value [8].

The strong association between fasting insulin, HOMA-IR, and total testosterone in our cohort mirrors the findings of recent meta-analyses, which have shown that insulin resistance is a key driver of hyperandrogenism in PCOS, acting both directly and indirectly through suppression of SHBG and enhancement of ovarian androgen synthesis [6,9]. Notably, our subgroup analysis revealed that this relationship was more pronounced in overweight and obese women, consistent with the findings of Chen et al. (2023), who reported that BMI amplifies the metabolic and reproductive manifestations of PCOS, particularly insulin resistance and androgen excess [9].

In contrast, our study did not identify a significant correlation between FSH and DHEAS, paralleling the results from a 2023 investigation by Al-Shami et al., which suggested that adrenal androgens may be regulated independently of gonadotropin dynamics in PCOS [10]. This highlights the syndrome's heterogeneity and underscores the importance of comprehensive hormonal profiling.

Collectively, these results reinforce the view that PCOS is a multifactorial disorder with intricate hormonal and metabolic interrelationships. Understanding these correlations is crucial for refining diagnostic strategies and tailoring individualized treatment plans. Our findings support recent calls for integrated management approaches addressing both metabolic and reproductive aspects of PCOS [13].

Limitations of this study include its cross-sectional design and single-centre recruitment, which may limit generalizability. Future longitudinal and multicentre studies are warranted to further clarify the temporal and causal relationships among these hormonal parameters.

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