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ASSESMENT OF POLYCYSTIC OVARY SYNDROME THE STUDY OF PROLACTIN AND FOLLICLE STIMULATING HORMONE

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Abstract

Background: *Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder in reproductive-age women, frequently accompanied by hyperprolactinemia. The interplay between prolactin, follicle-stimulating hormone (FSH), and metabolic parameters in PCOS remains incompletely characterized.* ***Objective:*** *To assess the prevalence of hyperprolactinemia among PCOS patients and examine its associations with FSH, LH/FSH ratio, BMI, and metabolic markers. Methods: A cross-sectional study was conducted at a tertiary hospital in Lahore involving 100 female participants (age 21–27 years; mean 23.95 ± 1.73). PCOS was diagnosed per Rotterdam criteria. Serum prolactin, FSH, LH, testosterone, and BMI were measured. Descriptive statistics, Pearson/Spearman correlations, independent t-tests, and binary logistic regression were performed in SPSS. Results: All 100 participants were hyperprolactinemic (prolactin >25 ng/mL; mean 35.78 ± 5.73 ng/mL), and 81% met PCOS criteria. The mean LH/FSH ratio was 2.82 (>2 threshold), and mean testosterone was 87.36 ng/dL. BMI was very strongly correlated with prolactin ($r = 0.984$, $p < 0.001$) and negatively with FSH ($r = -0.971$, $p < 0.001$). Prolactin and FSH were inversely correlated ($r = -0.969$; Spearman $\rho = -0.994$, $p < 0.001$). No statistically significant differences in hormonal parameters were found between PCOS-positive and PCOS-negative groups, attributable to uniform hyperprolactinemia across the cohort. Logistic regression was non-significant overall, though LH/FSH ratio showed the highest odds ratio (OR = 19.131).* ***Conclusion:*** *Hyperprolactinemia is common in PCOS and is strongly associated with elevated BMI and suppressed FSH, suggesting a hormonal axis disruption contributing to anovulation. Routine prolactin assessment is warranted in PCOS evaluation. Larger controlled studies are needed to establish causality and evaluate prolactin-targeted therapies.*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder and a leading cause of anovulatory infertility among women of reproductive age, affecting approximately 4–20% of women worldwide depending on the diagnostic criteria and population studied (Teede et al., 2023). According to the Rotterdam consensus, PCOS is diagnosed when at least two of the following three features are present: oligo/anovulation, clinical or biochemical hyperandrogenism,

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and polycystic ovarian morphology on ultrasound, after exclusion of related endocrine disorders (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004). Beyond reproductive dysfunction, PCOS is increasingly recognized as a complex metabolic disorder associated with obesity, insulin resistance, dyslipidemia, and chronic low-grade inflammation (Teede et al., 2023). Prolactin is a pleiotropic hormone primarily secreted by lactotroph cells of the anterior pituitary gland and plays important roles in lactation, reproductive physiology, immune regulation, glucose metabolism, and lipid homeostasis (Khan et al., 2024). Hyperprolactinemia, commonly defined as serum prolactin concentrations exceeding 25 ng/mL in women, is among the most frequent endocrine abnormalities affecting the hypothalamic–pituitary–gonadal axis (Melmed et al., 2011). Previous studies have reported that approximately 13–37% of women with PCOS exhibit elevated prolactin levels, suggesting a potential pathophysiological relationship between hyperprolactinemia and PCOS (Davoudi et al., 2021; Kamrul-Hasan & Aalpona, 2024). Elevated prolactin may impair reproductive function through suppression of pulsatile gonadotropin-releasing hormone (GnRH) secretion, resulting in reduced follicle-stimulating hormone (FSH) activity, impaired follicular maturation, menstrual irregularities, and anovulation (Khan et al., 2024).

Objectives

1. To investigate the impact of prolactin in Polycystic Ovary Syndrome (PCOS).
2. To investigate the impact of Follicle Stimulating Hormone (FSH) in Polycystic Ovary Syndrome (PCOS).
3. To determine the rate of prevalence of hyperprolactinemia in PCOS and their correlation with FSH levels.

RESEARCH QUESTIONS:

1. What is the difference in the LH/FSH ratio between several types of PCOS, and is it related to the degree of ovulatory dysfunction?
2. What is the prevalence of hyperprolactinemia in PCOS patients without macroprolactinoma?
3. Is there a relationship between prolactin concentration and insulin resistance among PCOS patients irrespective of BMI

Despite growing evidence supporting an association between prolactin and PCOS, the clinical significance of hyperprolactinemia and its relationship with gonadotropin dynamics, particularly FSH, remains insufficiently understood. Furthermore, data from South Asian populations are limited. Therefore, this study aimed to evaluate the prevalence of hyperprolactinemia in women with PCOS and investigate the relationships among prolactin, FSH, LH/FSH ratio, body mass index (BMI), and testosterone levels. Understanding these associations may contribute to improved diagnostic evaluation and personalized management strategies for women affected by PCOS.

2. Materials and Methods

Study design and setting: A cross-sectional observational study was conducted at a tertiary care gynecology and endocrinology outpatient department in Lahore, Pakistan. Institutional ethics approval was obtained, and all participants provided written informed consent.

Participants: Women aged 18–40 years presenting with irregular menstrual cycles, hirsutism, infertility, or suspected PCOS were screened. PCOS was diagnosed using Rotterdam criteria (2 of 3: oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovaries on ultrasound). **Sample size:** Using an estimated prevalence of hormonal imbalance of 70% and a 10% margin of error (Z = 1.96), the minimum sample size was calculated as 81, increased to 100 to account for attrition.

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Laboratory measurements: Serum FSH, LH, and prolactin were measured by chemiluminescence immunoassay (CLIA). Elevated prolactin (>25 ng/mL) was confirmed on repeat sampling. The LH/FSH ratio, BMI, fasting glucose, insulin (for HOMA-IR), lipid profile, and thyroid function tests were also obtained. Testosterone was measured by standard immunoassay.

Statistical analysis: Data were analyzed using IBM SPSS Statistics. Descriptive statistics included mean, SD, and frequency distributions. Normality was assessed using Kolmogorov-Smirnov and Shapiro-Wilk tests. Group comparisons (PCOS-positive vs. PCOS-negative) were made using independent samples t-tests. Pearson and Spearman rank correlations were computed. Binary logistic regression was used to assess the ability of hormonal variables to predict PCOS diagnosis.

3. Results

3.1 Participant Overview

A total of 100 female participants were enrolled (mean age 23.95 ± 1.73 years; range 21–27). All 100 participants had prolactin levels >25 ng/mL, making the entire cohort hyperprolactinemic. Of these, 81 (81%) were diagnosed with PCOS (PCOS-positive) and 19 (19%) were PCOS-negative.

Characteristic	n (%) or Mean ± SD
Age (years)	23.95 ± 1.73
Age Range (years)	21–27
Hyperprolactinemia (>25 ng/mL)	100 (100.0%)
PCOS-Positive	81 (81.0%)
PCOS-Negative	19 (19.0%)

3.2 Hormonal and Clinical Profile

Descriptive Statistics

Table 2 presents the descriptive statistics for all key hormonal and clinical variables. Prolactin levels ranged from 26.8 to 48.5 ng/mL with a mean of 35.78 ± 5.73 ng/mL, confirming elevated prolactin in all participants. Mean BMI was 28.59 ± 1.74 kg/m², indicating overweight status across the sample. LH/FSH Ratio had a mean of 2.82, exceeding the normal threshold of <2, which is a hallmark indicator of PCOS. Testosterone levels (mean 87.36 ng/dL) were also elevated beyond the normal female reference range.

Table 2 Descriptive Statistics of Key Hormonal and Clinical Parameters

Variable	N	Min	Max	Mean	SD	Reference Range
Prolactin (ng/mL)	100	26.8	48.5	35.78	5.73	2–29 ng/mL
BMI (kg/m²)	100	25.6	32.3	28.59	1.74	18.5–24.9 (normal)
Age (years)	100	21	27	23.95	1.73	21–27
FSH (mIU/mL)	100	4.0	5.3	4.63	0.36	3.5–12.5 mIU/mL
LH (mIU/mL)	100	8.6	17.9	12.97	2.83	2.4–12.6 mIU/mL
LH/FSH Ratio	100	1.71	4.22	2.82	0.64	<2 (normal)

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Variable	N	Min	Max	Mean	SD	Reference Range
Testosterone (ng/dL)	100	55.5	119.1	87.36	19.05	15–70 ng/dL (female)

Note: SD = Standard Deviation Reference ranges sourced from standard clinical laboratory guidelines. All N = 100 (no missing values).

3.3 Group Comparisons

Independent samples t-tests revealed no statistically significant differences in any hormonal parameter between PCOS-positive and PCOS-negative groups (all $p > 0.05$; Table 2). This null finding likely reflects the uniformly hyperprolactinemic state shared by both groups, which may mask inter-group hormonal distinctions.

Table 1 Independent Samples T-Test Group Statistics and Significance

Variable	PCOS-Positive (n = 81) *M* ± *SD*	PCOS-Negative (n = 19) *M* ± *SD*	*t*(98)	*p*
Prolactin (ng/mL)	35.65 ± 5.69	36.30 ± 6.03	-0.44	.661
FSH (mIU/mL)	4.64 ± 0.36	4.58 ± 0.36	0.58	.561
LH (mIU/mL)	12.82 ± 2.83	13.58 ± 2.81	-1.05	.296
LH/FSH Ratio	2.78 ± 0.63	2.98 ± 0.68	-1.25	.215
BMI (kg/m²)	28.54 ± 1.71	28.80 ± 1.86	-0.58	.560
Testosterone (ng/dL)	86.13 ± 18.97	92.58 ± 19.01	-1.33	.185

Note: All p-values are two-tailed. Equal variances assumed based on Levene's Test results. ns = not significant ($p > 0.05$).

3.4 Correlation Analysis

Pearson correlation analysis revealed robust relationships (Table 3). BMI was very strongly positively correlated with prolactin ($r = 0.984$, $p < 0.001$) and very strongly negatively correlated with FSH ($r = -0.971$, $p < 0.001$). Prolactin and FSH showed a very strong inverse association ($r = -0.969$, $p < 0.001$). LH was very strongly correlated with LH/FSH ratio ($r = 0.935$, $p < 0.001$). Spearman correlations confirmed these findings (BMI–Prolactin $\rho = 0.981$; FSH–Prolactin $\rho = -0.994$; all $p < 0.001$), validating robustness against non-normality. Testosterone showed no significant correlation with any variable (all $p > 0.05$).

Table 3.4

Pearson Correlation Matrix of Key Variables

Variable	1	2	3	4	5	6
1. BMI	—					
2. FSH	−.971**	—				
3. LH	−.048	.059	—			
4. Prolactin	.984**	−.969**	−.067	—		
5. LH/FSH Ratio	.295**	−.290**	.935**	.277**	—	
6. Testosterone	−.119	.100	−.034	−.119	−.069	—

Note: Correlation significant at 0.01 level (2-tailed). Values in bold indicate strong correlations ($|r| > 0.7$).

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3.5 Logistic Regression

Binary logistic regression including BMI, FSH, prolactin, LH, LH/FSH ratio, and testosterone as predictors of PCOS diagnosis was not statistically significant overall ($\chi^2 = 4.383$, df = 6, p = 0.625; Nagelkerke $R^2 = 0.069$). The Hosmer-Lemeshow test confirmed adequate model fit (p = 0.733). Among individual predictors, LH/FSH ratio showed the highest odds ratio (OR = 19.131), though it did not reach significance (p = 0.583), likely reflecting limited statistical power in this uniformly hyperprolactinemic sample.

Table 3.5

Binary Logistic Regression Analysis Predicting PCOS Status (N = 100)

Predictor	B	SE	Wald χ^2	df	*p*	OR (Exp[B])
BMI (kg/m²)	0.504	0.953	0.280	1	.597	1.66
FSH (mIU/mL)	0.723	4.084	0.031	1	.860	2.06
Prolactin (ng/mL)	-0.189	0.273	0.476	1	.490	0.83
LH (mIU/mL)	-0.550	1.182	0.217	1	.642	0.58
LH/FSH Ratio	2.951	5.376	0.301	1	.583	19.13
Testosterone (ng/dL)	0.020	0.014	1.985	1	.159	1.02
Constant	-15.488	34.274	0.204	1	.651	—

Note: B = regression coefficient; S.E. = standard error; Wald = Wald chi-square statistic; Exp (B) = Odds Ratio; ns = not significant (p > 0.05). Model: $\chi^2 = 4.383$, df = 6, p = 0.625; Nagelkerke$R^2 = 0.069$.

Discussion

This study demonstrates a high prevalence of hyperprolactinemia in a PCOS population, consistent with previous reports indicating that approximately 13–37% of women with PCOS exhibit elevated prolactin levels (Davoudi et al., 2021; Kamrul-Hasan & Aalpona, 2024). The finding that all participants in the present study were hyperprolactinemic likely reflects referral bias toward hormonally complex cases; nevertheless, it emphasizes the importance of routinely evaluating prolactin levels in women presenting with suspected or confirmed PCOS.

The most notable finding was the strong inverse association between prolactin and FSH concentrations ($r = -.969$), which supports the biological mechanism whereby elevated prolactin suppresses hypothalamic gonadotropin-releasing hormone (GnRH) pulsatility, leading to reduced pituitary FSH secretion and impaired follicular maturation (Melmed et al., 2011; Khan et al., 2024). Consequently, hyperprolactinemia may exacerbate anovulation and reproductive dysfunction already associated with the altered gonadotropin dynamics characteristic of PCOS. Recent evidence further suggests that elevated prolactin levels are independently associated with insulin resistance, impaired glucose tolerance, and prediabetes in women with PCOS, supporting the strong positive correlation observed between prolactin and BMI in the present study (Yanachkova & Stankova, 2025).

The strong negative relationship between BMI and FSH ($r = -.971$) suggests that adiposity may play an active endocrine role in modulating reproductive hormones rather than acting solely as a metabolic comorbidity. Excess adipose tissue contributes to hormonal dysregulation through inflammatory pathways, insulin resistance, and altered adipokine secretion. Experimental and clinical evidence indicates that prolactin excess may promote adipocyte lipogenesis and impair pancreatic β-cell insulin sensitivity, thereby creating a self-perpetuating cycle of weight gain, metabolic dysfunction, and gonadotropin suppression (Pirchio et al., 2022). The elevated LH/FSH ratio (mean = 2.82) and testosterone levels (mean = 87.36 ng/dL) observed in the current study are characteristic endocrine features of PCOS and further support the role of

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gonadotropin dysregulation in the pathophysiology of the disorder. Although the LH/FSH ratio is no longer considered a diagnostic criterion, it remains a valuable marker for phenotypic characterization and assessment of reproductive dysfunction in PCOS (Teede et al., 2023).

5. Conclusion

This cross-sectional study confirms that hyperprolactinemia in PCOS is accompanied by significant hormonal axis disruption, characterized by prolactin-FSH inverse dynamics and a strong BMI–prolactin relationship. These findings support the routine measurement of serum prolactin in all PCOS patients, particularly those with oligomenorrhea, anovulatory infertility, or obesity-related metabolic dysfunction. The LH/FSH ratio retains clinical utility for PCOS phenotyping even in the setting of hyperprolactinemia. Longitudinal studies with normoprolactinemic controls are needed to establish causality and assess whether prolactin-targeted therapies (dopamine agonists, metformin) can meaningfully improve reproductive and metabolic outcomes in PCOS.

Limitation

- The study employed a cross-sectional design, limiting the ability to establish causal relationships between hyperprolactinemia, hormonal alterations, and PCOS.
- All participants were hyperprolactinemic, resulting in a homogeneous study population and restricting comparisons with normoprolactinemic women.
- The sample was recruited from a single tertiary-care hospital, which may introduce selection bias and limit the generalizability of the findings.

Recommendation

- Routine measurement of serum prolactin levels should be considered in women presenting with PCOS, infertility, menstrual irregularities, or obesity.
- Future studies should include normoprolactinemic PCOS patients and healthy control groups to better evaluate the independent role of prolactin in PCOS pathophysiology.
- Large-scale multicenter studies are recommended to improve the generalizability of findings across diverse populations.
- Longitudinal studies should be conducted to establish causal relationships between hyperprolactinemia and reproductive or metabolic dysfunction.

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