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To Evaluate the Correlation Between Luteinizing Hormone (LH), Follicle Stimulating Hormone (FSH), LH/FSH Ratio and BMI Among Women with Polycystic Ovarian Syndrome (PCOS)

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities. Obesity is considered a major aggravating factor influencing disease severity and hormonal imbalance. **Objective:** To evaluate the correlation between body mass index (BMI) and gonadotropin dynamics, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and the LH/FSH ratio in women with PCOS. **Methods:** This case–control observational study included 150 women with PCOS aged 20–40 years. Participants were stratified into two groups based on BMI: normal (<25 kg/m²) and high (≥25 kg/m²). Serum levels of LH, FSH, prolactin, testosterone, and thyroid-stimulating hormone (TSH) were analyzed, and correlation coefficients were calculated. **Results:** Women in the high-BMI group exhibited significantly higher BMI and LH/FSH ratios compared to the normal-BMI group (28.86 ± 3.23 vs. 21.41 ± 1.79; 2.48 ± 0.42 vs. 1.46 ± 0.27; $p < 0.0001$). Correlation analysis showed a strong negative association between the LH/FSH ratio and FSH, and a significant positive correlation with LH. No statistically significant associations were observed with prolactin, testosterone, or TSH. **Conclusion:** Obesity serves as a key modifier of hormonal dysregulation in PCOS, aggravating gonadotropin imbalance and contributing to more severe reproductive and metabolic outcomes. These findings highlight the importance of BMI-based stratification for clinical management, underscoring lifestyle modification and weight reduction in obese PCOS patients, while emphasizing tailored pharmacological approaches for lean counterparts.

Keywords: Polycystic Ovary Syndrome, BMI, LH/FSH ratio, Obesity, Gonadotropins.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with a prevalence of 18.5 % depending on the diagnostic criteria used and the population studied [1]. It is characterized by a combination of hyperandrogenism (clinical or biochemical), ovulatory dysfunction, and polycystic ovarian morphology [2].

The syndrome is associated with a wide range of symptoms, including irregular menstrual cycles, hirsutism, acne, and infertility, as well as long-term metabolic consequences such as insulin resistance, type 2 diabetes, and cardiovascular disease[3].

The pathophysiology of PCOS is complex and multifactorial, involving genetic, environmental, and lifestyle factors. Insulin resistance and hyperinsulinemia play a central role in the development of PCOS, contributing to hyperandrogenism by stimulating ovarian androgen production and reducing sex hormone-binding globulin (SHBG) levels[4]. Additionally,

elevated levels of luteinizing hormone (LH) relative to follicle-stimulating hormone (FSH) further exacerbate ovarian androgen secretion[1]. Genetic studies have identified several susceptibility loci associated with PCOS, highlighting the role of familial predisposition in its etiology[5].

Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are key gonadotropins that regulate ovarian function, and their dysregulation plays a central role in the pathophysiology of Polycystic Ovary Syndrome (PCOS). In women with PCOS, elevated LH levels and an increased LH/FSH ratio are commonly observed, contributing to the characteristic hormonal and ovulatory disturbances seen in this condition[1]

The LH/FSH ratio is often used as a diagnostic marker in PCOS, with a ratio greater than 2:1 considered indicative of the condition[1]. This elevated ratio reflects the disproportionate secretion of LH relative to FSH, which exacerbates hyperandrogenism and contributes to anovulation. However, it is important to note that the LH/FSH ratio is not universally elevated in all women with PCOS, and its diagnostic utility may vary depending on the population and the assay methods used[6].

Thyroid-stimulating hormone (TSH) and thyroid dysfunction have been increasingly recognized as potential contributors to the pathophysiology of Polycystic Ovary Syndrome (PCOS). While PCOS is primarily characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, emerging evidence suggests that thyroid abnormalities, particularly subclinical hypothyroidism (elevated TSH with normal thyroid hormone levels), are more prevalent in women with PCOS compared to the general population[7].

Prolactin plays a secondary but notable role in polycystic ovary syndrome (PCOS). While PCOS is primarily characterized by hyperandrogenism, ovulatory dysfunction, and insulin resistance, some women with PCOS exhibit mild hyperprolactinemia. Elevated prolactin levels can disrupt gonadotropin-releasing hormone (GnRH) pulsatility, leading to menstrual irregularities and anovulation. Additionally, prolactin may contribute to androgen excess by stimulating adrenal androgen production, potentially worsening hirsutism and acne. However, significant hyperprolactinemia (>50 ng/mL) is uncommon in PCOS and warrants evaluation for other causes, such as pituitary adenomas[8].

Testosterone plays a central role in polycystic ovary syndrome (PCOS), contributing to key symptoms such as hirsutism, acne, and menstrual irregularities. Elevated testosterone levels result from ovarian and, to a lesser extent, adrenal hyperandrogenism due to dysregulated luteinizing hormone (LH) secretion and insulin resistance. High testosterone disrupts follicular development, leading to anovulation and infertility. Additionally, excess androgens contribute to metabolic disturbances, including insulin resistance and an increased risk of type 2 diabetes[1].

MATERIALS AND METHODS

Study Design and Setting

This case–control observational study was conducted from July 2023 to March 2025 in the Department of Obstetrics and Gynecology, Rajkiya Mahila Chikitsalaya, affiliated with J.L.N. Medical College, Ajmer, Rajasthan, India. Biochemical investigations were carried out in the Clinical Biochemistry Laboratory of the same institute.

Study Population and Sample Size

A total of 150 women with polycystic ovary syndrome (PCOS), aged 20–40 years, were recruited from the outpatient department. Diagnosis was established according to the Rotterdam criteria (2003). Based on a reported prevalence of 10% and a 5% allowable error, the sample size was calculated as 144 and rounded to 150.
Participants were stratified into two groups according to body mass index (BMI):

- Group I (n = 75): Normal BMI (<25 kg/m²)
- Group II (n = 75): High BMI (≥25 kg/m²)

Eligibility Criteria

Inclusion: women aged 20–40 years, fulfilling Rotterdam criteria, with ultrasonographic evidence of polycystic ovaries, and clinical/biochemical hyperandrogenism.

Exclusion: women with Cushing's syndrome, hypothyroidism, adrenal hyperplasia, ovarian tumours, hyperprolactinemia, smokers, alcohol users, or those unwilling to provide informed consent.

Data Collection and Measurements

After obtaining written informed consent, demographic and clinical details were recorded. Anthropometric measurements were taken, and BMI was calculated as:

$$BMI = \frac{\text{Weight in kilograms (kg)}}{(\text{Height in meters})^2}$$

Venous blood samples were collected under aseptic precautions, centrifuged, and serum was stored at –20 °C until analysis.

Biochemical Assays

Serum concentrations of luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin (PRL), thyroid stimulating hormone (TSH), and testosterone were measured using Chemiluminescence Immunoassay (CLIA) on the Maglumi Biochemistry Analyzer (Snibe Co. LTD, China). Assays were performed as per manufacturer's instructions, with appropriate calibration and internal quality control.

Statistical Analysis

Data were analysed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. The Chi-square test was applied for categorical variables. Correlations between biochemical parameters were assessed using Pearson's or Spearman's correlation coefficients. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee of J.L.N. Medical College, Ajmer. Written informed consent was obtained from all participants prior to enrollment.

RESULTS

Table 1: Distribution of study participants according to Age

Age	Mean	S.D.	p value	Significance
Case (High BMI with PCOS)	27.01	3.91	p < 0.0001	HS (Highly significant)
Control (Normal BMI with PCOS)	24.34	2.85		

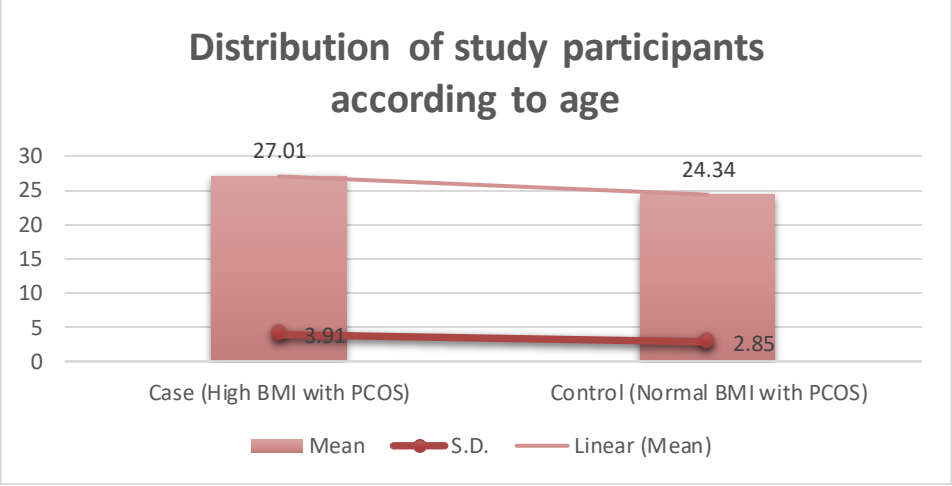


Figure 1

Table 2: Distribution of study participants according to BMI (Kg/m^2).

BMI(Kg/m^2)	Mean	S.D.	p value	Significance
Case (High BMI with PCOS)	28.86	3.23	p < 0.0001	HS (Highly significant)
Control (Normal BMI with PCOS)	21.41	1.79		

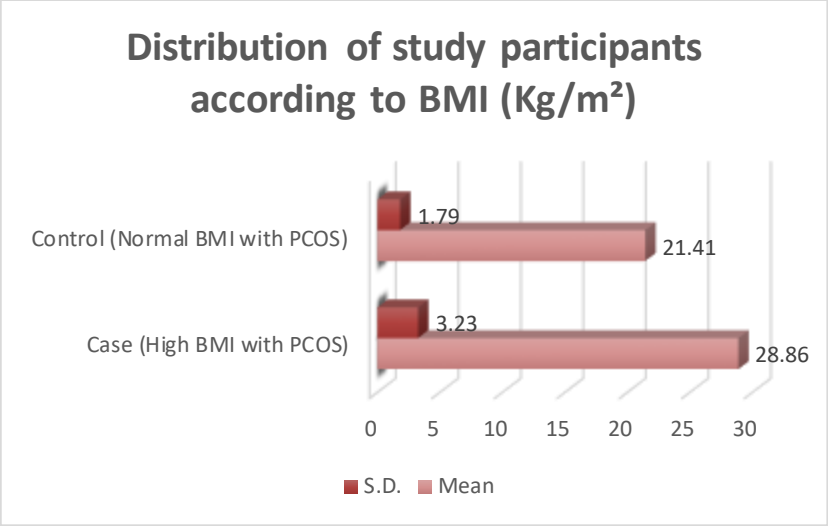


Figure 2

Table 3: Distribution of study participants according to LH/FSH ratio

LH/FSH ratio	Mean	S.D.	p value	Significance
Case (High BMI with PCOS)	2.48	0.42	p < 0.0001	HS (Highly significant)
Control (Normal BMI with PCOS)	1.46	0.27		

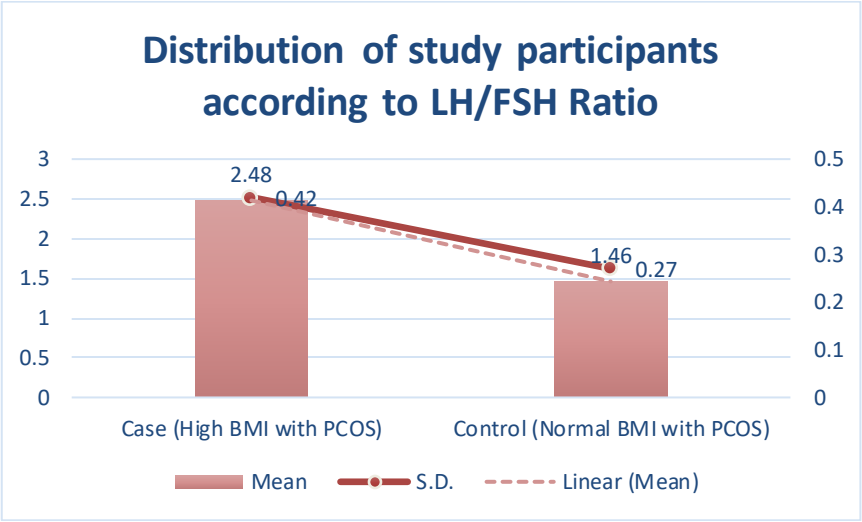


Figure 3

Table 4: Comparison of LH/FSH Ratio with other variables of PCOS in cases and controls

Parameters	Case (High BMI with PCOS)		Control (Normal BMI with PCOS)	
	p value	r (Pearson coefficient)	p value	r (Pearson coefficient)
BMI (Kg/m^2)	0.134	-0.174	0.806	0.028
PROLACTIN (ng/ml)	0.071	-0.209	0.478	-0.083
FSH (mIU/ml)	<0.0001	-0.748	<0.0001	-0.602
LH (mIU/ml)	0.005	0.318	<0.0001	0.616
TSH (µIU/ml)	0.688	0.047	0.099	0.191
TESTOSTERONE (ng/dL)	0.067	0.212	0.104	-0.189

Table 5: Distribution of study participants according to the hormonal parameters

Parameters	Group	Mean ± S.D.	P-value	Significance
Prolactin (ng/ml)	Case (High BMI with PCOS)	12.77 ± 1.74	P < 0.001	HS
	Control (Normal BMI with PCOS)	10.12 ± 1.63		
FSH (mIU/mL)	Case (High BMI with PCOS)	6.60 ± 1.07	P = 0.0188	Significant
	Control (Normal BMI with PCOS)	7.00 ± 0.99		
LH (mIU/mL)	Case (High BMI with PCOS)	16.40 ± 1.93	P < 0.0001	HS
	Control (Normal BMI with PCOS)	10.26 ± 1.46		
TSH (µIU/mL)	Case (High BMI with PCOS)	3.03 ± 0.84	P = 0.5801	NS
	Control (Normal BMI with PCOS)	2.96 ± 0.70		
Testosterone (ng/dL)	Case (High BMI with PCOS)	72.04 ± 3.82	P < 0.001	HS
	Control (Normal BMI with PCOS)	62.48 ± 3.88		

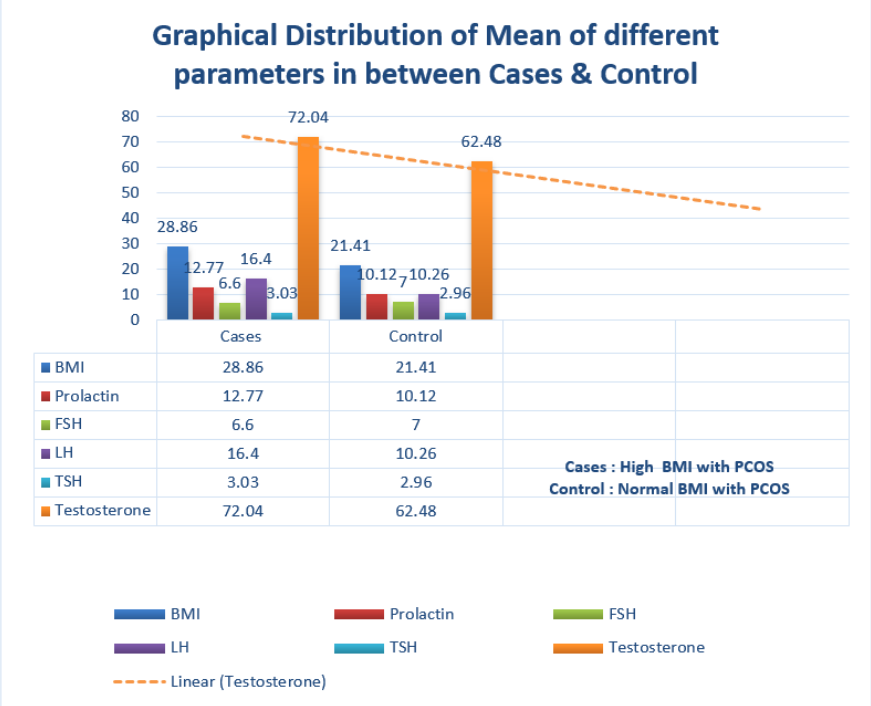
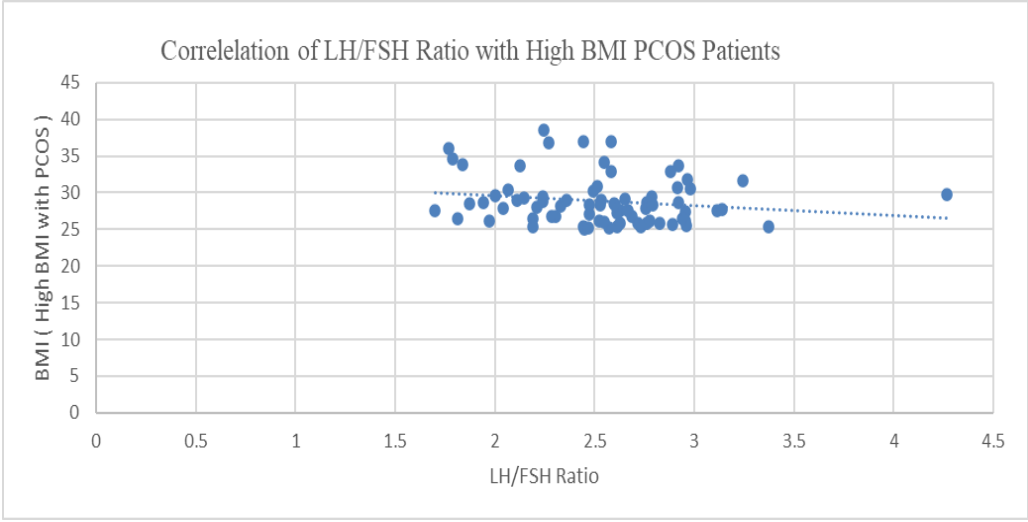


Figure 4



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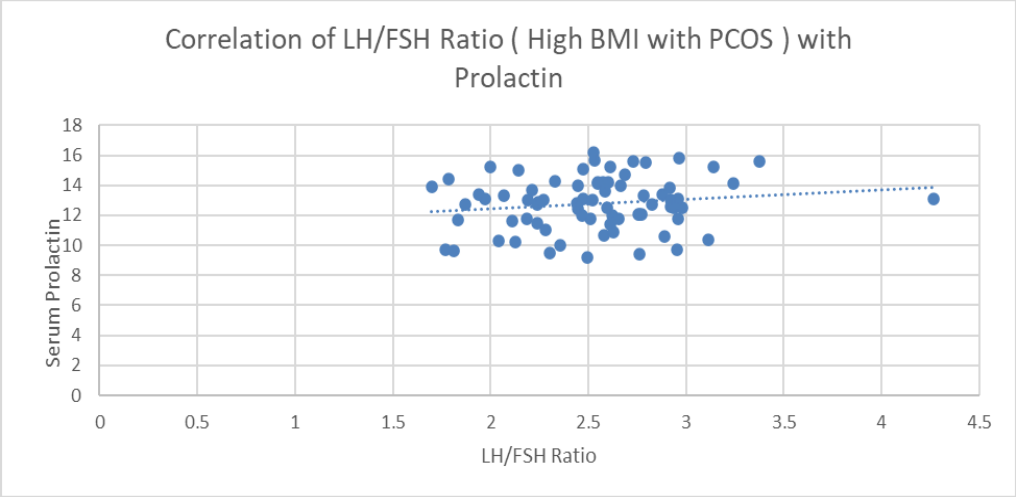

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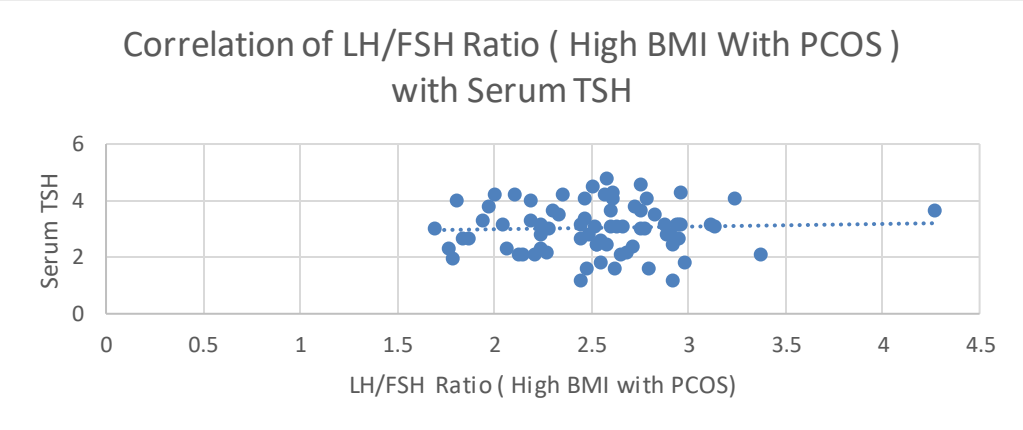

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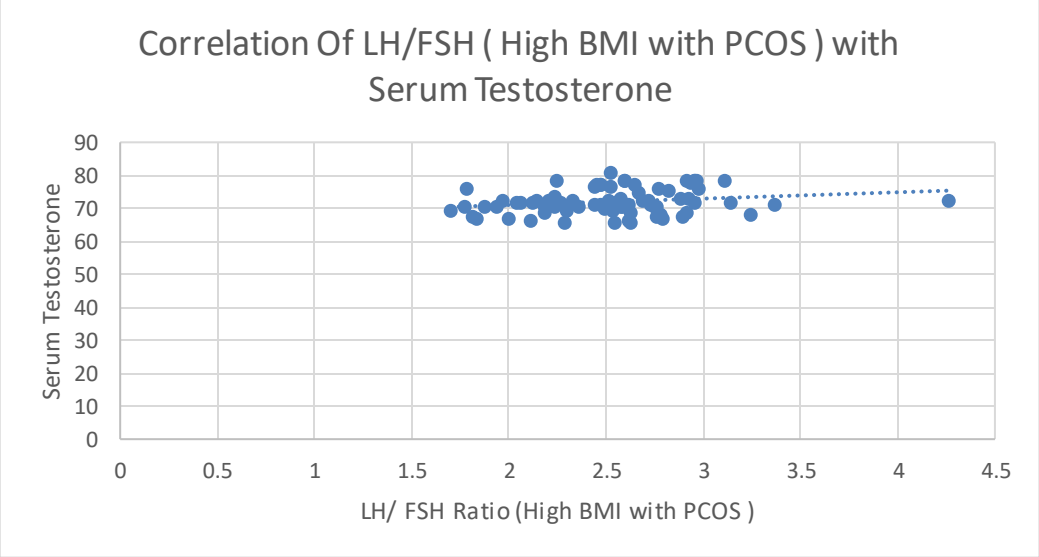

Figure 8

DISCUSSION

The present case–control study investigated the hormonal and biochemical alterations in women with Polycystic Ovary Syndrome (PCOS) with varying body mass indices (BMI). The findings revealed significant differences between normal

BMI and high BMI PCOS groups, underscoring the role of obesity as a critical modifier of the endocrine and metabolic profile in PCOS.

A significant age difference was observed between the two groups, with high BMI PCOS women being relatively older than their normal BMI counterparts. This finding suggests that advancing age may be associated with higher BMI and more severe manifestations of PCOS, consistent with the progressive nature of metabolic disturbances in this condition. The distribution according to age was in accordance with M Sharma et al (2022)[9] who also collected data of BMI, serum TSH, LH, FSH, FBS, of PCOS patients, from which they found the mean age of studied population was 24.76 ± 5.48 years. Mazin H. Daghestani et al (2021)[10] in a similar study found the mean age of studied population to be 25.76±3.24 years. (Table 1 and Figure 1)

The mean BMI in the high BMI PCOS group was markedly elevated compared to controls (28.86 ± 3.23 vs. 21.41 ± 1.79, $p < 0.0001$), reinforcing the role of obesity as a strong determinant of PCOS severity. Obesity is known to aggravate insulin resistance and hyperinsulinemia, which in turn potentiate hyperandrogenism and anovulatory cycles, thereby amplifying the clinical and metabolic burden of PCOS. A p-value < 0.0001 indicates that the difference in BMI between the two groups is highly statistically significant (HS). This suggests a strong association between higher BMI and a specific subset of PCOS patients. Wenjing Shi et al (2021)[11] in a similar study found the mean BMI of obese PCOS group to be 30.55±4.03 and of normal BMI PCOS group to be 21.99±3.59. Haolin Zhang et al (2023)[12] also in his original study found the mean BMI of case to be 27.73 ± 5.17 and of control to be 24.72 ± 4.68. (Table 2 and figure 2)

A striking finding of the study was the significantly elevated LH/FSH ratio in the high BMI group compared to the normal BMI group (2.48 ± 0.42 vs. 1.46 ± 0.27, $p < 0.0001$). This aligns with the classical endocrine hallmark of PCOS, where hypersecretion of luteinizing hormone drives ovarian androgen excess, while inadequate follicle-stimulating hormone contributes to follicular arrest and anovulation. The more pronounced elevation in the obese cohort may reflect synergistic effects of hyperinsulinemia and adiposity on gonadotropin dysregulation. The p value of present study is < 0.0001 which is statistically highly significant. In a study conducted by M Sharma et al (2022)[9] the mean LH/FSH ratio of obese PCOS group was 3.42 ±2.88 and of Non-obese PCOS was 2.70 ±1.60. This study data is in accordance with present study. Wenjing Shi et al (2021)[11] conducted a study in which mean LH/FSH ratio of obese PCOS group was 2.15±0.90 which is in accordance with our present study. In study done by Mazin H. Daghestani et al (2021)[10] the mean LH/FSH ratio of case group was 2.74 ± 0.84 and of control group was 1.01 ± 0.33 which is in accordance with present study. (Table 3 and Figure 3)

Correlation analysis provided deeper insights into the complex hormonal interplay in PCOS. In both groups, LH/FSH ratio showed a strong negative correlation with FSH (r = –0.748 in high BMI, r = –0.602 in normal BMI, $p < 0.0001$ for both), highlighting the disproportionate suppression of FSH relative to LH secretion. Conversely, LH demonstrated a significant positive correlation with LH/FSH ratio in both groups (r = 0.318 in high BMI, r = 0.616 in normal BMI), confirming the dominance of LH hypersecretion in defining the altered ratio. Interestingly, the relationship of LH/FSH ratio with prolactin, testosterone, and TSH did not reach statistical significance, suggesting that while these hormones may influence PCOS pathophysiology, their direct contribution to gonadotropin imbalance is less consistent. The observation that testosterone did not show a significant correlation with LH/FSH ratio in either group may be explained by the multifactorial origins of hyperandrogenism in PCOS. While elevated LH stimulates ovarian theca cells to produce androgens, adrenal contributions, insulin-mediated effects, and reduced SHBG levels also play key roles, particularly in obese women. Similarly, the lack of a strong association between prolactin and LH/FSH ratio supports the notion that mild hyperprolactinemia seen in some PCOS patients is a secondary rather than primary driver of reproductive dysfunction. (Table 4)

Parameter	Case (High BMI)	Control (Normal BMI)	Interpretation
BMI	$P = 0.134$, r = -0.174	$P = 0.806$, r = 0.028	No significant correlation with other variables in either group.
Prolactin	$P = 0.071$, r = -0.209	$P = 0.478$, r = -0.083	Trend toward negative correlation in high BMI group, but not significant.
FSH	$P < 0.0001$, r = -0.748	$P < 0.0001$, r = -0.602	Strong **negative correlation** with FSH in both groups; stronger in high BMI. Highly significant.
LH	$P = 0.005$, r = 0.318	$P < 0.0001$, r = 0.616	Significant **positive correlation**, stronger in normal BMI.
TSH	$P = 0.688$, r = 0.047	$P = 0.099$, r = 0.191	No significant correlation in either group.

Parameter	Case (High BMI)	Control (Normal BMI)	Interpretation
Testosterone	P = 0.067, r = 0.212	P = 0.104, r = -0.189	Near-significant trends, but no strong evidence of correlation.

(Figure 5,6,7 and 8)

The comparative analysis of hormonal profiles between PCOS women with high BMI and those with normal BMI revealed significant variations. Prolactin levels were markedly higher in the high-BMI group (12.77 ± 1.74 ng/ml) compared to the normal-BMI group (10.12 ± 1.63 ng/ml), with a highly significant difference ($P < 0.001$). Similarly, serum LH levels were elevated in obese PCOS women (16.40 ± 1.93 mIU/mL) relative to their counterparts (10.26 ± 1.46 mIU/mL), again demonstrating high statistical significance ($P < 0.0001$). Testosterone levels also showed a highly significant rise in the high-BMI group (72.04 ± 3.82 ng/dL vs. 62.48 ± 3.88 ng/dL; $P < 0.001$). Conversely, FSH levels were significantly reduced in obese PCOS women (6.60 ± 1.07 mIU/mL) compared to those with normal BMI (7.00 ± 0.99 mIU/mL; $P = 0.0188$). However, TSH levels did not differ significantly between the groups (3.03 ± 0.84 µIU/mL vs. 2.96 ± 0.70 µIU/mL; $P = 0.5801$). (Table 5)

Taken together, these findings reinforce the heterogeneity of PCOS and emphasize the compounding role of obesity in its clinical and biochemical presentation. High BMI not only exacerbates insulin resistance but also appears to magnify the disruption of gonadotropin dynamics, reflected in a significantly higher LH/FSH ratio. This altered hormonal milieu perpetuates the vicious cycle of anovulation, hyperandrogenism, and metabolic derangements characteristic of PCOS.

From a clinical standpoint, the results underscore the importance of stratifying PCOS patients by BMI when assessing hormonal parameters and tailoring management strategies. Lifestyle modification and weight reduction should be emphasized as first-line interventions, as they have the potential to restore hormonal balance, improve ovulatory function, and reduce long-term risks such as type 2 diabetes and cardiovascular disease.

BMI emerged as a significant modifier of hormonal profiles in PCOS. Obese women showed more severe gonadotropin imbalance compared to their normal BMI counterparts, supporting the view that adiposity exacerbates PCOS manifestations. This is explained by the interplay between adiposity, insulin resistance, and androgen excess. Conversely, normal BMI PCOS women, despite the absence of obesity, still displayed significant gonadotropin abnormalities, reinforcing the notion that PCOS is not solely a disorder of adiposity but also of intrinsic neuroendocrine dysfunction.

The study highlights the importance of stratifying PCOS patients by BMI when planning management strategies. For obese PCOS women, lifestyle interventions such as weight reduction and physical activity should remain the cornerstone of therapy, as these measures are proven to improve insulin sensitivity, reduce androgen levels, and restore ovulatory function. Lean PCOS women, however, may derive greater benefit from targeted pharmacological interventions aimed at correcting gonadotropin imbalance and improving insulin sensitivity, rather than weight reduction alone. This distinction is clinically significant, as a “one-size-fits-all” approach may not adequately address the heterogeneity of PCOS.

Limitations and Future Directions

The study is not without limitations. The single-institution design and moderate sample size may limit the generalizability of the findings. Additionally, the focus on hormonal parameters and BMI, without inclusion of metabolic markers such as fasting insulin, lipid profile, or inflammatory biomarkers, restricts a comprehensive understanding of the metabolic derangements associated with obesity in PCOS. Finally, the cross-sectional design precludes determination of causal relationships between obesity, hormonal imbalance, and reproductive dysfunction.

Future research should include multicentric, longitudinal studies with larger cohorts, integrating hormonal, metabolic, and inflammatory profiles. Interventional trials exploring the impact of weight reduction and insulin-sensitizing therapies on hormonal dynamics across BMI subgroups would further clarify the causal pathways and help design individualized therapeutic strategies for women with PCOS.

CONCLUSION

The present study highlights that obesity profoundly modifies the hormonal profile of women with PCOS, with high BMI patients exhibiting significantly elevated LH/FSH ratios and more severe gonadotropin imbalance compared to their normal BMI counterparts. These findings confirm that while intrinsic neuroendocrine dysfunction is central to PCOS, adiposity acts as a critical aggravating factor, intensifying hyperandrogenism, anovulation, and associated metabolic risks. From a clinical perspective, stratifying PCOS patients by BMI is crucial for effective management, as obese women benefit most from lifestyle modification and weight reduction, whereas lean women may require more targeted pharmacological

approaches. Overall, the study reinforces the heterogeneity of PCOS and advocates for individualized, BMI-specific therapeutic strategies aimed at optimizing reproductive outcomes and minimizing long-term metabolic complications.

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