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CORRELATION BETWEEN BODY MASS INDEX AND FSH AND LH RATIO IN POLYCYSTIC OVARIAN SYNDROME WOMEN

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ABSTRACT

Objective: To determine the correlation between body mass index and FSH and LH ratio in polycystic ovarian syndrome women.
Study Design: Cross sectional study.
Duration and Place of Study: Conducted from 15 November 2025 to 15 February 2026 at Department of Obstetrics and Gynaecology, PAF Hospital Sargodha.
Methodology: A total of 80 women of age 18 to 40 years with polycystic ovarian syndrome were included. Body mass index was determined using the standard formula of body weight in kilograms divided by the square of height in meters. Venous blood specimens were collected to quantify serum follicle-stimulating hormone and luteinizing hormone concentrations after which the LH/FSH ratio was computed. Statistical evaluation of the collected data was performed using the SPSS version 26. Pearson correlation test was applied to assess association between body mass index and hormone ratio.
Results: Mean age was 27.22 ± 5.75 years and mean body mass index was 27.30 ± 2.63 kg/m². Mean hormone ratio was 2.67 ± 0.28. Married women were 83.8%. A statistically significant positive association was observed between body mass index and the hormone ratio ($r = 0.311$, $p = 0.005$). Strongest correlation was seen in women with disease duration ≤ 24 months ($r = 0.400$, $p = 0.005$).
Conclusion: Body mass index was positively associated with follicle stimulating hormone to luteinizing hormone ratio in polycystic ovarian syndrome women.

KEYWORDS: Body Mass Index; Gonadotropins; Luteinizing Hormone; Polycystic Ovary Syndrome; Reproductive Hormones

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a hormonal disorder seen in women of childbearing age, characterized by ovulatory dysfunction, excess androgen levels and polycystic ovarian morphology on ultrasonography.[1] It is a major cause of menstrual irregularities and infertility. Patients present with clinical findings of oligomenorrhea/amenorrhea, hirsutism, acne, and weight gain.[2] Though the exact pathogenesis is unknown, insulin resistance and hormonal imbalance are key components of the syndrome.[2] Hypersecretion of luteinizing hormone and ovarian androgen production affect follicular maturation, resulting in multiple small follicles within the ovary and anovulation.[3] Long-term complications include type 2 diabetes mellitus, dyslipidemia, and cardiovascular diseases.[4]
Body mass index (BMI) is an anthropometric indicator calculated by dividing body weight in kilograms by height in meters squared.[5] Elevated BMI is frequently observed among women diagnosed with PCOS and is associated with aggravation of clinical manifestations. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are gonadotropic hormones secreted by the anterior pituitary gland.[5] Under normal circumstances, the ratio of LH to FSH should be 1:1. However, this ratio is often elevated to 2:1 or 3:1 in women suffering from PCOS.[6] High levels of LH stimulate the theca cells to produce excess androgens.
The complex association between body mass index and the LH/FSH ratio among women suffering from polycystic ovarian syndrome cannot be taken at face value.[7] From the empirical perspective, the study suggests that a high BMI, which indicates greater metabolic disorders, also shows a concomitant reduction in the levels of LH.[8] For example, the study shows that the LH to FSH ratio might be higher among lean women suffering from PCOS, whereas obese

women might experience insulin resistance and hyperandrogenism.[9] It is a fact that the expanded adipose tissue might affect the hypothalamic-pituitary-ovarian axis through the insulin-leptin hypothesis.[10]
This study was deemed justifiable and worthwhile, considering the high prevalence of polycystic ovarian syndrome among women of reproductive age which is also accompanied by hormonal imbalance and obesity. Though the altered ratio of luteinizing hormone to follicle-stimulating hormone has been clearly demonstrated in PCOS patients the impact of body mass index on the hormonal status remains obscure, with varying results from different studies. In the present population the correlation between BMI and the LH/FSH ratio in PCOS patients has not been clearly demonstrated.

METHODOLOGY

A cross-sectional investigation was conducted at the Department of Obstetrics and Gynaecology, PAF Hospital Sargodha, spanning from 15th November 2025 to 15th February 2026. Prior to initiating data acquisition, formal clearance was obtained from the hospital's institutional ethics committee (MSF(H)/308/3/1/Trg)**.** Sample size was calculated by using Spearman's correlation coefficient (r) = 0.309,[11] with significance level (α) = 0.05 (two tailed) and power = 0.80. By applying Fisher's transformation formula $n = ((Z\alpha + Z\beta)/C)^2 + 3$, where $C = 0.5 \times \ln((1+r)/(1-r))$, the calculated sample size was 80 patients. Women aged 18 to 40 years, both married and unmarried, diagnosed cases of polycystic ovarian syndrome were included. Polycystic ovarian syndrome was considered present if any 2 of following findings were noted: oligoovulation defined as menstrual cycles ≥36 days or <8 cycles per year or anovulation presenting as irregular menstrual periods; biochemical hyperandrogenism defined as free Testosterone level 0.06 to 2.57 pg per ml (0.20 to 8.90 pmol per L) on laboratory testing; and polycystic ovaries on transvaginal ultrasound showing ≥12 follicles in each ovary measuring 2 to 9 mm in diameter with ovarian volume >10 mL. Patients having history of Cushing disease, hypothyroidism, hyperprolactinemia, adrenal hyperplasia, or ovarian tumors were excluded from study. Written informed consent was obtained from all patients before enrollment and before any investigation was performed. Demographic variables including age, marital status, weight, height and duration of PCOS were recorded. Detailed clinical history regarding menstrual pattern and duration of PCOS was taken. General physical examination was done including measurement of weight in kilogram and height in meters using standard calibrated instruments.
After the clinical examination, a 5 mL sample of venous blood was collected in vacuum tubes following aseptic techniques. The sample was transported to the laboratory for analysis within 30 to 60 minutes. Serum FSH and LH levels were analyzed, and the values were expressed in international units per liter (IU/L). The FSH to LH ratio was calculated by dividing the FSH level by the LH level. FSH was defined as the FSH level, and LH was defined as the LH level. Similarly, the FSH to LH ratio was calculated as the FSH level divided by the LH level.
All data were coded and processed using IBM SPSS Statistics version 26. Qualitative variable including marital and residential status were summarized as frequencies and percentage. Continuous variables such as age, body weight, height, duration of PCOS, BMI and LH/FSH ratio were reported as mean ± standard deviation. The Pearson correlation coefficient was employed to determine the relationship between BMI and LH/FSH ratio. Statistical significance was defined at a p-value ≤0.05. Potential confounders, including age, marital status and duration of PCOS were addressed through stratification. Following stratified analysis the Pearson correlation test was reapplied with p ≤0.05 considered statistically significant.

RESULTS

A total of 80 PCOS women was enrolled and their demographic and hormonal parameters was assessed. The mean age of the study participants were 27.22 ± 5.75 years, and the mean duration of PCOS was recorded as 23.73 ± 10.49 months. The mean weight and height of the women was found to be 68.19 ± 8.05 kg and 1.57 ± 0.05 m respectively, which corresponded to a mean BMI of 27.30 ± 2.63 kg/m², suggesting that the sample was falling in the overweight category on average. The mean FSH/LH ratio was observed to be 2.67 ± 0.28 IU/L across all participants. Regarding marital status, large majority of the women was married, comprising 67 participants that is 83.8% of total, while 13 women was unmarried representing 16.3% of the sample (Table-I).

Table- I: Patient Demographics of study

Demographics	**Mean ± SD**
Age (years)	27.22 ± 5.75
Duration of PCOS (months)	23.73 ± 10.49
Weight (Kg)	68.19 ± 8.05
Height (m)	1.57 ± 0.05
BMI (Kg/m²)	27.30 ± 2.63
FSH/LH Ratio (IU/L)	2.67 ± 0.28

Marital Status	
Married n (%)	67 (83.8%)
Unmarried n (%)	13 (16.3%)

The mean BMI and FSH/LH ratio of the participants was 27.30 ± 2.63 kg/m² and 2.67 ± 0.28 IU/L respectively. A positive and statistically significant correlation was found to be exist between BMI and FSH/LH ratio in PCOS women, with Pearson correlation coefficient of 0.311 and p value of 0.005, which indicating that as BMI was increasing, the FSH/LH ratio was also tend to increase. This correlation was significant at 0.01 level on two-tailed testing, which suggesting a meaningful hormonal disturbance that was associated with higher body weight in these women (Table-II).

Table- II: Correlation Between BMI and FSH/LH Ratio

Variables	**Mean ± SD**	**Pearson Correlation**	**P value**
BMI (Kg/m²)	27.30 ± 2.63	0.311**	0.005
FSH/LH Ratio (IU/L)	2.67 ± 0.28		

**. Correlation is significant at the 0.01 level (2-tailed).

Among women who was aged 30 years or below, BMI was 26.86 ± 2.40 kg/m² and FSH/LH ratio was 2.66 ± 0.27 IU/L, with a statistically significant Pearson correlation of 0.314 at p = 0.016, which suggesting that the BMI and FSH/LH ratio relationship was holding true even in younger PCOS women. In the married subgroup of women, who was representing the majority of the sample, BMI was 27.44 ± 2.71 kg/m² and FSH/LH ratio was 2.69 ± 0.28 IU/L, and the correlation was found to be significant with Pearson r of 0.342 and p = 0.005. Among those women whose PCOS duration was 24 months or lesser, mean BMI was 27.07 ± 2.42 kg/m² and FSH/LH ratio was 2.63 ± 0.29 IU/L, and notably the strongest correlation across all subgroups was observed in this category with Pearson r of 0.400 and p = 0.005, which may be suggesting that the BMI and FSH/LH ratio association was more pronounced in earlier stages of disease (Table-III).

Table- III: Subgroup Analysis for Correlation Between BMI and FSH/LH Ratio

Subgroup	**Variables**	**Mean ± SD**	**Pearson Correlation**	**P value**
Age ≤30 years	BMI (Kg/m²)	26.86 ± 2.40	0.314*	0.016
	FSH/LH Ratio (IU/L)	2.66 ± 0.27		
Age >30 years	BMI (Kg/m²)	28.52 ± 2.94	0.255	0.264
	FSH/LH Ratio (IU/L)	2.72 ± 0.31		
Married	BMI (Kg/m²)	27.44 ± 2.71	0.342**	0.005
	FSH/LH Ratio (IU/L)	2.69 ± 0.28		
Unmarried	BMI (Kg/m²)	26.56 ± 2.14	-0.025	0.934
	FSH/LH Ratio (IU/L)	2.58 ± 0.26		
PCOS Duration ≤24 months	BMI (Kg/m²)	27.07 ± 2.42	0.400**	0.005
	FSH/LH Ratio (IU/L)	2.63 ± 0.29		
PCOS Duration >24 months	BMI (Kg/m²)	27.63 ± 2.92	0.171	0.342
	FSH/LH Ratio (IU/L)	2.73 ± 0.26		

*p < 0.05, **p < 0.01

DISCUSSION

In this study mean age of women with PCOS was found to be 27.22 ± 5.75 years and the mean BMI was found to be 27.30 ± 2.63 kg/m², indicating that the subjects of the study mostly belonged to the overweight category. It has been established that overweight people have more adipose tissue, which increases the peripheral aromatization of androgens, resulting in the suppression of FSH through negative feedback mechanisms at the pituitary level, and simultaneously stimulating the release of LH, leading to the disruption of the FSH/LH balance. It was found that BMI

was significantly correlated with the FSH/LH ratio, where r = 0.311, p = 0.005, indicating a positive correlation between BMI and FSH/LH ratio, suggesting that women with a higher BMI have a higher FSH/LH ratio. In PCOS, overweight women have more adiposity, leading to insulin resistance, which increases the release of LH by the pituitary, but the higher FSH/LH ratio observed in the study subjects could be a compensatory mechanism of the pituitary, where FSH release is also altered.

The present study was found a statistically significant positive correlation between BMI and FSH/LH ratio in PCOS women (r = 0.311, p = 0.005), which was supporting the notion that increased adiposity was having a measurable influence on gonadotropin dynamics in this population. This finding was in agreement with Kumar *et al.* [12] who also reported a significantly higher LH/FSH ratio in high-BMI PCOS women (2.48 ± 0.42) compared to normal BMI group (1.46 ± 0.27, p<0.0001), and with Memon *et al.* [13] who was observed a positive correlation between increased BMI, elevated LH/FSH ratio and serum testosterone in PCOS women. Similarly, Khmil *et al.* [14] was reported a direct correlation between BMI and LH/FSH ratio, with LH/FSH ratio being 30.35% higher in Class 2 obesity compared to normal BMI group, which was further consolidating that adiposity was directly disturbing the gonadotropin axis in PCOS. In contrast, Saadia *et al.* [15] was reported a non-significant difference in LH/FSH ratio between normal BMI (2.76) and high BMI (2.79) PCOS women (p = 0.48), and Khayyat *et al.* [16] was also found no significant relationship between LH/FSH ratio and BMI (p = 0.65). Alnakash *et al.* [17] similarly reported no statistically significant correlation between LH/FSH ratio and BMI despite 63.55% of their sample being overweight or obese. These contradictory findings may be explained by differences in sample size, study design and the statistical methods was applied, as both Saadia *et al.* [15] and Khayyat *et al.* [16] was used Spearman correlation rather than Pearson method, and sample sizes was relatively smaller which may have reduced statistical power to detect a significant association. The mean BMI of 27.30 ± 2.63 kg/m² in present study was comparable to the overweight group reported by Bhattacharya *et al.* [18] where mean BMI in overweight PCOS women was 27.44 kg/m², and among those overweight women, 46.93% was having elevated LH/FSH ratio, 59.18% hyperandrogenism and 44.2% hirsutism, which was indicating that the overweight BMI range was carrying significant hormonal and clinical burden. Akram *et al.* [19] was also reported significantly elevated LH, LH/FSH ratio and androstenedione in PCOS women compared to controls, which was further supporting that hormonal dysregulation was a consistent feature in this disease and was closely tied to increased body weight.

The subgroup analysis of present study was revealed that the correlation between BMI and FSH/LH ratio was significant only in women aged 30 years or below (r = 0.314, p = 0.016), in married women (r = 0.342, p = 0.005), and among those with PCOS duration of 24 months or lesser (r = 0.400, p = 0.005), while it was not significant in women above 30 years or with longer disease duration. This finding was partially supported by Bohlke *et al.* [20] who was observed that LH levels was decreasing with increasing BMI in premenopausal women, with women in highest BMI quintile (>27.1 kg/m²) having 40% lower LH than second quintile (p = 0.003), suggesting that the gonadotropin response to BMI may become altered or attenuated over time as the disease was progressing and the hypothalamic-pituitary axis was becoming less responsive to metabolic stimuli. Regarding hormonal profile, Akhter *et al.* [21] was reported mean LH/FSH ratio of 2.05 ± 0.18 in PCOS women versus 1.25 ± 0.18 in controls, with 22.5% of PCOS cases having elevated LH, which was consistent with the elevated FSH/LH ratio of 2.67 ± 0.28 IU/L was observed in present study, although direct comparison was limited due to differences in how the ratio was calculated and reported across studies. Sajjad *et al.* [22] was found LH/FSH ratio >2 in only 31% of their 86 PCOS women despite 71% being obese, and gonadotropins was showing no significant association with clinical features, which may be suggesting that obesity alone was not always sufficient to produce measurable gonadotropin abnormalities and that other metabolic and genetic factors was also playing a role in modulating this relationship.

This study has some limitations, and these limitations should be taken into account in understanding the results of this study. Firstly, this study was conducted in only one institution. This may limit the results of this study in relation to the larger population of women with polycystic ovary syndrome. Secondly, the population of this study was only composed of 80 participants. Therefore, a larger study in multiple centers is required to confirm these results. Thirdly, hormonal studies were done in a single time point, and no follow-up studies were done to assess the dynamic change in BMI and FSH/LH ratio.

CONCLUSION

The present study reveals that there is a positive correlation between body mass index (BMI) and the FSH/LH ratio in women with polycystic ovarian syndrome (PCOS), which indicates that BMI plays a significant role in the disturbance of the pattern of secretion of gonadotropins. Furthermore, this correlation is more marked in younger individuals and in women with shorter disease duration.

Disclaimer:

Nothing to declare.

Acknowledgement:
The author thank the department medical staffs for their help and support. They keep patient record properly and managed the data carefully which helped in completing this research.

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