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Association Between Hormonal Profile and Ovulatory Dysfunction in Infertile Women with Polycystic Ovary Syndrome (PCOS): A Cross-Sectional Study

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ABSTRACT

Background: Polycystic ovary syndrome is one of the most prevalent endocrine conditions among reproductive-aged females that features hormonal imbalance (high levels of LH, high LH/FSH ratio, hyperandrogenism) and insulin resistance causing ovulation dysfunction and infertility. The current paper aims at establishing the relationship between hormonal and metabolic factors and ovulatory dysfunction, determining the predictors of anovulation. **Materials & Methods:** This cross-sectional study was conducted among 110 infertile women aged 18–40 years diagnosed with PCOS based on the Rotterdam criteria at Department of Gynaecology & Obstetrics, Central Police Hospital, Dhaka, Bangladesh, from January 2024 to December 2024. Socio-demographic, anthropometric, and clinical data were recorded, and hyperandrogenism was assessed using the modified Ferriman-Gallwey (mFG) score. Fasting blood samples were analyzed for LH, FSH, testosterone, DHEA-S, SHBG, insulin, and glucose; HOMA-IR was calculated to assess insulin resistance. Data were analyzed using SPSS 26.0. **Results:** Among 110 infertile women with PCOS, most were young (mean age 28.6 years), urban residents, and overweight or obese (79%, mean BMI 29.8 kg/m²). Hirsutism (55.5%) was the most common hyperandrogenic feature. Elevated LH/FSH ratio (59.1%), high testosterone (46.4%), and insulin resistance (67.3%) were frequent. Ovulation was negatively correlated with LH/FSH ratio, testosterone, fasting insulin, and HOMA-IR, but positively with SHBG. Logistic regression analysis revealed that a higher LH/FSH ratio, testosterone, and HOMA-IR independently predicted anovulation, whereas a higher SHBG level was protective. **Conclusion:** The study concludes that hormonal imbalances, particularly elevated LH, LH/FSH ratio, and testosterone, are significant predictors of anovulation in infertile women with PCOS, while higher progesterone reflects preserved ovulation. Hormonal profiling is essential for early diagnosis and personalized management to enhance reproductive outcomes.

Keywords: *Hormonal Profile, Ovulatory Dysfunction, Infertile Women, and Polycystic Ovary Syndrome (PCOS)*

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is recognized as one of the most common endocrines-metabolic disorders among women of reproductive age, characterized by heterogeneous manifestations including hyperandrogenism, chronic anovulation or oligo-ovulation, and polycystic ovarian morphology. From a pathophysiological standpoint, the interlocking triad of androgen excess, gonadotrophin dysregulation and insulin resistance underpin ovulatory dysfunction in PCOS [1]. Women with PCOS frequently display elevated luteinizing hormone (LH) secretion relative to follicle-stimulating hormone (FSH), increased LH/FSH ratios, reduced sex hormone-binding globulin (SHBG) levels, hyperinsulinaemia and a resultant cascade of ovarian theca-cell stimulation and disrupted folliculogenesis [2]. Investigating the hormonal profile in women with PCOS, therefore, offers insight into the mechanisms of ovulatory failure and infertility. Globally, the prevalence of PCOS among reproductive-aged women is estimated at approximately 5–15%, depending on diagnostic criteria and population studied [3]. The syndrome is not only a reproductive disorder, but also confers increased risk of infertility, metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease [4]. Regionally, in Bangladesh, smaller studies report high prevalence of PCOS manifestations among infertile women: for example, one cross-sectional study found approximately 35 % of infertile women attending a tertiary centre had PCOS, with oligomenorrhea and central obesity the most typical presentations [5]. Another study among infertile women with PCOS reported a high prevalence of insulin resistance (65.9 %), dyslipidaemia (93.7 %) and metabolic syndrome (42.9 %) in Bangladesh [1]. These data indicate a considerable burden of PCOS and its complications in the Bangladeshi infertile population. Clinically, ovulatory dysfunction is a key pathway by which PCOS causes infertility. Regular ovulation is essential for conception, and disruption of follicular maturation leads to anovulation, long cycles, and subfertility [6]. Hormonal abnormalities such as elevated LH/FSH ratio, hyperandrogenemia, reduced SHBG and insulin resistance have been associated with poor ovulatory response in PCOS [7]. For instance, an extensive multicentre Chinese study demonstrated that an elevated baseline LH/FSH ratio was significantly associated with poor ovulatory response following induction, although paradoxically with higher clinical pregnancy and live birth rates [8]. Likewise, SHBG has been investigated as a marker of insulin resistance and androgen excess in PCOS, though its relationship with ovulation remains under-elucidated [9]. Despite this, much of the literature has focused on metabolic outcomes or general PCOS phenotypes rather than specifically on infertile women with PCOS and their ovulatory status in a South Asian context. There is scarce information about how hormonal profiles (LH, FSH, LH/FSH ratio, total testosterone, DHEA-S, SHBG, fasting insulin, HOMA-IR) relate quantitatively to ovulatory dysfunction specifically in infertile women with PCOS, especially in Bangladesh and neighbouring regions. Many studies report descriptive hormonal data, but fewer examine direct relationships between hormonal markers and documented ovulatory status in PCOS patients (for example, through progesterone testing or ultrasonography) or identify independent predictors of anovulation [10,11]. Additionally, the interplay of metabolic (insulin resistance) and endocrine (gonadotropin, androgen) factors in ovulatory dysfunction in infertile PCOS populations in developing countries remains under-explored. Given the high burden of PCOS, infertility and metabolic derangements among reproductive-aged

women in Bangladesh, there is a compelling need for rigorous, locally relevant data linking hormonal/metabolic profiles to ovulatory outcomes in infertile PCOS patients. Therefore, this study aims to determine the association between hormonal and metabolic profiles and ovulatory dysfunction in infertile women with PCOS, and to identify independent predictors of anovulation in this population.

METHODS & MATERIALS

This cross-sectional study was conducted at the Gynecology and Infertility Department of Gynaecology & Obstetrics, Central Police Hospital, Dhaka, Bangladesh, from January 2024 to December 2024. The study population comprised infertile women aged 18–40 years who were clinically diagnosed with Polycystic Ovary Syndrome (PCOS) according to the Rotterdam criteria (presence of at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound). Women with thyroid disorders, hyperprolactinemia, adrenal disorders, diabetes mellitus, or those using hormonal therapy within three months prior to the study were excluded to minimize confounding factors [12]. A total of 110 participants were recruited using a purposive sampling technique after obtaining informed written consent, and ethical approval was secured from the institutional review board. Data collection included detailed socio-demographic information such as age, residence, education, occupation, and monthly household income. Anthropometric measurements, including height, weight, waist circumference, hip circumference, and body mass index (BMI), were recorded following standard protocols. Blood pressure was measured in a seated position after a five-minute rest. The clinical assessment of hyperandrogenism included hirsutism, scored using the modified Ferriman–Gallwey (mFG) system, and the presence of acne, seborrhea, acanthosis nigricans, and galactorrhea was noted [13]. Fasting venous blood samples were collected in the early follicular phase (days 2–5) of the menstrual cycle or after a progesterone withdrawal test in amenorrheic women. Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone, dehydroepiandrosterone sulfate (DHEA-S), sex hormone-binding globulin (SHBG), prolactin, thyroid-stimulating hormone (TSH), fasting glucose, and fasting insulin were measured using standardized laboratory assays. The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated to evaluate insulin resistance. Data were analyzed using SPSS version 26. Descriptive statistics, including mean ± standard deviation (SD), frequency, and percentage, were calculated for demographic, clinical, and biochemical variables. Pearson or Spearman correlation analysis was performed to examine associations between hormonal/metabolic parameters and ovulatory status. Variables with significant associations in univariate analysis were included in a multivariable logistic regression model to identify independent predictors of anovulation. A p-value <0.05 was considered statistically significant.

RESULTS

The socio-demographic profile of the 110 infertile women with PCOS shows that the majority were in the 25–29 years age group (29.1%), with a mean age of 28.6 ± 6.1 years, indicating a predominantly young reproductive-age population. Most participants resided in urban areas (63.6%) and had at least secondary-level education (88.2% had secondary education or higher). In terms of occupation, over half were housewives (58.2%), while the remainder were employed, engaged in business, or students/other occupations. Regarding household income, the most significant proportion of participants (43.6%) reported a monthly income of 15,000–30,000 BDT, followed by 25.5% in the 30,001–60,000 BDT range, indicating that most women belonged to low- to middle-income households (*Table I*).

Table I
Socio-Demographic Characteristics of Infertile Women with Polycystic Ovary Syndrome (PCOS).

Category	Frequency (n)	Percentage (%)
Age groups		
15–19	6	5.45
20–24	25	22.73
25–29	32	29.09
30–34	22	20.00
35–39	14	12.73
≥40	11	10.00
Mean ± SD	28.6 ± 6.1	
Residence		
Urban	70	63.64
Rural	40	36.36
Education level		
Primary or less	13	11.82
Secondary	42	38.18
Higher secondary	29	26.36
Graduate or above	26	23.64
Occupation		
Housewife	64	58.18
Service (employed)	22	20.00
Business	13	11.82
Student / Other	11	10.00
Monthly household income (BDT)		
<15,000	23	20.91
15,000–30,000	48	43.64
30,001–60,000	28	25.45
>60,000	11	10.00

The anthropometric and clinical assessment of women with PCOS shows that the mean body mass index (BMI) was 29.8 ± 5.4 kg/m², with 40% classified as obese, 39% as overweight, and only 21% within the normal BMI range, indicating a high prevalence of overweight and obesity in this population. The mean waist and hip circumferences were 92.6 ± 12.5 cm and 106.2 ± 10.9 cm, respectively, reflecting central adiposity commonly associated with metabolic risk in PCOS. Blood pressure measurements were within normal limits on average, with a mean systolic BP of 118 ± 12 mmHg and diastolic BP of 76 ± 9 mmHg (*Table II*).

Table II
Anthropometric and Clinical Parameters of Women with PCOS.

Parameter	Mean ± SD	Range
BMI category	Normal (18.5–24.9)	21 (21.0%)
	Overweight (25–29.9)	39 (39.0%)
	Obese (≥30)	40 (40.0%)
BMI (kg/m²), Mean ± SD	29.8 ± 5.4	18.2 – 44.6
Waist circumference (cm)	92.6 ± 12.5	68 – 132
Hip circumference (cm)	106.2 ± 10.9	84 – 136
Systolic BP (mmHg)	118 ± 12	90 – 150
Diastolic BP (mmHg)	76 ± 9	56 – 96

The distribution of clinical features of hyperandrogenism among women with PCOS indicates that hirsutism was the most prevalent feature, observed in 55.5% of participants, with a mean modified Ferriman–Gallwey (mFG) score of 10.8 ± 4.6. Moderate-to-severe acne was present in 33.6% of women, while acanthosis nigricans, a marker of insulin resistance, was noted in 29.1% of participants. Seborrhea was observed in 18.2% of the women, and galactorrhea was the least common feature, affecting only 3.6% (*Table III*).

Table III
Distribution of Clinical Features of Hyperandrogenism among Women with PCOS.

Feature	Frequency (n)	Percentage (%)
Hirsutism (mFG ≥8)	61	55.45
mFG score mean	10.8 ± 4.6	
Acne (moderate–severe)	37	33.64
Acanthosis nigricans	32	29.09
Seborrhea	20	18.18
Galactorrhea	4	3.64

According to Table 4, the mean LH level was 11.8 ± 6.2 IU/L, with 48.2% of participants exceeding the normal range. In comparison, FSH was elevated in only 11.8%, resulting in a mean LH/FSH ratio of 2.5 ± 1.4, with 59.1% showing a ratio above the typical threshold (<2), reflecting gonadotrophin imbalance. Hyperandrogenism was evident, with 46.4% of women displaying elevated total testosterone (mean 78 ± 24 ng/dL) and 28.2% elevated DHEA-S. SHBG levels were low in 53.6% of participants (mean 28 ± 11 nmol/L), consistent with increased free androgen bioavailability. Other endocrine markers, including prolactin and TSH, were abnormal in 10% and 8.2% of cases, respectively. Metabolic assessment showed that 60.9% had elevated fasting insulin, 67.3% demonstrated insulin resistance by HOMA-IR (>2.5), and 42.7% had hypertriglyceridemia (TG >150 mg/dL). At the same time, mean fasting glucose remained largely within normal limits (94 ± 12 mg/dL) *Table IV*.

Table IV
Hormonal and Metabolic Profile of Infertile Women with PCOS.

Parameter (unit)	Mean ± SD	Range	Lab ref. range (typical)	Abnormal n (%)
LH (IU/L)	11.8 ± 6.2	1.5 – 34.0	1.9–12.5	53 (48.18%)
FSH (IU/L)	5.1 ± 1.9	1.8 – 10.2	3.0–8.0	13 (11.82%)
LH/FSH ratio	2.5 ± 1.4	0.5 – 6.8	<2 (typical)	65 (59.09%)
Total testosterone (ng/dL)	78 ± 24	18 – 170	15–70	51 (46.36%)
DHEA-S (µg/dL)	285 ± 110	80 – 640	35–430	31 (28.18%)
SHBG (nmol/L)	28 ± 11	8 – 62	18–144	59 (53.64%)
Prolactin (ng/mL)	14.7 ± 6.2	5 – 46	4–23	11 (10.00%)
TSH (µIU/mL)	2.1 ± 0.9	0.4 – 6.8	0.4–4.0	9 (8.18%)
Fasting glucose (mg/dL)	94 ± 12	68 – 140	70–99	15 (13.64%)
Fasting insulin (µIU/mL)	20.9 ± 9.6	4 – 60	lab dependent	67 (60.91%)
HOMA-IR	4.2 ± 2.3	0.6 – 12.8	>2.5 indicates IR (approx)	74 (67.27%)
Lipid - TG (mg/dL)	165 ± 62	72 – 360	<150	47 (42.73%)

The correlation analysis between hormonal and metabolic parameters and ovulatory status in women with PCOS demonstrates significant associations. The LH/FSH ratio showed a negative correlation with ovulation ($r = -0.31$, $p = 0.002$), indicating that higher ratios are associated with reduced ovulatory function. Similarly, elevated total testosterone ($r = -0.29$, $p = 0.004$) and higher fasting insulin levels ($r = -0.36$, $p < 0.001$) were significantly associated with anovulation. Insulin resistance, as measured by HOMA-IR, exhibited the strongest negative correlation with ovulation ($r = -0.42$, $p < 0.001$), highlighting its critical role in ovulatory dysfunction. In contrast, SHBG levels were positively correlated with ovulation ($r = 0.28$, $p = 0.005$), suggesting a protective effect of higher SHBG on ovulatory capacity. Total antral follicle count (AFC) did not show a statistically significant correlation ($r = 0.12$, $p = 0.24$) *Table V*.

Table V
Correlation Between Hormonal and Metabolic Parameters and Ovulatory Status in Women with PCOS.

Parameter	r (with ovulatory status)	p-value	Interpretation
LH/FSH ratio	−0.31	0.002	Higher LH/FSH associated with less ovulation
Total testosterone	−0.29	0.004	Higher T associated with anovulation
Fasting insulin	−0.36	<0.001	Higher insulin associated with anovulation
HOMA-IR	−0.42	<0.001	Strongest negative correlation with ovulation
SHBG	0.28	0.005	Higher SHBG associated with ovulation
AFC (total)	0.12	0.24	Not statistically significant in unadjusted analysis

The multivariable logistic regression analysis identifies independent predictors of anovulation among women with PCOS. Age and BMI were not significantly associated with anovulation ($p = 0.78$ and $p = 0.09$, respectively). In contrast, higher LH/FSH ratio significantly increased the odds of anovulation (adjusted OR 1.25, 95% CI 1.04–1.50, $p = 0.017$), as did elevated total testosterone (adjusted OR 1.13 per 10 ng/dL increase, 95% CI 1.02–1.26, $p = 0.018$) and higher HOMA-IR (adjusted OR 1.43 per unit increase, 95% CI 1.13–1.81, $p = 0.003$). Conversely, higher SHBG was protective, reducing the odds of anovulation (adjusted OR 0.81 per 10 nmol/L increase, 95% CI 0.67–0.98, $p = 0.031$) *Table VI*.

Table VI
Multivariable Logistic Regression Analysis Predicting Anovulation Among Women with PCOS.

Predictor	Adjusted OR	95% CI	p-value
Age (per year)	0.99	0.93 – 1.05	0.78
BMI (per kg/m²)	1.05	0.99 – 1.12	0.09
LH/FSH ratio (per unit)	1.25	1.04 – 1.50	0.017
Total testosterone (per 10 ng/dL)	1.13	1.02 – 1.26	0.018
HOMA-IR (per unit)	1.43	1.13 – 1.81	0.003
SHBG (per 10 nmol/L)	0.81	0.67 – 0.98	0.031

DISCUSSION

In our cross-sectional study of 110 infertile women with Polycystic Ovary Syndrome (PCOS) in Bangladesh, we found that gonadotrophin imbalance (elevated LH/FSH ratio), biochemical hyperandrogenism (higher total testosterone), and insulin resistance (higher HOMA-IR) were independently associated with anovulation, while higher SHBG was protective. These findings both align with and extend the existing literature on hormonal and metabolic drivers of ovulatory dysfunction in PCOS, and carry important clinical implications for the management of infertile PCOS women. Regarding the LH/FSH ratio, our finding of an adjusted OR of 1.25 per unit increase (95% CI, 1.04–1.50; $p = 0.017$) for anovulation mirrors prior observations of gonadotropin dysregulation in PCOS. Elevated LH relative to FSH contributes to reduced conception and increased miscarriage rates [14]. More recently, a study by Xia et al. found that an elevated baseline LH/FSH ratio in PCOS was associated with poorer ovulatory response in assisted reproduction.[7] Our results are therefore consistent with the mechanistic model in which excess LH promotes theca-cell androgen production and follicular arrest, while inadequate FSH fails to support mature follicle growth. On the other hand, our observed mean ratio (2.5 ± 1.4, with 59.1% >2) is somewhat lower than in some studies where LH/FSH >3 or >2.5 was more common [15]. This may reflect phenotypic variation by ethnicity, BMI, or infertility-specific sampling. In terms of androgen excess, our finding that elevated total testosterone (adjusted OR 1.13 per 10 ng/dL increase, 95% CI 1.02–1.26, $p = 0.018$) predicts anovulation aligns with evidence linking hyperandrogenism to disrupted folliculogenesis [6,16]. Atakul et al. found that free androgen index (FAI) and testosterone indices correlated strongly with HOMA-IR in PCOS, suggesting coupling between androgen excess and metabolic dysfunction [16]. Our data emphasize that, in infertile PCOS women, androgen elevation remains an independent ovulatory risk factor even when controlling for age and BMI, which were not significant in our multivariable model. Unlike some studies that highlight DHEA-S as a predictor of anovulation, our DHEA-S elevation (28.2%) did not independently predict ovulatory dysfunction [6]. This may reflect a dominant role of ovarian versus adrenal androgens or sample-specific characteristics. Our finding for insulin resistance (HOMA-IR: adjusted OR 1.43 per unit increase, 95% CI 1.13–1.81, $p = 0.003$) being the strongest predictor of anovulation ($r = -0.42$, $p < 0.001$) is consistent with the centrality of insulin resistance in both reproductive and metabolic dimensions of PCOS [4]. Zhao et al. emphasized IR's role in ovulatory dysfunction and the necessity of insulin-sensitizing interventions.[4] Similarly, Wiweko et al. found significant correlations between AMH and HOMA-IR in anovulatory PCOS phenotypes, linking follicle pool markers, insulin resistance, and ovulatory dysfunction [17]. Our study extends these findings to infertile Bangladeshi women, showing that HOMA-IR remains an independent predictor after adjusting for gonadotrophin imbalance and androgen levels. Regarding SHBG, our observation that higher SHBG reduced odds of anovulation (adjusted OR 0.81 per 10 nmol/L increase, 95% CI 0.67–0.98, $p = 0.031$) is consistent with studies showing SHBG as a marker of both

metabolic and reproductive health [18]. Chang et al. reported SHBG as the strongest predictor for ovulation in infertile women with PCOS, independent of testosterone [18]. Low SHBG, often suppressed by hyperinsulinemia and obesity, signals higher ovulatory risk and may guide clinical management. In terms of consistencies and differences with existing literature: The triad of LH/FSH imbalance, hyperandrogenism, and insulin resistance repeatedly emerges in PCOS ovulation [7,6,14-16]. The protective effect of higher SHBG is also well-documented [18]. BMI and age were not significant predictors, highlighting the predominant role of metabolic/hormonal variables in infertile PCOS women [4,17]. Many prior studies focus on ovulation induction outcomes or assisted reproduction rather than spontaneous or cross-sectional anovulatory status [7,19,20]. Our study contributes geographically specific data from Bangladesh. Some studies report higher LH/FSH ratios in lean PCOS [15]; our cohort's overweight/obese profile (mean BMI 29.8 ± 5.4 kg/m²) may explain moderate LH/FSH levels. DHEA-S was not independently predictive in our sample, diverging from studies emphasizing adrenal androgen roles. [6] AFC did not correlate with ovulation ($r = 0.12$, $p = 0.24$), whereas other studies have often shown that higher AFC correlates with PCOS severity [21].

LIMITATIONS

This study was conducted at a single tertiary care center, which may limit the generalizability of the findings. The cross-sectional design precludes establishing causality between hormonal profiles and ovulatory dysfunction. Some hormonal assays were performed at different phases of the menstrual cycle due to patient variability, which might have influenced the results.

CONCLUSION

The study demonstrates a significant association between hormonal imbalances and ovulatory dysfunction among infertile women with PCOS. Elevated LH, LH/FSH ratio, and testosterone levels were key predictors of anovulation, while higher progesterone levels indicated preserved ovulatory function. These findings highlight the importance of hormonal profiling for early diagnosis, individualized management, and improved reproductive outcomes in women with PCOS.

RECOMMENDATIONS

Regular hormonal assessment should be incorporated into the evaluation of infertile women with PCOS to identify anovulatory patterns early. Lifestyle modification and individualized hormonal therapy are recommended to restore ovulation and enhance fertility outcomes. Future large-scale, multicenter longitudinal studies are needed to confirm these associations and explore causal relationships.

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CONFLICT OF INTEREST

None declared

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