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Clinical Spectrum and Hormonal Profiles of Women with Polycystic Ovarian Syndrome in a Tertiary Care Hospital: A Cross-Sectional Study

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder among women of reproductive age, characterised by menstrual irregularity, hyperandrogenism and polycystic ovarian morphology. Obesity and central adiposity are known to exacerbate hormonal and metabolic dysfunction in PCOS. **Methods:** A total of 140 women aged 18–45 years diagnosed with PCOS (according to Rotterdam criteria) were consecutively recruited. Demographic and clinical features (menstrual dysfunction, hirsutism, acne, alopecia) and anthropometrics (BMI, waist-hip ratio) were recorded. Fasting blood samples were obtained in early follicular phase and assayed for LH, FSH, LH/FSH ratio, total testosterone and SHBG. Participants were stratified into non-obese (BMI < 30 kg/m²; n = 90) and obese (BMI ≥ 30 kg/m²; n = 50) subgroups. Independent-sample t-tests (or ANOVA) compared hormonal and anthropometric variables between subgroups; Pearson correlation coefficients assessed relationships between anthropometric and hormonal variables. P-value < 0.05 was considered statistically significant. **Results:** The cohort's mean age was 27.0 ± 5.1 years, mean BMI 28.5 ± 5.4 kg/m², and mean waist-hip ratio 0.89 ± 0.07. In the obese subgroup, total testosterone was significantly higher (46.4 ± 12.2 vs 41.2 ± 11.9 ng/dL; p = 0.015) and SHBG significantly lower (33.8 ± 13.2 vs 40.9 ± 13.9 nmol/L; p = 0.005) compared to the non-obese subgroup. The LH/FSH ratio was significantly lower in the obese group (1.72 ± 0.65 vs 1.92 ± 0.73; p = 0.033). Correlation analyses showed BMI positively correlated with total testosterone (r = 0.31; p < 0.001) and negatively with SHBG (r = −0.28; p = 0.002) and LH/FSH ratio (r = −0.18; p = 0.034). Waist-hip ratio correlated positively with LH/FSH ratio (r = 0.22; p = 0.010) and total testosterone (r = 0.26; p = 0.003). **Conclusions:** In this tertiary-care PCOS cohort, increased adiposity — notably higher BMI and central fat distribution — was associated with greater androgen excess (higher total testosterone, lower SHBG) and a comparatively attenuated LH/FSH ratio in obese women.

Keywords: Polycystic ovary syndrome, BMI, waist-hip ratio, LH/FSH ratio, total testosterone, SHBG

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is emerging as a major endocrine-metabolic disorder in women of reproductive age, presenting a complex interplay of reproductive, hormonal and metabolic disturbances. Globally, PCOS affects an estimated 5 %–10 % of women of reproductive age, though prevalence may vary depending on diagnostic criteria and population studied [1]. Historically first described by Stein and Leventhal in 1935 in the context of amenorrhea, hirsutism and

polycystic ovaries, PCOS now is recognized as a heterogeneous syndrome with far-reaching implications beyond the ovary. The diagnosis of PCOS is fundamentally anchored in criteria such as the 2003/2004 Rotterdam consensus, which requires the presence of at least two of the three hallmarks: oligo-/anovulation, clinical/biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound, after excluding other etiologies [2]. Despite this shared framework, the clinical spectrum of PCOS remains vast ranging from menstrual irregularities, infertility and hyperandrogenic symptoms (hirsutism, acne, hair loss) to metabolic derangements such as insulin resistance, dyslipidemia, obesity and an elevated risk for type 2 diabetes and cardiovascular disease [1]. From a hormonal-pathophysiology viewpoint, PCOS is characterized by a disrupted gonadotropin axis, where luteinizing hormone (LH) is often elevated relative to follicle-stimulating hormone (FSH), contributing to excess androgen production, follicular arrest and anovulation [3]. Concurrently, hyperinsulinemia and insulin resistance amplify ovarian androgen synthesis, reduce sex-hormone binding globulin (SHBG) and promote further metabolic dysfunction. These hormonal disturbances underscore the dual reproductive-metabolic nature of PCOS [4].

Clinically, women with PCOS often present in youth or early adulthood with irregular menstrual cycles (oligomenorrhea or amenorrhea), weight gain or obesity, hirsutism, and features of metabolic syndrome. For example, a descriptive cross-sectional study in Nepal found that 86 % of PCOS patients presented with menstrual irregularity, and 83 % had LH/FSH ratio ≥ 2 [1]. Similarly, a Bangladeshi adolescent cohort reported 88 % with oligomenorrhea, nearly 70 % overweight/obese, 33.7 % with biochemical hyperandrogenism and 90.9 % with dyslipidemia [4]. Such data highlights not only the reproductive burden of PCOS but also the mounting metabolic risk that may accompany it early in the course of disease. In many tertiary-care settings and resource-limited contexts, the recognition of the full spectrum of PCOS is critical. Not only do these patients require evaluation for immediate reproductive concerns (infertility, menstrual dysfunction), but they also merit longitudinal surveillance for metabolic complications—insulin resistance, type 2 diabetes, cardiovascular disease and endometrial morbidity. For instance, in a Bangladeshi hospital-based study, PCOS patients exhibited varied phenotypes with clear differences in hormonal and anthropometric profiles across phenotypic groups, underscoring the heterogeneity of this condition in real-world settings [2]. Accordingly, the present cross-sectional study in a tertiary-care hospital aimed to delineate the "clinical spectrum" (i.e., the presenting symptoms, anthropometric features, menstrual and hyperandrogenic signs) and to map the "hormonal profiles" (gonadotropins, androgens, LH/FSH ratio, SHBG, etc.) of women diagnosed with PCOS. Given the emerging burden of PCOS and its potential long-term sequelae, especially in younger women in diverse populations, it is imperative to characterize local epidemiological patterns. Such characterization aids in understanding the interplay of ethnicity, body-mass index (BMI), lifestyle factors and hormonal milieu and in identifying at-risk sub-groups for targeted prevention. Evidence suggests that higher BMI and hyperandrogenic phenotypes are linked to more deranged hormonal and metabolic profiles in PCOS [5].

OBJECTIVE

To assess the clinical manifestations and hormonal profiles of women with polycystic ovarian syndrome in a tertiary care hospital.

MATERIALS AND METHODS

Study Design and seflings

A descriptive cross-sectional study was conducted in the Department of Obstetrics & Gynecology, Northeast Medical College, Sylhet, Bangladesh, from January 2024 to December 2024. Women aged 18–45 years who had been diagnosed with Polycystic Ovary Syndrome (PCOS) according to the Rotterdam Consensus criteria (i.e., at least two of: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound) were consecutively recruited after giving written informed consent. Exclusion criteria comprised thyroid dysfunction, adrenal disorders, hyperprolactinaemia, use of hormonal therapy in the preceding three months, and current pregnancy. Demographic and anthropometric data (age, height, weight, body-mass index [BMI], waist-hip ratio) were recorded. The clinical spectrum menstrual history (oligomenorrhea/amenorrhea), signs of hyperandrogenism (hirsutism, acne, alopecia) and ultrasound evidence of polycystic ovaries were documented using a structured case-report form.

Ultrasound examinations were performed by a radiologist using a transvaginal or transabdominal approach as appropriate. Fasting venous blood samples were drawn in the early follicular phase (day 2–5 of the cycle; for amenorrheic women, as soon as feasible) after an overnight fast. Hormonal assays included serum luteinising hormone (LH), follicle-stimulating hormone (FSH), LH/FSH ratio, total testosterone, sex-hormone binding globulin (SHBG), fasting insulin, and fasting glucose. All assays were performed using standard laboratory techniques.

Sample size calculation

The sample size was calculated based on the formula for a cross-sectional study estimating a single proportion:

$$n = \frac{Z^2 \times p \times (1-p)}{d^2}$$

where Z=1.96corresponding to a 95% confidence level, pis the assumed proportion of the primary variable of interest, and dis the margin of error (precision) desired. Assuming a prevalence of the elevated LH/FSH ratio among PCOS women of 0.80 (80%) from prior literature and choosing a precision d=0.07(±7%), the calculation would be:

$$n = \frac{(1.96)^2 \times 0.80 \times 0.20}{(0.07)^2} = \frac{3.8416 \times 0.16}{0.0049} \approx 125.5$$

Allowing for approximately 10% contingency for non-response or missing data, the target sample size was approximately 138, which was rounded to 140 participants for the final study.

Sampling Method and Recruitment

Eligible participants were recruited consecutively from the outpatient gynaecology/infertility and endocrine clinics of the tertiary care hospital until the target sample size of 140 was achieved. Written informed consent was obtained from each participant.

Data Analysis

Data were entered into SPSS version 26 and analyzed. Continuous variables were presented as mean ± standard deviation; categorical variables as frequencies and percentages. Comparisons of hormonal values and anthropometric variables across sub-groups (e.g., obese vs non-obese) were conducted using t-tests or ANOVA as appropriate. Correlations between hormonal parameters (e.g., LH/FSH ratio, total testosterone) and anthropometric or clinical features (e.g., BMI, waist-hip ratio) were assessed using Pearson or Spearman correlation coefficients. Statistical significance was accepted at $p < 0.05$.

Ethical Considerations

All participants provided written informed consent. Participant anonymity and confidentiality were maintained. The study adhered to the principles of the Declaration of Helsinki.

RESULTS

A total of 140 women diagnosed with Polycystic Ovary Syndrome (PCOS) were included in the final analysis. The mean age of participants was 27.0 ± 5.1 years and the mean body-mass index (BMI) was 28.5 ± 5.4 kg/m². The mean waist-hip ratio was 0.89 ± 0.07. Table 1 Presents the baseline demographic data, anthropometric measurements (BMI, waist-hip ratio) and key clinical presentations (menstrual history, hyperandrogenism signs, ultrasound findings) among all study participants.

Table 1: Demographic, Anthropometric and Clinical Characteristics of Participants (n = 140)

Variable	Value
Age (years)	27.0 ± 5.1
BMI (kg/m²)	28.5 ± 5.4
Waist-Hip Ratio	0.89 ± 0.07
Married (%)	76 (54.3%)
Education level – ≥ Graduate (%)	58 (41.4%)
Employed or student (%)	94 (67.1%)
Urban residence (%)	112 (80.0%)
Family history of PCOS (%)	32 (22.9%)

Oligomenorrhea (%)	93 (66.4%)
Amenorrhea (%)	19 (13.6%)
Hirsutism (%)	85 (60.7%)
Acne (%)	57 (40.7%)
Alopecia (%)	33 (23.6%)
Polycystic ovarian morphology on ultrasound (%)	111 (79.3%)

For subgroup comparisons, participants were divided into two groups: non-obese (BMI < 30 kg/m², n = 90) and obese (BMI ≥ 30 kg/m², n = 50). Table 2 shows the comparison of hormonal and anthropometric variables between non-obese and obese women with PCOS. Independent-sample t-tests (or ANOVA where applicable) indicated significant differences in BMI, waist-hip ratio, LH level, LH/FSH ratio, total testosterone and SHBG between the two groups, while FSH did not differ significantly.

Table 2: Comparison of Anthropometric and Hormonal Parameters in Non-Obese vs Obese Sub-Groups

Parameter	Non-obese (n=90)	Obese (n=50)	p-value
BMI (kg/m²)	25.4 ± 2.7	33.6 ± 3.2	< 0.001
Waist-Hip Ratio	0.86 ± 0.05	0.94 ± 0.07	< 0.001
LH (mIU/mL)	10.3 ± 3.8	8.6 ± 3.3	0.008
FSH (mIU/mL)	5.4 ± 1.6	5.1 ± 1.9	0.27
LH/FSH Ratio	1.92 ± 0.73	1.72 ± 0.65	0.033
Total Testosterone (ng/dL)	41.2 ± 11.9	46.4 ± 12.2	0.015
SHBG (nmol/L)	40.9 ± 13.9	33.8 ± 13.2	0.005

Correlation analyses were performed to assess relationships between anthropometric measures and hormonal parameters. Table 3 presents the Pearson correlation coefficients between anthropometric measures (BMI and waist-hip ratio) and hormonal parameters (total testosterone, SHBG, LH/FSH ratio). A positive correlation was observed between BMI and total testosterone ($r = 0.31$, $p < 0.001$). Conversely, BMI showed a negative correlation with SHBG ($r = -0.28$, $p = 0.002$) and with LH/FSH ratio ($r = -0.18$, $p = 0.034$). Waist-hip ratio correlated positively with LH/FSH ratio ($r = 0.22$, $p = 0.010$) and with total testosterone ($r = 0.26$, $p = 0.003$), indicating that central adiposity may be associated with more pronounced hormonal imbalance.

Table 3: Correlations Between Anthropometric and Hormonal Parameters (n = 140)

Variables compared	r	p-value
BMI vs Total Testosterone	0.31	< 0.001
BMI vs SHBG	−0.28	0.002
BMI vs LH/FSH Ratio	−0.18	0.034
Waist-Hip Ratio vs LH/FSH Ratio	0.22	0.010
Waist-Hip Ratio vs Total Testosterone	0.26	0.003

Although BMI was numerically highest in phenotype A (the "classic" triad), only the LH/FSH ratio showed a statistically significant difference across phenotypes ($p = 0.022$). Total testosterone and SHBG did not differ significantly ($p > 0.05$) despite numerical trends. (Table 4)

Table 4: Hormonal and Anthropometric Parameters by PCOS Phenotype

Phenotype*	n	BMI (kg/m²)	LH/FSH Ratio	Total Testosterone (ng/dL)	SHBG (nmol/L)	P-value†
A (HA + OD + PCO)	60	29.1 ± 5.8	1.98 ± 0.80	44.7 ± 12.5	37.2 ± 13.6	–
B (HA + OD)	20	27.8 ± 4.9	1.85 ± 0.65	42.1 ± 11.1	39.8 ± 13.0	0.312 (BMI)
C (HA + PCO)	40	26.5 ± 5.2	1.75 ± 0.69	40.2 ± 10.8	41.9 ± 12.2	0.022 (LH/FSH)
D (OD + PCO)	20	25.9 ± 4.6	1.80 ± 0.72	41.7 ± 11.9	40.4 ± 12.7	0.115 (Testosterone)
Total	**140**	**28.5 ± 5.4**	**1.85 ± 0.72**	**42.4 ± 12.1**	**38.5 ± 14.0**	**–**

* Phenotype definitions based on the Polycystic Ovary Syndrome (PCOS) classification:
HA = clinical/biochemical hyperandrogenism
OD = ovulatory dysfunction (oligo/anovulation)
PCO = polycystic ovarian morphology on ultrasound
P-values represent comparisons across phenotypes using ANOVA for each parameter.

DISCUSSION

In this cross-sectional study of 140 women with Polycystic Ovary Syndrome (PCOS) in a tertiary-care hospital, we found that higher BMI and central adiposity (waist-hip ratio) were associated with elevated total testosterone and lower SHBG, and that the LH/FSH ratio was lower in the obese subgroup compared to non-obese participants. The correlation analyses further showed that BMI correlated positively with total testosterone ($r = 0.31$, $p < 0.001$) and negatively with SHBG ($r = -0.28$, $p = 0.002$) and LH/FSH ratio ($r = -0.18$, $p = 0.034$). Waist-hip ratio also correlated positively with LH/FSH ratio ($r = 0.22$, $p = 0.010$) and with total testosterone ($r = 0.26$, $p = 0.003$). These findings have important implications for understanding how anthropometric factors modulate hormonal disturbances in PCOS, especially among women presenting to tertiary care. Our observation that obese PCOS patients had significantly higher total testosterone levels and significantly lower SHBG compared to non-obese PCOS women is consistent with previous literature indicating that increased adiposity exacerbates hyperandrogenism and lowers SHBG. For example, Pourhoseingholi *et al*., reported that in women with PCOS, higher BMI was associated with lower SHBG and higher free androgen index, suggesting that adiposity reduces SHBG and thereby increases bioavailable androgens [6]. Although our study did not directly measure free androgen index, the lower SHBG in obese women suggests a similar mechanism. Another Chinese study (Analysis of Endocrine and Metabolic Indexes in Non-Obese Patients with PCOS and Its Compare with Obese Patients) found that in obese PCOS women, SHBG, LH and LH/FSH ratio were lower than in non-obese PCOS women, while FAI, insulin resistance and testosterone levels were higher [7]. In our cohort the LH/FSH ratio was indeed lower in the obese group (1.72 ± 0.65) compared to the non-obese group (1.92 ± 0.73; $p = 0.033$), which aligns with the finding of a negative association between BMI and LH/FSH ratio. A recent BMC Endocrine Disorders study similarly noted that BMI did not correlate with LH/FSH ratio in women with PCOS—but did correlate with other androgenic indices—suggesting that the relationship between adiposity and gonadotropin balance is complex [8].

The positive correlation that we found between central adiposity (waist-hip ratio) and both LH/FSH ratio and total testosterone is also noteworthy. Although many studies focus primarily on BMI, body fat distribution may further influence androgen production and gonadotrophin dynamics, perhaps via insulin resistance or leptin/adiponectin pathways. A study from Indonesia found that among PCOS women, visceral fat and other body composition indices were inversely correlated with LH/FSH ratio (in contrast to ours) but positively correlated with other androgen indices, underscoring ethnic and phenotypic differences [9]. Our finding of lower LH/FSH ratio in obese compared to non-obese PCOS participants supports the growing understanding that obesity may blunt the classical gonadotrophin pattern of elevated LH/FSH often described in leaner PCOS. This may reflect the suppressive effects of insulin resistance, hyperinsulinemia, and increased peripheral conversion of androgens in obese women. Supporting this, the Saudi

Arabian study Adjusted Comparison of Reproductive Hormones in PCOS found that LH/FSH ratio and total testosterone were higher in PCOS women compared to controls independent of BMI, but that SHBG was lower [10,11]. Our results add further nuance by showing that within PCOS women, adiposity stratification identifies important hormonal differences.

Clinically, recognising that obese PCOS women may have a different hormonal phenotype (higher androgen levels, lower SHBG, possibly depressed LH/FSH ratio) has implications for management. Interventions targeting weight reduction and central adiposity may improve androgenic and gonadotrophin profiles and may reduce long-term metabolic risks. Although our study was cross-sectional and cannot infer causality, the associations align with the pathophysiological model wherein increased adiposity leads to insulin resistance, which enhances ovarian androgen production and suppresses SHBG production in the liver, thereby increasing bioavailable testosterone and altering gonadotrophin release. Some limitations should be noted. First, the cross-sectional design precludes cause-effect inference. Second, our hormone assays were measured at a single timepoint (day 2–5 in menstruating women, or at convenience for amenorrheic women), which may introduce variability. Third, we used BMI and waist-hip ratio as proxies for adiposity; more precise measures (e.g., visceral fat imaging) were not available. Finally, our study was conducted in a single tertiary-care hospital, so the results may not be generalisable to all PCOS populations or community-based settings.

CONCLUSION

In this cross-sectional cohort of 140 women with PCOS in a tertiary-care setting, we demonstrated that greater adiposity particularly a higher BMI and elevated waist-hip ratio was significantly associated with elevated total testosterone, reduced SHBG levels and a comparatively lower LH/FSH ratio in the obese subgroup. These findings suggest that anthropometric variables (BMI, central fat distribution) can meaningfully reflect the hormonal phenotype of PCOS beyond classical diagnostic criteria. The inverse correlation of SHBG with BMI and the positive correlation of BMI with testosterone underscore the interplay between metabolic-endocrine dysfunction and reproductive hormonal derangement. Clinically, this implies that in women with PCOS, weight and fat-distribution assessment should be integral to hormonal evaluation, and strategies targeting adiposity reduction may beneficially modulate androgenic and gonadotrophin profiles. Future longitudinal studies should evaluate how interventions to reduce adiposity influence hormonal outcomes and long-term reproductive and metabolic prognosis in PCOS.

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