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Baseline Clinical, Hormonal, and Insulin Resistance Profile of Women with Polycystic Ovary Syndrome

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Abstract

Background: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder in which insulin resistance (IR) drives reproductive and metabolic abnormalities. Baseline characterization of clinical, hormonal, and IR profiles is critical for personalized management. ***Objective:*** To establish baseline clinical, hormonal, and IR profiles in women with PCOS and examine correlations between IR severity and phenotypic features. ***Methods:*** This randomized controlled trial enrolled 90 women with PCOS (Rotterdam 2003 criteria) and IR (HOMA-IR >2.0) aged 18–40 years at BSMMU, Dhaka, Bangladesh (July 2023–June 2024). Clinical, hormonal, and metabolic parameters were assessed. Correlations with HOMA-IR and comparisons between mild-moderate (HOMA-IR 2.1–3.5) and severe IR (>3.5) groups were performed. ***Results:*** Mean age was 25.3±3.7 years; mean BMI 26.1±2.5 kg/m². Oligomenorrhea (96.7%), hirsutism (90.0%), acanthosis nigricans (73.3%), and primary infertility (81.1%) were common. Hormonal profile showed elevated LH/FSH ratio (1.72±0.59), elevated total testosterone (2.8±0.9 nmol/L), elevated free androgen index (10.5±4.2), and low SHBG (28.4±8.6 nmol/L). Mean HOMA-IR was 3.46±0.96 despite normal fasting glucose. HOMA-IR correlated positively with BMI (r=0.52), waist circumference (r=0.48), testosterone (r=0.41), and FAI (r=0.46), and negatively with SHBG (r=-0.38), but not with LH or LH/FSH ratio. Severe IR group had significantly higher adiposity and androgens and lower SHBG than mild-moderate IR group, with no difference in gonadotropins. ***Conclusion:*** In Bangladeshi women with PCOS, IR severity is associated with greater adiposity and hyperandrogenemia but not with gonadotropin abnormalities. Routine IR assessment is essential for phenotype-guided therapy.

Keywords: Polycystic Ovary Syndrome, insulin resistance, HOMA-IR, hyperandrogenism, LH/FSH ratio, acanthosis nigricans, Bangladeshi women.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is identified as the most prevalent endocrine and metabolic disorder among women of reproductive age, affecting an estimated 8–13% of the global female population [1]. The reported prevalence of PCOS varies based on the diagnostic criteria used; it is approximately 11.8% when diagnosed using the Rotterdam criteria, whereas it decreases to about 7.7% with the more restrictive NIH criteria[1]. Traditionally, PCOS diagnosis relies on the presence of at least two of three features: clinical or biochemical signs of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology seen on ultrasound [2]. However, this diagnostic framework does not fully capture the complex hormonal and metabolic disturbances characteristic of the condition[2]. A crucial aspect of PCOS is insulin resistance, which significantly contributes to its heterogeneity. Elevated insulin levels stimulate increased ovarian androgen production, reduce hepatic synthesis of sex hormone-binding globulin (SHBG), and elevate the risk for developing type 2 diabetes mellitus and cardiovascular diseases over time [3]. Recent evidence underscores a bidirectional relationship between insulin sensitivity and secretion, perpetuating the reproductive and metabolic

abnormalities observed in PCOS [4]. Furthermore, insulin resistance and clinical manifestations differ widely across populations, influenced by factors such as diagnostic criteria, body mass index (BMI), and age [5]. Conducting a comprehensive baseline assessment—including clinical examination, hormonal profiling, and evaluation of insulin resistance—is essential in both research and clinical management of PCOS. Such evaluations provide detailed phenotypic characterization, facilitate comparisons across studies, and create reference points for monitoring the effectiveness of treatments such as lifestyle interventions and medications like metformin. Phenotyping also enables stratification of patients, which is critical for personalized treatment plans and risk stratification [6]. The significance of understanding the metabolic implications of PCOS was highlighted in the 2023 International Evidence-based Guideline for PCOS, endorsed by multiple professional organizations. This guideline emphasizes the importance of thorough patient evaluation and evidence-based, individualized management strategies[6]. The present study aims to characterize the baseline clinical, hormonal, and insulin resistance profiles of women diagnosed with PCOS. By systematically assessing anthropometric measurements, menstrual history, hirsutism severity, circulating androgen levels, gonadotropin ratios, and surrogate markers of insulin resistance such as HOMA-IR and fasting glucose-to-insulin ratio, we intend to delineate the phenotypic diversity within our cohort. These data will improve understanding of PCOS heterogeneity and serve as a reference for future research and therapeutic approaches aimed at evaluating long-term health outcomes in this population [7,8]. Accurate baseline characterization is fundamental for advancing personalized management strategies and improving patient outcomes in women with PCOS.

OBJECTIVES

General Objective

This study aimed to carefully establish and characterize baseline clinical, hormonal, and insulin-resistance profiles in women with Polycystic Ovary Syndrome (PCOS).

Specific objectives

1. To document clinical features (menstrual pattern, hirsutism, BMI, acanthosis nigricans).
2. To measure hormonal parameters (LH, FSH, LH/FSH, testosterone, SHBG, FAI, DHEAS).
3. To assess insulin resistance (fasting glucose, insulin, HOMA-IR) and its correlation with clinical and hormonal parameters.

MATERIALS & METHODOLOGY

This randomized controlled trial was conducted from July 2023 to June 2024 in the Department of Reproductive Endocrinology & Infertility at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh. The study population comprised women with Polycystic Ovary Syndrome (PCOS) who had documented insulin resistance and were attending the outpatient department of Reproductive Endocrinology and Infertility at BSMMU. A total of 90 women were enrolled using simple random sampling. To be eligible, participants had to be aged 18 to 40 years and diagnosed with PCOS according to the Rotterdam 2003 criteria, requiring at least two of the following three features: oligo-ovulation or anovulation (menstrual cycles exceeding 35 days or fewer than eight cycles per year), clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound (defined as twelve or more follicles measuring 2–9 mm or an ovarian volume greater than 10 mL). Additionally, all enrolled women were required to have insulin resistance, defined as a Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) value greater than 2.0. Exclusion criteria included pregnancy, lactation, use of hormonal contraceptives, metformin, or anti-androgens within the three months prior to enrolment, known type 1 or type 2 diabetes mellitus, other causes of hyperandrogenism (such as congenital adrenal hyperplasia, androgen-secreting tumours, Cushing's syndrome, hyperprolactinaemia, or thyroid dysfunction), and chronic systemic illnesses. After obtaining written informed consent, a detailed medical and menstrual history was recorded for each participant, including age, menstrual pattern, presence of hirsutism, acne, acanthosis nigricans, and primary infertility. Anthropometric measurements were performed: body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared, with overweight defined as BMI 23.0–27.5 kg/m² and obesity as greater than 27.5 kg/m² according to Asian criteria; waist circumference was measured at the midpoint between the lowest rib and the iliac crest. Hirsutism was assessed using the modified Ferriman-Gallwey scoring system, with a score of eight or more indicating clinical significance. Venous blood samples were collected between days two and five of a spontaneous menstrual cycle, or on any random day in amenorrhoeic women, after an overnight fast of ten to twelve hours. Serum was separated and stored at −20°C until analysis. Hormonal parameters, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone, sex hormone-binding globulin (SHBG), and dehydroepiandrosterone sulfate (DHEAS), were measured using chemiluminescence immunoassays. The free androgen index (FAI) was calculated as (total testosterone × 100)/SHBG. Fasting plasma glucose was measured by the glucose oxidase method, and fasting insulin by enzyme-linked immunosorbent assay. HOMA-IR was calculated as [fasting glucose (mmol/L) × fasting insulin (μIU/mL)] / 22.5. Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation. Pearson's correlation coefficient was used to assess relationships between HOMA-IR and clinical or hormonal parameters. Patients were stratified into mild-moderate

insulin resistance (HOMA-IR 2.1–3.5) and severe insulin resistance (HOMA-IR >3.5), and comparisons between groups were made using the independent t-test. A p-value less than 0.05 was considered statistically significant. The study protocol was approved by the Institutional Review Board of BSMMU, and all procedures followed the ethical standards of the Declaration of Helsinki.

RESULTS

A total of 90 women with PCOS were enrolled. The baseline data were used to characterize the clinical, hormonal, and insulin resistance profiles. All participants had insulin resistance defined by HOMA-IR >2.

Table 1: Clinical features of the study population (n=90)

Clinical parameter	Value
Age (years, mean ± SD)	25.3 ± 3.7
BMI (kg/m², mean ± SD)	26.1 ± 2.5
Waist circumference (cm, mean ± SD)	90.0 ± 6.1
Oligomenorrhea [n (%)]	87 (96.7)
Hirsutism [n (%)]	81 (90.0)
Acne [n (%)]	41 (45.6)
Acanthosis nigricans [n (%)]	66 (73.3)
Primary infertility [n (%)]	73 (81.1)

Table 1 summarizes the clinical features of 90 women, with a mean age of 25.3 years. The average BMI was 26.1 kg/m², classified as overweight per Asian standards, and mean waist circumference was 90.0 cm. Oligomenorrhea was noted in 96.7%, hirsutism in 90.0%, and acanthosis nigricans in 73.3%. Acne occurred in 45.6%, and primary infertility was reported in 81.1%. The typical PCOS phenotype here features menstrual irregularity, hyperandrogenism, and metabolic issues.

Table 2: Hormonal parameters at baseline (n=90)

Hormonal parameter	Mean ± SD	Reference range (follicular phase)
Serum LH (µIU/mL)	9.2 ± 3.0	1.9 – 12.5
Serum FSH (µIU/mL)	5.5 ± 1.4	2.5 – 10.2
LH/FSH ratio	1.72 ± 0.59	<1.0 (typical for PCOS >1.5)
Total testosterone (nmol/L) *	2.8 ± 0.9	0.5 – 2.6
SHBG (nmol/L)	28.4 ± 8.6	18 – 114
Free androgen index (FAI)	10.5 ± 4.2	<5.0
DHEAS (µg/dL)	285 ± 72	35 – 430

Total testosterone values are estimated; free testosterone was 2.92±2.02 pg/mL (normal 0.7–3.6). FAI = (total testosterone / SHBG) × 100.

Table 2 shows the baseline hormonal profile. The serum luteinizing hormone (LH) averaged 9.2±3.0 µIU/mL, within the normal follicular phase range of 1.9–12.5. Follicle-stimulating hormone (FSH) was 5.5±1.4 µIU/mL, within 2.5–10.2. The LH/FSH ratio was 1.72±0.59, above the 1.5 cutoff for PCOS. Total testosterone was elevated at 2.8±0.9 nmol/L (normal 0.5–2.6), free testosterone was 2.92±2.02 pg/mL (normal 0.7–3.6). SHBG levels were 28.4±8.6 nmol/L, normal to low (18–114). The FAI was high at 10.5±4.2, with normal below 5.0. DHEAS was 285±72 µg/dL, within 35–430. The profile indicates elevated LH, increased LH/FSH ratio, mild hyperandrogenemia with raised testosterone and FAI, and normal to low SHBG.

Table 3: Insulin resistance parameters at baseline (n=90)

Parameter	Mean ± SD	Cut-off for IR
Fasting glucose (mmol/L)	5.15 ± 0.40	<5.6 (normal)
Fasting insulin (µIU/mL)	15.2 ± 4.2	>10 (elevated)
HOMA-IR	3.46 ± 0.96	>2.0 (insulin resistant)

Table 3 shows baseline insulin resistance parameters. Mean fasting glucose was 5.15±0.40 mmol/L, within normal (<5.6 mmol/L). Fasting insulin was elevated at 15.2±4.2 µIU/mL (normal <10 µIU/mL). The HOMA-IR value was 3.46±0.96, range 2.1–5.8. All participants had HOMA-IR > 2.0, confirming insulin resistance. Fasting glucose was normal in all women, indicating normoglycemic insulin resistance, a key PCOS feature.

Table 4: Correlation between HOMA-IR and clinical/hormonal parameters

Parameter	Correlation coefficient (r) with HOMA-IR	P value
BMI (kg/m²)	0.52	<0.001
Waist circumference (cm)	0.48	<0.001
Fasting insulin (µIU/mL)	0.95	<0.001
Total testosterone	0.41	0.002
Free testosterone	0.44	<0.001
SHBG	-0.38	0.004
Free androgen index (FAI)	0.46	<0.001
LH	0.21	0.052
LH/FSH ratio	0.19	0.078
DHEAS	0.28	0.009

Pearson correlation coefficient used for normally distributed variables; Spearman for non-normal.

Table 4 shows correlations between HOMA-IR and clinical and hormonal parameters. HOMA-IR strongly correlates with fasting insulin ($r = 0.95$, $p < 0.001$). Moderate positive correlations include BMI ($r = 0.52$), waist circumference ($r = 0.48$), total testosterone ($r = 0.41$), free testosterone ($r = 0.44$), free androgen index ($r = 0.46$), and DHEAS ($r = 0.28$). HOMA-IR negatively correlates with SHBG ($r = -0.38$, $p = 0.004$). Weak, non-significant correlations were found with LH ($r = 0.21$, $p = 0.052$) and LH/FSH ratio ($r = 0.19$, $p = 0.078$). These results suggest that increased insulin resistance is associated with higher adiposity, hyperandrogenemia, and lower SHBG, but not with changes in gonadotropins.

Table 5: Comparison of clinical and hormonal parameters by insulin resistance severity

Parameter	Mild-moderate IR (HOMA-IR 2.1–3.5) (n=48)	Severe IR (HOMA-IR >3.5) (n=42)	P value
BMI (kg/m²)	24.8 ± 2.1	27.6 ± 2.4	<0.001
Waist circumference (cm)	87.2 ± 5.4	93.1 ± 5.8	<0.001
Fasting insulin (µIU/mL)	12.1 ± 2.5	18.9 ± 3.6	<0.001
Total testosterone (nmol/L)	2.4 ± 0.7	3.2 ± 0.9	0.002
Free testosterone (pg/mL)	2.3 ± 1.5	3.6 ± 2.2	0.001
SHBG (nmol/L)	32.1 ± 8.2	24.2 ± 7.5	<0.001
FAI	7.8 ± 3.2	13.6 ± 4.5	<0.001
LH (µIU/mL)	8.9 ± 2.9	9.5 ± 3.1	0.342
LH/FSH ratio	1.68 ± 0.55	1.76 ± 0.63	0.512

Data are mean ± SD. P values from an independent t-test.

Table 5 compares clinical and hormonal parameters between women with mild-moderate insulin resistance (HOMA-IR 2.1–3.5, n=48) and those with severe IR (HOMA-IR >3.5, n=42). Women with severe IR had higher BMI (27.6±2.4 vs. 24.8±2.1 kg/m², $p < 0.001$) and waist circumference (93.1±5.8 vs. 87.2±5.4 cm, $p < 0.001$). Fasting insulin was higher in severe IR (18.9±3.6 vs. 12.1±2.5 µIU/mL, $p < 0.001$). Total testosterone (3.2±0.9 vs. 2.4±0.7 nmol/L, $p = 0.002$), free testosterone (3.6±2.2 vs. 2.3±1.5 pg/mL, $p = 0.001$), and FAI (13.6±4.5 vs. 7.8±3.2, $p < 0.001$) were higher in severe IR, while SHBG was lower (24.2±7.5 vs. 32.1±8.2 nmol/L, $p < 0.001$). No significant differences in LH levels (9.5±3.1 vs. 8.9±2.9 µIU/mL, $p = 0.342$) or LH/FSH ratio (1.76±0.63 vs. 1.68±0.55, $p=0.512$). These results show that severe IR associates with worse anthropometric and androgen profiles, but gonadotropin levels stay similar.

DISCUSSION

This randomized controlled trial was conducted from July 2023 to June 2024 at BSMMU's Department of Reproductive Endocrinology and Infertility in Dhaka. It involved women with PCOS and insulin resistance attending the outpatient department, selected by simple random sampling. 90 women were enrolled, with baseline data characterizing their clinical, hormonal, and insulin resistance profiles. All had insulin resistance defined by HOMA-IR >2.

Our cohort's mean age (25.3 years) aligns with typical reproductive age affected by PCOS [9]. The mean BMI of 26.1 kg/m² indicates overweight status per Asian criteria, reflecting prevalent obesity in PCOS [10]. Waist circumference (90.0 cm) surpasses the Asian cut-off (≥80 cm), indicating abdominal adiposity [11]. Oligomenorrhea was observed in 96.7%, similar to large cohort reports with 75-90% menstrual irregularities [12]. Hirsutism (90.0%) and acne (45.6%) show clinical hyperandrogenism, comparable to South Asian studies [13,14]. Acanthosis nigricans in 73.3%, much higher than Western populations (30-50%) [15], may indicate severe insulin resistance, possibly due to genetic factors or severity in Bangladeshi women [16]. Primary infertility (81.1%) highlights PCOS's reproductive impact.

The mean LH level (9.2 µIU/mL) was within the normal follicular range but near the upper limit. The elevated LH/FSH ratio (1.72) is typical of PCOS, found in about 60-70% of cases [17]. However, a normal ratio doesn't exclude PCOS, supporting its diagnostic value [18. Hyperandrogenemia was shown by high total testosterone (2.8 nmol/L) and free testosterone (2.92 pg/mL). The FAI (10.5) was over twice the normal (<5.0), indicating significant androgen excess. SHBG levels (28.4 nmol/L) were low-normal, expected due to insulin resistance reducing SHBG [19]. DHEAS (285 µg/dL) was normal, suggesting adrenal androgen excess isn't prominent here, contrasting with some studies showing elevated DHEAS in up to 25% of PCOS women [20].

All participants had HOMA-IR >2.0 (mean 3.46), confirming insulin resistance despite normal fasting glucose (5.15 mmol/L). This pattern of normoglycemic insulin resistance is characteristic of PCOS and precedes the development of impaired glucose tolerance and type 2 diabetes [21]. The mean fasting insulin (15.2 µIU/mL) was markedly elevated, consistent with compensatory hyperinsulinemia. Similar HOMA-IR values have been reported in PCOS cohorts from India and Bangladesh [22,23].

HOMA-IR correlated strongly with fasting insulin (r=0.95). Moderate positive correlations with BMI (r=0.52) and waist circumference (r=0.48) confirm adiposity, especially central obesity, as a key driver of insulin resistance in PCOS [24]. These results align with Lim et al.'s meta-analysis, showing each 1 kg/m² increase in BMI links to a 0.2 unit rise in HOMA-IR [25]. HOMA-IR also correlated positively with total testosterone (r=0.41), free testosterone (r=0.44), and FAI (r=0.46), and negatively with SHBG (r=−0.38). These support the bidirectional relationship between hyperinsulinemia and hyperandrogenism: insulin promotes ovarian androgen production and reduces hepatic SHBG, while androgens can worsen insulin sensitivity [26]. Similar correlations are reported in large PCOS studies [27]. Notably, the correlations between HOMA-IR and LH (r=0.21, p=0.052) and LH/FSH ratio (r=0.19, p=0.078) were weak and nonsignificant, indicating insulin resistance likely does not directly influence gonadotropin secretion. Elevated LH in PCOS mainly results from increased hypothalamic GnRH pulse frequency, probably independent of metabolic status [28]. Pagán *et al.,* also found no correlation between LH and insulin sensitivity in PCOS women [29].

Stratifying by HOMA-IR severity revealed that women with severe IR (HOMA-IR >3.5) had significantly higher BMI, waist circumference, and fasting insulin compared to those with mild-moderate IR (HOMA-IR 2.1-3.5). This confirms a dose-dependent relationship between adiposity and insulin resistance [30]. More importantly, the severe IR group had significantly higher total testosterone, free testosterone, and FAI, and lower SHBG. These findings indicate that more severe insulin resistance is associated with a more pronounced hyperandrogenic phenotype. This has clinical implications: PCOS women with severe IR may require more aggressive metabolic and antiandrogen therapy [31]. However, LH levels and LH/FSH ratio did not differ between the two groups, reinforcing that gonadotropin abnormalities are relatively independent of metabolic status.

Limitations of this study

This study's strengths include its randomized controlled trial design, a well-characterized cohort with strict Rotterdam diagnostic criteria, comprehensive hormonal and metabolic profiling, and analyses of correlations and severity. Simple random sampling minimized bias. Limitations include its cross-sectional baseline analysis preventing causality, use of HOMA-IR instead of the gold standard hyperinsulinemic-euglycemic clamp, single-center tertiary hospital setting potentially causing selection bias, not measuring free testosterone by equilibrium dialysis, and lack of lifestyle factor assessments such as diet and physical activity.

CONCLUSION

This randomized controlled trial at BSMMU from July 2023 to June 2024 involved 90 PCOS women selected randomly. The typical phenotype included oligomenorrhea, hirsutism, acanthosis nigricans, high LH/FSH ratio, hyperandrogenemia, low SHBG, and insulin resistance. HOMA-IR positively correlated with adiposity and androgens, negatively with SHBG, but not with LH or LH/FSH ratio. Severe insulin resistance linked to worse anthropometric and hyperandrogenic profiles but similar gonadotropin levels. These results emphasize insulin resistance's role in PCOS's hyperandrogenic phenotype without affecting gonadotropins. Long-term studies are needed to see if reducing insulin resistance lowers androgens and improves ovulation. Trials comparing spironolactone doses in metabolically stratified PCOS women could personalize treatment. Genetic studies are also needed to understand why South Asian PCOS women have more severe insulin resistance.

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