

Research Article

Impact of Insulin Resistance in Women with Polycystic Ovary Syndrome

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ABSTRACT

Background: Polycystic ovary syndrome is the most common endocrinopathy in reproductive-aged women, throughout the life, from adolescence to menopause. Insulin resistance is its central pathophysiological feature. Polycystic ovary syndrome can have an impact on both reproductive and metabolic health.

Objective: To assess the impact of insulin resistance, in polycystic ovarian syndrome, in reproductive-age women.

Methodology: This descriptive, cross-sectional study was conducted at Department of Medicine, M. Islam Medical & Dental College, Gujranwala, Pakistan from 1st October 2025 to 31st March 2026 and 295 women aged 18-45 years diagnosed with polycystic ovarian syndromes were enrolled. Demographic, anthropometric, clinical, and hormonal data were recorded. Fasting blood glucose and fasting serum insulin levels were measured, and insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).

Results: The mean age of participants was 27.8±5.6 years, and the mean body mass index was 27.4±4.8 kg/m². Insulin resistance (HOMA-IR ≥2.5) was present in 75.6% of women, with a mean HOMA-IR value of 4.1±2.0. Insulin resistance was more prevalent among overweight and obese women, affecting 85.3% of those with body mass index ≥25 kg/m². Clinical features such as oligo/anovulation (82.8%), hyperandrogenism (84.1%), and elevated luteinizing hormone/follicle-stimulating hormone ratio (81.5%) were significantly associated with insulin resistance.

Conclusion: Insulin resistance is highly prevalent in reproductive-age women with polycystic ovarian syndromes and has impact on both metabolic and reproductive features of the syndrome.

Keywords: Polycystic Ovarian Syndrome, Insulin Resistance, HOMA-IR, Reproductive-Age Women, Metabolic Dysfunction.

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is among the most widespread endocrine diseases in women of reproductive age. Its prevalence is between 6 and 20% worldwide, depending on the diagnostic numbers and the population under investigation.^{1,2} Polycystic ovarian syndrome is a diagnosis of exclusion, based primarily on the presence of hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology (PCOM).² Biochemically, insulin

resistance and elevated luteinizing hormone (LH) levels are frequently observed, even in the absence of evident metabolic disease. Insulin resistance is a central pathophysiological feature, contributing not only to hyperandrogenism and ovulatory dysfunction but also to metabolic complications such as obesity, dyslipidemia, and impaired glucose tolerance; these abnormalities may occur in lean individuals, emphasizing that metabolic

imbalances are intrinsic to the syndrome rather than purely a consequence of excess weight.³ In a modern environment, poor-quality diet and a range of environmental and lifestyle factors result in chronic activation of the immune system and metaflammation. Inflammation induces a rapid adaptive metabolic response by down regulating insulin signaling and GLUT4 translocation, resulting in reduced insulin sensitivity and glucose uptake, Insulin Resistance (IR), and increased serum glucose levels. This is a physiological response to redirect energy to immune cells and vital organs. Both IR and inflammation can increase ovarian androgen production, which augments IR and down regulates ovulation. Insulin directly stimulates androgen production by theca cells. Insulin may increase anti-Müllerian hormone (AMH) production by granulosa cells, potentially contributing to follicular arrest, the accumulation of small antral follicles, and ovulatory disturbance.⁴ PCOS extends beyond the reproductive system, involving metabolic, cardiovascular, dermatological, and psychological aspects (anxiety, depression, and body image issues related to hirsutism, acne, and weight). Therefore it has been suggested that the name should be changed and several alternative names have been proposed including polyendocrine chronic ovulatory syndrome (PCOS).³

Current clinical practice emphasizes glucose-centric approaches in PCOS management, and IR testing is often overlooked. However, this approach has led to delayed diagnosis and progressive metabolic and reproductive consequences. Especially as in its early stages, PCOS can be considered a reversible disturbance of physiology in response to environmental stressors, rather than a true disease entity, and many of the symptoms and features of PCOS are reversible following diet, exercise, and other lifestyle interventions. An insulin-centric model for PCOS management is a new approach. By focusing on IR, clinicians can potentially limit the progression of PCOS-related reproductive and metabolic diseases.⁴

The homeostasis model assessment of insulin resistance (HOMA-IR) is an efficient and noninvasive method to estimate insulin sensitivity from the steady glucose and insulin concentrations measured under fasting conditions. In epidemiologic studies, HOMA-IR is accepted as the standard method to detect insulin resistance.⁵

The diagnostic criteria of polycystic ovary syndrome have changed over time. Initially, a

subset of criteria was suggested at a 1990 meeting of the National Institutes of Health (NIH) in which both clinical/biochemical hyperandrogenism and chronic anovulation were required for the diagnosis.⁶ The consensus workshop group, sponsored by the European Society of Human Reproduction and Embryology and the American Society for Reproductive Medicine, proposed that at least two of the following three criteria were mandatory: oligo-anovulation, clinical/biochemical hyperandrogenism and polycystic ovary (PCO) appearance on ultrasonography (Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group, 2004). Most recently, the Androgen Excess and PCOS (AE-PCOS) Society² recommended that clinical or biochemical hyperandrogenism should be essential for diagnosis, but also ovulatory dysfunction was required in the form of oligo-anovulation or PCO.

Polycystic ovarian syndrome manifestations vary in severity and define four clinical phenotypes. PCOS phenotypes A: hyperandrogenism (HA) + ovulatory dysfunction (OD) + polycystic ovary morphology (PCOM), B: (HA + OD), C: (HA + PCOM), and D: (OD + PCOM). D is the only type not having hyperandrogenism (HA). All phenotypes exhibited elevated HOMA-IR.⁷ Studies have shown that patients with phenotype D had higher mean values of waist circumference and higher mean levels of serum testosterone, androstenedione, and LH than controls.^{7,8} Phenotype D had milder endocrine and metabolic abnormalities than phenotype A.⁸ Insulin resistance and hyperandrogenism, especially in PCOS, are important to the relationship. High insulin levels stimulate theca cells to produce ovarian androgens and inhibit hepatic production of sex hormone-binding globulin, resulting in high levels of serum free androgens.⁹ This hormonal derangement leads to such clinical manifestations as hirsutism, acne, anovulation, and infertility, which supports the directional association between metabolic and reproductive dysfunction.¹⁰ Therapeutic approaches should target hyperandrogenism, the consequences of ovarian dysfunction and/or the associated metabolic disorders.¹¹ The severity of hirsutism has impact on the metabolic syndrome in PCOS. The severity of hirsutism (assessed using mFG score) showed the mean mFG score was 5.5. Metabolic syndrome was present in 44%. About 65.2% of patients with score ≥ 8 had metabolic syndrome, whereas only 37.7% of patients with score < 8 had metabolic syndrome ($P = 0.019$).¹²

Treatment of PCOS must be tailored to the specific needs of each patient; goals of therapy may include ameliorating hyperandrogenic symptoms, inducing ovulation, regulating menstruation and preventing cardiometabolic complications.¹³

Early detection of insulin resistance among PCOS women is essential in taking early measures to control insulin resistance to lower the chances of metabolic complications over time. Diagnostic disparities, lifestyle variations, and genetic variations are some of the factors that add to the discrepancies in reported prevalence and severity of insulin resistance in women with PCOS.¹⁴

MATERIALS AND METHODS

This was a descriptive, cross-sectional study conducted at Department of Medicine, M. Islam Medical & Dental College, Gujranwala, Pakistan from 1st October 2025 to 31st March 2026. A total of 295 women of reproductive-age were included. All women aged 18-45 years presenting for evaluation, diagnosed as PCOS based on established diagnostic criteria, having regular or irregular menstrual cycles, having complete clinical, biochemical, and hormonal data records were included in this study. The pregnant, lactating women, known cases of Diabetes Mellitus, Thyroid disorders, Cushing's syndrome, hyperprolactinemia, using hormonal therapy, insulin-sensitizing agents, or oral contraceptives within the last 3 months, known cases of chronic systemic illness affecting glucose metabolism were excluded. Recorded variables included age, body mass index, menstrual history, clinical features of hyperandrogenism, and ultrasound findings. Biochemical evaluation included fasting blood glucose and fasting serum insulin levels. Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index. PCOS diagnosis was established based on clinical, biochemical, and ultrasonographic findings. All laboratory investigations were performed using standard methods under fasting conditions to ensure accuracy and consistency. The BMI (kg/m²) categories used were overweight (25-29) and obese (30 or greater BMI). A waist circumference of < 80 cm and a waist – hip ratio of < 0.85 were taken as normal. Likely insulin resistance was taken as a HOMA-IR ≥ 2.5 . Fasting insulin levels of >15 microunits per millilitre is taken as indicative of insulin resistance.

Data were analyzed using SPSS version 24.0. The association between PCOS and insulin resistance was assessed using chi-square test and independent t-test where appropriate. Correlation analysis was performed to evaluate relationships between insulin resistance indices and clinical parameters. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

The mean age was 27.8 ± 5.6 years, indicating a predominantly young adult population, although over one-third (34.6%) were aged 30 years or above. Age at menarche averaged 12.9 ± 1.4 years. The mean body mass index of two-thirds of participants (66.8%) was ≥ 25 kg/m². Central obesity was common. The mean waist circumference was 88.6 ± 9.7 cm while waist-hip ratio was 0.87 ± 0.06 (Table 1).

Hirsutism showed the highest mean HOMA-IR at 4.9 ± 2.3 , and affected 55.3%. It was followed by a mean HOMA-IR of 4.8 seen in clinical hyperandrogenism and infertility/subfertility. A mean HOMA-IR of 4.7 ± 2.1 was found in menstrual irregularity. The association of clinical and ultrasonographic features of PCOS with HOMA-IR and BMI are shown in Table 2.

The most common metabolic abnormality was a HOMA-IR of ≥ 2.5 demonstrating insulin resistance and seen in 75.6% of women. The most common endocrine dysfunction was LH/FSH ratio of ≥ 2 seen in 66.1%, followed by FSH (IU/L) ≤ 5 found in 58.6% (Table 3).

The severity of insulin resistance was categorized into mild, moderate and severe, according to HOMA-IR range. HOMA-IR range of 2.5–3.9, suggesting mild insulin resistance was most common and was seen in 32.5%. This was associated with mean BMI of 26.4 ± 3.8 kg/m² and waist circumference to 86.7 ± 7.9 cm (Table 4).

Women with BMI ≥ 25 kg/m² had insulin resistance in 85.3% of cases, with an odds ratio of 3.92 (95% CI: 2.32–6.61). Central obesity was similarly associated, 84.0% of women with waist circumference ≥ 80 cm had insulin resistance (OR 3.46, 95% CI 2.01-5.97). Oligo- or anovulation was associated with insulin resistance in 82.8% of cases (OR 2.87, 95% CI 1.68-4.89), while hyperandrogenism showed insulin resistance in 84.1% (OR 3.54, 95% CI 2.02-6.19). Polycystic ovarian morphology and elevated LH/FSH ratio were also significantly associated with insulin resistance, confirming the close metabolic-reproductive link in PCOS (Table 5).

Table 1: Baseline Demographic and Anthropometric Characteristics (N = 295)

Parameter	Mean±SD	Category	No.(%)
Age	27.8±5.6	≥30 years	102 (34.6)
Body mass index	27.4±4.8	≥25 kg/m ²	197 (66.8)
Waist circumference	88.6±9.7	≥80 cm	213 (72.2)
Waist-hip ratio	0.87±0.06	≥0.85	186 (63.1)
Age at menarche	12.9±1.4	≤12 years	121 (41.0)
Duration of symptoms	4.1±2.6	≥5 years	104 (35.3)

Table 2: Clinical And Ultrasonographic Features of PCOS (N = 295)

Feature	Present	Absent	HOMA-IR	BMI
Oligo/anovulation	198 (67.1%)	97 (32.9%)	4.6±2.1	28.6±4.9
Clinical hyperandrogenism	176 (59.7%)	119 (40.3%)	4.8±2.2	29.1±5.0
Polycystic ovaries (USG)	214 (72.5%)	81 (27.5%)	4.4±2.0	27.9±4.6
Hirsutism (FG ≥ 8)	163 (55.3%)	132 (44.7%)	4.9±2.3	29.4±5.1
Acne	141 (47.8%)	154 (52.2%)	4.2±1.9	26.8±4.3
Menstrual irregularity	187 (63.4%)	108 (36.6%)	4.7±2.1	28.8±4.8
Infertility/subfertility	172 (58.3%)	123 (41.7%)	4.8±2.2	29.2±5.0

Table 3: Metabolic and Hormonal Profile (N = 295)

Parameter	Mean±SD	Abnormal Category	No. (%)
Fasting blood glucose (mg/dL)	96.8±12.4	≥100	83 (28.1)
Fasting serum insulin (μIU/mL)	16.9±7.8	≥15	164 (55.6)
HOMA-IR	4.1±2.0	≥2.5	223 (75.6)
Total testosterone (ng/dL)	68.5±21.6	≥70	138 (46.8)
LH (IU/L)	9.8±4.1	≥10	126 (42.7)
FSH (IU/L)	4.5±1.9	≤5	173 (58.6)
LH/FSH ratio	2.3±0.9	≥2	195 (66.1)

Table 4: Prevalence and Severity of Insulin Resistance (N = 295)

Category	HOMA-IR Range	No.	%	BMI	Waist Circumference
Normal sensitivity	<2.5	72	24.4	23.6±3.1	78.9±6.8
Mild resistance	2.5–3.9	96	32.5	26.4±3.8	86.7±7.9
Moderate resistance	4.0–5.9	78	26.4	29.1±4.2	92.4±8.3
Severe resistance	≥6.0	49	16.6	32.2±4.7	98.8±9.6
Overall resistance	≥2.5	223	75.6	28.9±4.9	91.2±9.1

Table 5: Association Between PCOS Features and Insulin Resistance (N = 295)

Feature	IR Present	IR Absent	Odds Ratio	95% CI	p-value
BMI ≥25 kg/m ²	168 (85.3%)	29 (14.7%)	3.92	2.32–6.61	<0.001
Waist circumference ≥80 cm	179 (84%)	34 (16%)	3.46	2.01–5.97	<0.001
Oligo/anovulation	164 (82.8%)	34 (17.2%)	2.87	1.68–4.89	<0.001
Hyperandrogenism	148 (84.1%)	28 (15.9%)	3.54	2.02–6.19	<0.001
Polycystic ovaries	171 (79.9%)	43 (20.1%)	2.01	1.21–3.32	0.002
LH/FSH ratio ≥2	159 (81.5%)	36 (18.5%)	2.68	1.61–4.45	<0.001

DISCUSSION

Polycystic ovarian syndrome represents a complex condition at the intersection of reproductive and metabolic health, with insulin

resistance playing a pivotal role in its pathogenesis.³ Our study assessed the impact of insulin resistance in polycystic ovarian syndrome of reproductive-age women. It was able to show

a high metabolic load in PCOS. Over three-quarters of the research sample had insulin resistance, which demonstrated its pivotal role in the pathophysiology of PCOS. These results underline that PCOS is not merely a reproductive disorder but is also a major metabolic disorder, having long term health consequences. The average HOMA-IR in the current research was 4.1 ± 2.0 , and the data showed that insulin resistance was observed in 75.6 % of the participants. Other studies have also found similar prevalence rates of insulin resistance in women with PCOS, with insulin resistance being found in a significant majority of affected women irrespective of body mass index.^{15,16} Majid et al¹⁵ discovered that insulin resistance based on HOMA-IR was observed in 69%. In this study, insulin resistance had a strong relationship with obesity and central adiposity. Women whose BMI is equal to or above 25 kg/m^2 were insulin-resistant in 85.3%, and those whose waist circumference is equal to or greater than 80 cm, were insulin-resistant in 84%. There were progressive increments in BMI and waist circumference between mild, moderate, and severe dimensions of insulin resistance accompanied by a mean BMI of $23.6 \pm 3.1 \text{ kg/m}^2$ and $32.2 \pm 4.7 \text{ kg/m}^2$ in women who were insulin sensitive and severe insulin resistance respectively. Tosi et al¹⁷ demonstrated that in PCOS women, IR may play a central role in the pathophysiology, which occur with a prevalence of 77.5% in overweight and 93.9% in obese subjects.

Polycystic ovarian syndromes were strongly associated with increased insulin resistance indices. Women who had oligo- or anovulation, hyperandrogenism, hirsutism, and menstrual abnormalities always had a higher mean HOMA-IR, between 4.4 ± 2.0 to 4.9 ± 2.3 . Majid et al¹⁵ observed that hyperandrogenism was present in 53.8% ($n=49$, mean HOMA-IR 3 ± 1.5), obesity in 29% ($n=26$, HOMA-IR 3.5 ± 1.8) while 38.5% ($n=35$) women had primary infertility/subfertility (HOMA-IR 3.5 ± 1.7) and menstrual irregularity in 65.9% ($n=60$, HOMA-IR 3 ± 1.5). These women having IR, have higher frequencies of hyperandrogenism, menstrual abnormality, infertility/subfertility and obesity compared to the subjects without IR. Similar results have been noted by earlier studies,^{18,19} which indicate that insulin resistance is one of the factors leading to ovarian dysfunction through the inhibition of follicular growth and the promotion of androgen excess. The present study demonstrated a significant correlation between hyperandrogenism and insulin resistance with

84.1% of all women with clinical hyperandrogenism showing insulin resistance. Compelling evidence suggests that hyperandrogenism is not just a primary feature of PCOS. Instead, it may be a causative factor for this condition. The majority (60%-80%) of women with PCOS have elevated androgen levels.²⁰ High levels of insulin have been found to stimulate ovarian androgen and decrease sex hormone-binding globulin, and this elevates the free androgen. The association between the severity of insulin resistance and anthropometry and the degree of metabolic dysfunction in PCOS is graded in this study, which highlights a significant degree of metabolic dysfunction development. The waists of the severely insulin-resistant women were significantly larger and their body mass index was high in comparison to the insulin-sensitive. Tabassum et al²¹, observed that prevalence of IR was 34.78% in PCOS. Mean age of patients was 23.72 ± 4.37 years. Waist Hip Ratio (WHR) was raised in 42 (91.30%), impaired FBS was seen in 9 (19.6%) and raised fasting insulin in 16 (34.8%) patients.

These findings highlight the importance of systematic screening for metabolic comorbidities in all patients with PCOS regardless of their age and BMI. Early recognition is critical to prevent long-term complications, including infertility, T2D, and cardiovascular disease.

CONCLUSION

Insulin resistance is highly prevalent among reproductive-age women with polycystic ovarian syndrome and has strong impact on both metabolic and reproductive manifestations of the disorder. A substantial proportion of women demonstrated insulin resistance irrespective of body mass index, although excess body weight and central adiposity significantly intensified its severity. Clinical features such as oligo-anovulation, hyperandrogenism, menstrual irregularities, and elevated luteinizing hormone/follicle-stimulating hormone ratio were closely linked with higher insulin resistance indices.

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