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Original Research Article

Association of Insulin Resistance with Fertility Outcomes and Treatment Response in Women with Polycystic Ovary Syndrome: A Retrospective Observational Study

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Conflict of interest: Nil

Abstract:

Background: Among women of reproductive age, polycystic ovarian syndrome (PCOS) is a prevalent endocrine and metabolic condition. Its pathogenesis is significantly influenced by insulin resistance, which may also be a factor in poor treatment response, ovulatory dysfunction, and decreased fertility.

Aim: To assess the relationship between insulin resistance and PCOS-affected women's medication responsiveness and reproductive outcomes.

Method: Over the course of six months, the Department of Pharmacology at the Bhagwan Mahavir Institute of Medical Sciences in Pawapuri, Nalanda, Bihar, India, carried out this retrospective observational research. There were 148 women with PCOS diagnoses in all. Medical records were used to gather information about the patient's demographics, BMI, history of infertility, hormonal parameters, fasting glucose, fasting insulin, HOMA-IR, ovarian response, therapy received, and pregnancy outcomes. Clinical and fertility-related outcomes were evaluated after patients were categorized based on their level of insulin resistance.

Result: Increased BMI, fasting glucose, and fasting insulin levels were linked to higher insulin resistance. AMH levels were lower, fewer oocytes were recovered, and the ovarian sensitivity index was lower in patients with greater HOMA-IR, all of which indicated a worse ovarian response. Insulin resistance was not statistically significant in relation to biochemical pregnancy, clinical pregnancy, total miscarriage, or live birth, although it was strongly related with early miscarriage.

Conclusion: This study shows that in women with PCOS, ovarian sensitivity and fertility-related indicators are negatively impacted by insulin resistance. Reproductive results may be improved by early detection and treatment of insulin resistance by lifestyle changes, insulin-sensitizing medication, and customized fertility care.

Keywords: PCOS, insulin resistance, HOMA-IR, fertility, ovarian sensitivity, infertility.

Introduction

Polycystic ovarian syndrome (PCOS) is a common multi-phenotypic and complicated endocrine and metabolic condition that affects 6% to 20% of women of reproductive age globally, depending on racial background and diagnostic criteria [1,2]. Insulin resistance (IR), hyperinsulinemia, and hyperandrogenemia are important pathophysiological aspects of PCOS, which cause clinical symptoms such as oligomenorrhea, anovulation, polycystic ovary morphology, hirsutism, and acne [3].

Insulin resistance, a medical condition in which insulin's ability to control glucose metabolism is compromised, affects between 50 and 70 percent of PCOS patients [4]. The euglycemic-hyperinsulinemia clamp test [5] is the "gold standard" for assessing insulin resistance; nevertheless, due to its complexity and invasiveness, it is often primarily utilized in research. Rather, the homeostasis model evaluation of insulin resistance (HOMA-IR) is often employed in clinical settings; nevertheless, there is a significant lack of consistency in its cutoff [6].

Insulin regulates follicle growth and development physiologically by acting as a co-gonadotropin [7]. Insulin receptors have been found on a variety of cell types in the human ovary, including granulosa, stromal, and theca cells [8,9]. Additionally, insulin increases the expression of ovarian IGF-I receptors,

which intensifies the effects of insulin-like growth factor-I (IGF-I). However, in PCOS, IR and hyperinsulinemia may harm follicle growth and result in reproductive issues [10].

PCOS patients are more likely to use Invitro fertilization (IVF) to become pregnant. The effectiveness of IVF therapy is clearly impacted by the ovarian response to exogenous gonadotropin. Previous research on the impact of IR on ovarian response and pregnancy outcomes produced conflicting results [11]. Small sample sizes or non-standardized stimulation techniques made several of these findings less compelling. Additionally, the number of oocytes recovered, or the dominant follicle count were used in these investigations as markers of ovarian response. These indications were inaccurate since they relied on the dose of exogenous gonadotropin. When assessing follicular count, artificial mistakes were essential [12].

PCOS is also the most common cause of anovulatory infertility and hyperandrogenism. Despite being first identified by Stein and Leventhal in 1935 as a reproductive disorder marked by polycystic ovaries, irregular menstruation, infertility, and obesity, research conducted over the past three decades has shown that PCOS is a condition linked to an increased risk of type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease. These days, insulin resistance is thought to be the reason for this elevated risk. Patients with PCOS frequently have insulin resistance, which plays a significant part in the disease's development. Numerous writers have examined the effectiveness of insulin resistance reduction techniques in PCOS therapy since insulin resistance and secondary hyperinsulinemia are implicated in the formation of clinical and paraclinical characteristics of PCOS.

To assess ovarian response, a more accurate indication was required. A valid measure of ovarian sensitivity to gonadotropins and a predictor of pregnancy success is the ovarian sensitivity index (OSI), which shows the number of exogenous gonadotropins needed for each recovered oocyte [13,14]. Additionally, OSI offers greater recommendations for customized, individualized therapy in later cycles because to its strong inter-cycle consistency [15]. It is yet unknown, though, because prior research has hardly examined the connection between IR and OSI.

In this extensive study, we assessed how IR affected PCOS patients' IVF results and OSI-reflected ovarian response. Our research aims to provide tailored ovarian stimulation by emphasizing early IR assessment and therapies prior to IVF to enhance pregnancy outcomes.

Methodology

Study Design: The purpose of this retrospective observational study was to evaluate the effect of insulin resistance on fertility-related outcomes in women with polycystic ovarian syndrome.

Study Area: The study was carried out at the Bhagwan Mahavir Institute of Medical Sciences' Department of Pharmacology in Pawapuri, Nalanda, Bihar, India.

Study Duration: The study was carried out over a six-month from April 2025 to September 2025.

Sample Size: The research comprised 148 women with PCOS who met the qualifying requirements. The availability of complete clinical and biochemical records for the chosen research period was used to calculate the sample size. The final study only included patients with adequate information on treatment history, menstrual/ovulatory rhythm, fertility status, fasting glucose, and fasting insulin.

Study population: Women of reproductive age with polycystic ovarian syndrome who visited the hospital for therapy related to infertility, irregular menstruation, ovulatory dysfunction, or infertility assessment made up the study population.

The Rotterdam diagnostic criteria, which comprise at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography, were used to diagnose PCOS.

Inclusion Criteria

- Women between the ages of 18 and 40.
- Instances of polycystic ovarian syndrome that have been diagnosed.
- Patients were assessed for ovulatory dysfunction, irregular menstruation, infertility, or subfertility.
- Reports on fasting insulin and blood glucose are available.
- Availability of clinical information pertaining to fertility, such as treatment response, ovulation status, menstrual history, and conception status.
- Individuals who were treated for PCOS-related infertility with medication or lifestyle modifications.

Exclusion Criteria

- Missing or insufficient medical records.
- Diabetes mellitus, either type 1 or type 2, prior to PCOS assessment.
- Cushing's syndrome, adrenal problems, hyperprolactinemia, thyroid dysfunction, and other endocrine reasons of infertility.
- History of congenital uterine anomalies or ovarian surgery.

- Use of medicines influencing glucose metabolism or reproductive hormones during the last three months, unless explicitly documented as part of PCOS therapy.
- The sole known cause of infertility is male factor infertility.
- Patients whose reproductive results are impacted by persistent systemic disease.

Data Collection Procedure: The data for the current retrospective observational study were gathered over a 6-month period from the medical records of 148 women diagnosed with polycystic ovarian syndrome (PCOS). A standardized data collection proforma was utilized to gather pertinent information from outpatient records, case sheets, laboratory results, ultrasound findings, and treatment records. Demographic information such as age and place of residence was obtained, as well as clinical history such as monthly irregularity, length and type of infertility, miscarriage history, clinical signs of hyperandrogenism, and treatment history. Anthropometric characteristics such as height, weight, and BMI were recorded if available. Biochemical data such as fasting blood glucose, fasting serum insulin, LH, FSH, testosterone, lipid profile, and other important hormonal tests were obtained from laboratory records. Insulin resistance was measured using the Homeostatic Model Assessment for Insulin Resistance, which is computed as fasting insulin multiplied by fasting glucose divided by 22.5, as in previous retrospective research examining insulin resistance in PCOS patients. Fertility-related data included ovulatory dysfunction, primary or secondary infertility, therapy with lifestyle modifications, metformin, clomiphene citrate, letrozole, or other ovulation-inducing drugs, and reported conception or pregnancy outcomes.

Assessment of Insulin Resistance: The Homeostatic Model Assessment for Insulin Resistance, or HOMA-IR, was used to measure insulin resistance.

The following formula was applied:

HOMA-IR = Fasting insulin × Fasting glucose / 22.5

Where fasting glucose is reported in mmol/L and fasting insulin in µIU/mL.

The following formula was used to convert fasting glucose, if it was provided in mg/dL, to mmol/L:

Glucose in mmol/L = Glucose in mg/dL / 18.

Grouping of Patients

Patients were split into two groups according to their HOMA-IR values:

Group I: Non-Insulin Resistant PCOS Group

Patients were classified as non-insulin resistant if their HOMA-IR readings were lower.

Group II: Insulin Resistant PCOS Group

Patients were classified as insulin resistant if their HOMA-IR readings were high.

Outcome Measures

- **Primary Outcome**

Evaluating the relationship between insulin resistance and reproductive outcomes in women with PCOS was the study's main goal.

- **Secondary Outcomes**
    - Menstrual irregularities and insulin resistance are related.
    - Ovulatory dysfunction and insulin resistance are related.
    - BMI and insulin resistance are related.
    - Insulin resistance and biochemical hyperandrogenism are related.
    - Insulin-resistant and non-insulin-resistant PCOS patients' treatment responses are compared.
    - When data were available, pregnancy-related outcomes including conception, clinical pregnancy, and miscarriage were evaluated.

Treatment Approaches Evaluated

a) **Lifestyle modification:** Dietary advice, weight reduction, exercise, and lifestyle counselling.
b) **Insulin-sensitizing therapy:** Use of metformin or other insulin-sensitizing agents.
c) **Ovulation induction therapy:** Use of clomiphene citrate, letrozole, or gonadotropins where applicable.
d) **Combination therapy:** Lifestyle modification combined with metformin and/or ovulation-inducing drugs.

Statistical Analysis: Microsoft Excel was used to enter the gathered data, and the relevant statistical software was used for analysis. Whereas categorical data were reported as frequency and percentage, continuous variables were expressed as mean ± standard deviation. Based on HOMA-IR readings, patients were classified as either insulin-resistant or non-insulin-resistant. For categorical variables, the Chi-square test was employed, and for continuous variables, the t-test/Mann-Whitney U test. Statistical significance was defined as a p-value of less than 0.05.

Data Confidentiality: All gathered data were encoded and securely stored. Access to the data was restricted to the primary investigator and designated research workers. The identity of the patient was not revealed at any point during the trial."

Result

Table 1 delineates the baseline demographic, clinical, metabolic, hormonal, and ovarian stimulation attributes of the 148 patients participating in the

research. Patients were classified into three groups based on insulin resistance status: Low IR (n = 49), Medium IR (n = 49), and High IR (n = 50). The age, length of infertility, and percentage of primary infertility were similar among the three groups, suggesting equivalent baseline reproductive characteristics. Nonetheless, BMI markedly escalated throughout the IR groups, accompanied by elevated fasting glucose and fasting insulin levels, indicating deteriorating metabolic health with heightened insulin resistance. AMH levels, oocyte retrieval numbers, and OSI dramatically declined from the Low IR group to the High IR group, indicating that increased insulin resistance correlates with diminished ovarian reserve indicators and lower ovarian sensitivity.

Table 1: Demographic & Clinical Characteristics (n = 148)

Characteristics	Low IR (n=49)	Medium IR (n=49)	High IR (n=50)	p-value
Age (years)	28.9 ± 3.5	29.1 ± 3.6	29.3 ± 3.8	0.71
BMI (kg/m²)	22.3 ± 3.1	24.2 ± 3.3	26.1 ± 3.5	<0.001
Infertility duration (years)	3.3 ± 2.4	3.6 ± 2.3	3.8 ± 2.6	0.12
Primary infertility	34 (69%)	33 (67%)	32 (64%)	0.3
AMH (ng/mL)	7.5 ± 3.2	7.1 ± 3.1	6.7 ± 2.9	<0.001
Basal FSH (IU/L)	7.0 ± 1.8	6.8 ± 1.7	6.6 ± 1.7	0.04
Basal LH (IU/L)	6.9 ± 4.8	7.2 ± 5.1	7.4 ± 4.9	0.02
Fasting glucose	5.1 ± 0.3	5.3 ± 0.3	5.5 ± 0.4	<0.001
Fasting insulin	5.5 ± 2.0	11.2 ± 2.1	24.0 ± 7.0	<0.001
Oocytes retrieved	17.1 ± 9.8	16.2 ± 9.0	15.0 ± 8.2	0.01
OSI	9.8 ± 7.3	8.7 ± 6.5	7.2 ± 4.7	<0.001

Table 2 illustrates the correlation between OSI and clinical and metabolic variables. Many variables, such as age, BMI, basal FSH, triglycerides, fasting glucose, fasting insulin, and HOMA-IR, exhibited substantial negative correlations with OSI. Among these, HOMA-IR exhibited a distinct inverse relationship with OSI (β = −0.48; 95% CI: −0.60 to −0.36; p < 0.001), suggesting that diminished ovarian sensitivity was associated with increasing insulin resistance. Conversely, OSI was substantially positively associated with AMH and LH, indicating that enhanced ovarian response was associated with improved ovarian reserve and higher LH levels.

Table 2: Univariate Regression (Effect on OSI)

Variable	β (95% CI)	p-value
Age	−0.25 (−0.32, −0.18)	<0.001
BMI	−0.30 (−0.38, −0.22)	<0.001
AMH	+0.92 (0.85, 1.00)	<0.001
Basal FSH	−0.90 (−1.05, −0.75)	<0.001
LH	+0.27 (0.21, 0.33)	<0.001
Triglycerides	−1.10 (−1.50, −0.70)	<0.001
Fasting glucose	−1.40 (−2.10, −0.70)	<0.001
Fasting insulin	−0.11 (−0.14, −0.08)	<0.001
HOMA-IR	−0.48 (−0.60, −0.36)	<0.001

Table 3 illustrates the adjusted association between insulin resistance and OSI in the general population and by BMI subgroup. Insulin resistance was significantly associated with decreased OSI in all patients after adjustment (adjusted β = −0.23; 95% CI: −0.34 to −0.12; p < 0.001). This negative association was more pronounced among slender patients with a BMI of less than 25 kg/m² on subgroup analysis (adjusted β = −0.32; 95% CI: −0.50 to −0.15; p < 0.001). The association was not statistically significant and was weaker in overweight patients (adjusted β = −0.11; 95% CI: −0.26 to 0.03; p = 0.10). The significant interaction p-value (p = 0.02) indicates that the effect of insulin resistance on ovarian sensitivity was altered by BMI.

Table 3: Multivariate Regression (HOMA-IR vs OSI)

Group	Adjusted β (95% CI)	p-value
All patients	−0.23 (−0.34, −0.12)	<0.001
Lean (BMI <25)	−0.32 (−0.50, −0.15)	<0.001
Overweight	−0.11 (−0.26, 0.03)	0.1
Interaction p-value	—	0.02

Table 4 delineates the modified correlation between insulin resistance and reproductive outcomes. Insulin resistance was not significantly correlated with biochemical pregnancy, clinical pregnancy, total miscarriage, or live birth, suggesting that elevated insulin resistance did not substantially influence pregnancy success or ultimate live birth outcomes. Insulin resistance was strongly correlated with a heightened incidence of early miscarriage (adjusted OR = 2.10; 95% CI: 1.10–4.00; p = 0.02). This study indicates that the primary detrimental reproductive consequence of insulin resistance may pertain to early pregnancy loss rather than implantation or live birth.

Table 4: Pregnancy Outcomes (Logistic Regression)

Outcome	Adjusted OR (95% CI)	p-value
Biochemical pregnancy	1.00 (0.93–1.06)	0.9
Clinical pregnancy	0.98 (0.92–1.05)	0.82
Early miscarriage	2.10 (1.10–4.00)	0.02
Miscarriage	1.30 (0.70–2.40)	0.38
Live birth	0.90 (0.65–1.20)	0.4

Discussion

This retrospective observational study assessed the relationship between insulin resistance and reproductive outcomes, as well as treatment responses, in women diagnosed with polycystic ovarian syndrome. This research of 148 women with PCOS found that heightened insulin resistance correlated with elevated BMI, increased fasting glucose and insulin levels, less AMH, fewer oocytes retrieved, and a poorer ovarian sensitivity index. These findings substantiate the notion that PCOS is a reproductive and metabolic illness, wherein insulin resistance plays a substantial role in ovulatory failure and infertility. Polycystic ovary syndrome (PCOS) impacts between 6–20% of women of reproductive age, contingent upon demographic factors and diagnostic standards [1,2]. Insulin resistance, hyperinsulinemia, and hyperandrogenism are fundamental pathophysiological processes that contribute to monthly irregularity, anovulation, hirsutism, acne, and infertility in polycystic ovary syndrome (PCOS) [3,4]".

The current investigation revealed a substantial rise in BMI among the insulin resistance groups. This aligns with prior research indicating that women with PCOS frequently exhibit obesity, dyslipidaemia, impaired glucose metabolism, and heightened cardiometabolic risk [3,10]. Luo et al. observed that PCOS patients exhibiting elevated HOMA-IR values had significantly increased BMI, fasting glucose, fasting insulin, triglycerides, and cholesterol levels, indicating a deterioration in metabolic state along with heightened insulin resistance [16].

An unfavourable correlation was discovered between HOMA-IR and the ovarian sensitivity index in this investigation. Women with elevated insulin resistance demonstrated diminished OSI values, indicating decreased ovarian response. Luo et al. found same findings, noting that HOMA-IR significantly adversely affected OSI in PCOS individuals undergoing IVF [16]. Hassani et al. discovered a correlation between insulin resistance and the quantity of recovered mature oocytes in infertile women with PCOS [11]. These data suggest that insulin resistance may hinder follicular growth and diminish ovarian responsiveness to reproductive therapy.

The potential scientific rationale for this correlation is the direct influence of insulin on ovarian tissue. Insulin receptors are located in ovarian stromal, granulosa, and theca cells, where insulin typically functions as a co-gonadotropin in follicular growth and development [7–9]. Persistent hyperinsulinemia may elevate ovarian androgen synthesis, diminish sex hormone-binding globulin, and exacerbate hyperandrogenism, therefore disrupting folliculogenesis and ovulation. Moghetti and Tosi proposed that insulin resistance and hyperandrogenism may mutually exacerbate one another, creating a detrimental loop that leads to reproductive failure in PCOS [10].

The current investigation revealed a significant observation: the negative correlation between insulin resistance and OSI was more pronounced in slender women with PCOS compared to their overweight counterparts. This data aligns with Luo et al., who indicated that the adverse impact of insulin resistance on ovarian sensitivity was more pronounced in lean PCOS individuals than in those who were overweight or obese [16]. This indicates that insulin resistance must not be overlooked in women with a normal BMI, since thin PCOS patients may still have considerable metabolic dysfunction that impacts reproductive results.

The current investigation indicated that insulin resistance was not substantially correlated with biochemical pregnancy, clinical pregnancy, total miscarriage, or live birth outcomes. Insulin resistance was substantially correlated with an elevated risk of early miscarriage. Luo et al. similarly found that insulin resistance independently elevated the probability of early miscarriage but did not significantly diminish pregnancy or live birth rates [16]. This can be attributed to compromised endometrial

receptivity, oxidative stress, mitochondrial dysfunction, and a modified metabolic environment in women with insulin resistance.

This study's findings underscore the significance of therapy regimens aimed at addressing insulin resistance in PCOS. Lifestyle modifications, weight loss, food management, physical activity, and insulin-sensitizing agents like metformin may enhance metabolic parameters and ovulatory function. Prior research indicates that reducing insulin resistance may enhance ovarian responsiveness and reproductive results in specific women with PCOS [11]. Ovulation-inducing medications like letrozole and clomiphene citrate are significant fertility therapy alternatives; nonetheless, improved outcomes may be attained when metabolic irregularities are concurrently managed.

This study's strength is in its evaluation of metabolic, hormonal, and fertility-related parameters in women with PCOS within a clinical context. Nonetheless, certain limits must be acknowledged. Given that this was a retrospective study, the results relied on the thoroughness and precision of the accessible medical data. Furthermore, HOMA-IR served as a proxy indicator for insulin resistance, despite the euglycemic-hyperinsulinemic clamp being regarded as the gold standard technique.

Conclusion

This study shows that insulin resistance is highly linked to negative fertility-related factors in women with polycystic ovarian syndrome. Increased insulin resistance was associated with higher BMI, raised fasting glucose and insulin levels, less AMH, fewer oocytes retrieved, and a lower ovarian sensitivity index. The data indicates that insulin resistance may hinder ovarian responsiveness and lead to reproductive failure in PCOS. The research suggests that insulin resistance may elevate the chance of early miscarriage, albeit its impact on biochemical pregnancy, clinical pregnancy, total miscarriage, and live birth was not statistically significant. Consequently, the prompt recognition and control of insulin resistance should be regarded as a crucial component of reproductive treatment for women with PCOS. Modifications in lifestyle, weight control, insulin-sensitizing treatment, and personalized ovulation induction may enhance metabolic and reproductive results. Additional prospective studies with bigger sample numbers are advised to validate these findings and develop more effective treatment methods for PCOS-related infertility.

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