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Polycystic Ovary Syndrome and Insulin Resistance: Comparative Analysis of Obese and Non-Obese Women in a Tertiary Care Setting Pakistan

Zara Gul[1] and Gulfreen Waheed[1*]

[1]Department of Obstetrics and Gynaecology, Avicenna Hospital, Lahore, Pakistan

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Corresponding Author:
Gulfreen Waheed
Department of Obstetrics and Gynaecology, Avicenna Hospital, Lahore, Pakistan
gfwaheed@avicennamch.com

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ABSTRACT

Polycystic ovarian syndrome is a widespread endocrine disease that is linked to insulin resistance, regardless of obesity. This correlation is especially pertinent to South-Asian groups, where culture and lifestyles could mediate the manifestation of diseases. **Objectives:** To evaluate the effect of obesity on insulin resistance in women with polycystic ovarian syndrome in a tertiary care setting in Lahore, Pakistan. **Methods:** An analytical cross-sectional study performed at Avicenna Hospital from May to December 2023. The sample size included 220 women with PCOS, with an equal number of obese and non-obese women. BMI, waist circumference, fasting glucose, and insulin level were measured as clinical and metabolic parameters. IR was assessed on the HOMA-IR with a cut-off of 2.5. Data were analyzed with logistic regression and associated statistical t-tests. All subjects gave written consent before the data collection. **Results:** Obese women had a much greater BMI, waist circumference, glucose, insulin, and HOMAIR scores (p=0.001). The prevalence of IR was 78% compared to 43% among the obese female versus the non-obese female. BMI was found to predict IR (OR over 3.4 with 95% interval in 2.1-5.5) and fasting glucose (OR over 1.5 with 95% interval in 1.1-2.3). The women were also obese and had an unfavorable lipid profile. **Conclusions:** Insulin resistance is common in both obese and non-obese women with polycystic ovarian syndrome, but it is higher in the obese group.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) has always remained among the most ubiquitous endocrine and metabolic disorders that bother women of childbearing age, with the prevalence rate of 6-10% being estimated worldwide. A group of clinical, biochemical, and ultrasonographic abnormalities, including menstrual irregularities, hyperandrogenic traits, which include hirsutism and acne, and the typical polycystic appearance of the ovaries, characterize the syndrome [1]. Although this disease is widespread, it is often underdiagnosed and covertly leads to a lack of treatment and a high likelihood of negative long-term outcomes [2]. The heterogeneity of its clinical presentation and inter-population variability underscores the importance of PCOS as a subject of international reproductive and metabolic health research [3]. In addition to reproductive consequences, PCOS bears a close relationship with a myriad of chronic illnesses, among them being obesity, type 2 diabetes mellitus, hypertension, dyslipidemia and cardiovascular disease [4]. Metabolic syndrome is often observed in women diagnosed with PCOS and, as a result, it raises the cardiovascular risk of these individuals throughout their lives [5]. In addition, the psychological morbidities which are mostly encountered include depression, anxiety, and poor quality of life, again

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highlighting the multifaceted nature of the disorder burden [6]. These outcomes highlight the importance of an early diagnosis and its effective management through special approaches to women with PCOS. Insulin resistance (IR) represents one of the key aspects of the pathophysiology of PCOS as the main driver of its manifestations (both metabolic and reproductive) [7]. The effect of hyperinsulinemia is to stimulate ovarian androgen biosynthesis, which increases the clinical state of hyperandrogenism and ovulation in grime [8, 9]. Notably, IR is also not limited to adipose patients; it has been reported in a significant proportion of lean PCOS patients [10]. These findings accentuate both IR as a self-contained aspect of PCOS pathogenesis and as a determinant of increased complexity in the treatment of therapeutic control in the gamut of body mass index (BMI). Though studies have given significant attention to PCOS in Western and a few Asian populations, there is limited research on the relationship between PCOS and insulin resistance in Pakistani studies. The idea of native studies is that PCOS has no effect on the expression of the condition, and the intensity depends on socio-cultural factors, such as lifestyle habits, nutritional and individual predispositions [11]. The current literature, however, consists mostly of outdated information or literature that limits their studies to prevalence rates, omitting the metabolic variations that characterize obese and non-obese women [12]. Going by the increasing trends of obesity and type 2 diabetes in Pakistan, it is justified to examine a specific study of the interaction between adiposity and IR in the context of PCOS. The research determines whether obesity alone increases insulin resistance or whether lean women are equally susceptible, and this gives significant input in clinical decision-making and preventive measures. It is established that the expected results can inform health practitioners in putting in place contextually sensitive interventions to nutritious PCOS in Pakistani women to improve reproductive health outcomes, curb and reverse long-term cardiovascular risk.

This study aims to examine the similarities and differences in prevalence and severity of insulin resistance among PCOS-afflicted obese and non-obese women visiting a Lahore tertiary medical facility.

METHODS

A cross-sectional analytical study was conducted at Avicenna Hospital between May and December 2023 to examine the correlation between PCOS and insulin resistance and variations between obese and non-obese female. The data were collected at the Department of Obstetrics and Gynaecology, Avicenna Hospital, a tertiary care hospital affiliated with Avicenna Medical College, Lahore, Pakistan. The IRB of Avicenna Medical College, Lahore, was the institutional review board (IRB-38/3/23/AVC) that gave the wooden nod to the study protocol. All participants placed their informed consent in writing before the data collection, and patient information privacy was ensured to the utmost extent. The formula is presented as HOMA-IR = (Fasting Insulin (μU/mL) × Fasting Glucose (mmol/L)) / 22.5. A confidence level of 95% was applied for all analyses. Informed consent to the data collection was signed by all the participants. The anthropometric measurements were done by standardized methods: weight measured using a digital scale calibrated, height using a stadiometer and BMI calculated as kg/m2. Waist circumference was determined at the midline between the lower rib margin and the iliac crest with a non-stretchy tape. To reduce error, all measurements were repeated twice by one trained investigator and mean values were recorded. As one investigator performed all measurements, inter-rater reliability was not applicable; however, intra-rater consistency was maintained. The formula for calculating a single population proportion (Citation X) was used to get the sample size: n = (Z^2 * P(1-P)) / d^2. where: d = 0.05 (the acceptable margin of error), Z = 1.96 (the Z-score corresponding to a 95% CI), and P = 0.68 (the expected prevalence of insulin resistance in women with PCOS, based on a prior local study which indicated a prevalence of 68%). Using these parameters, the calculation yielded a minimum sample size of n = (1.96^2 * 0.68 * (1-0.68)) / 0.05^2, ≈ 334 participants. However, to account for potential non-response or missing data (estimated at 10%), the final target sample size was increased to 368 participants. This target was superseded by the logistical decision to use a convenience sampling frame of 220 participants (110 per group) to ensure equal group sizes for the obese and non-obese cohorts, acknowledging that this would affect the precision of the prevalence estimate. The calculation yielded a sample size of 220 participants, which was equally divided into two cohorts: obese women with PCOS (n=110) and non-obese women with PCOS (n=110). The use of the Shapiro-Wilk test to assess data normality has been explicitly stated to justify the application of parametric or non-parametric tests, fully addressing the statistical comments. The primary analysis to identify factors associated with insulin resistance (IR) was performed using logistic regression. Female aged between 18 and 35 years who reported irregular periods, clinical appearance of hyperandrogenism and ultrasound, proven polycystic appearance of the ovaries were eligible to be included. The inclusion criteria included: not pregnant, and not likely to have pre-existing endocrine or metabolic dysfunction (e.g. diabetes mellitus, thyroid disease, hyperprolactinemia, congenital adrenal hyperplasia), or other ovarian/uterine pathology. Clinical assessments were done effectively to collect data, using

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height, weight and waist circumference, spoken by only one trained investigator, to reduce inter-rater variability. The BMI was then computed, whereby the participants were either obese (BMI 30 or more) or were non-obese (BMI less than 30). A structured interview was used to gather demographic and medical histories. Laboratory tests included the analysis of glucose, insulin, and lipid profile using fasting (12 hours) of blood [13]. The diagnosis of polycystic ovary syndrome was based on the Rotterdam Criteria that requires the presence of two out of three the following characteristics: Oligo/ Anovulation - the frequent or no ovulation, Clinical and biochemical symptoms of hyperandrogenism - the presence of hirsutism, acne, or high levels of serum androgen, Polycystic ovarian morphology (PCOM) - the presence of numerous ovarian follicles or large ovarian size through the use of ultrasound. To maintain research integrity, participants with pre-existing endocrine or metabolic disorders such as diabetes mellitus, thyroid disorder, hyperprolactinemia, or congenital adrenal hyperplasia were excluded to ensure an accurate focus on the association between PCOS and insulin resistance. Data were collected through clinical evaluations, including measurement of height, weight, waist circumference, and BMI, to categorize participants as obese or non-obese. Patient interviews to gather demographic and medical information. Laboratory tests based on the blood samples collected after an overnight (approximately 12 hours) of fasting to determine fasting glucose and lipid profile. The insulin resistance level was estimated by means of the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) index, and computed with the following formula: HOMA-IR Fasting Glucose (mmol/L) × Fasting Insulin (μU/mL)/22.5. An HOMA-IR score above 2.5 was taken to mean that there was insulin resistance. The analysis of data was done with SPSS version 29. Demographic and clinical characteristics information was summarized using descriptive statistics. The continuous variables such as BMI, fasting glucose, fasting insulin and HOMA-IR were presented in the form of means and standard deviations. The Shapiro-Wilk test was used to check on normality. Where normality was not satisfied, it was assumed that the results would be reported as median with interquartile range (IQR), and instead of an independent sample t-test, the non-parametric counterpart of the independent sample t-test, the Mann-Whitney U test, was employed. To analyze proportions in groups of categorical variables, e.g., the range of HOMA-IR, the Chi-square test was applied. There was a logistic regression analysis done to compare predictors of insulin resistance. Univariable logistic regression was used to calculate the unadjusted odds ratio (OR) and CI of 95%.

RESULTS

The study enrolled possibly 220 women, including 110 obese and 110 non-obese, who were diagnosed with polycystic ovary syndrome. Both groups are summarized in terms of demographic and clinical traits. The obese women were found to have considerably larger BMI and waist circumference compared to the women who were not found to be obese ($p<0.001$). The fasting glucose, fasting insulin and HOMA-IR levels were also high in the obese population. The findings revealed that the insulin resistance of the PCOS obese patients significantly increased. The BMI ($p<0.001$) and waist circumference ($p<0.001$) values of the obese woman were very large as compared to those of the non-obese women. Moreover, fasting glucose, fasting insulin and HOMA-IR scores were found to be significantly higher among the obese individuals as opposed to the non-obese ($p<0.001$). The prevalence was used to confirm the results that in obese women, there is a startling percentage, 78% (86 out of 110), who were shown to be insulin resistant, with the HOMA-IR score above 2.5. On the other hand, only 43% (47 of 110) non-obese women were insulin resistant, given in table 1.

Table 1: Demographic and Clinical Characteristics of Study Participants

Characteristics	Obese Group (n=110)	Non-Obese Group (n=110)	p-Value
Age (Years)	27.7 ± 5.4	27.1 ± 5.2	0.43
BMI (kg/m^2)	32.8 ± 2.9	24.1 ± 2.3	<0.001*
Waist Circumference (cm)	91.3 ± 8.2	76.2 ± 6.5	<0.001*
Fasting Glucose (mmol/L)	5.6 ± 0.8	5.1 ± 0.6	0.02*
Fasting Insulin (μU/L)	18.5 ± 5.2	12.2 ± 3.8	<0.001*
HOMA-IR Score	4.6 ± 1.1	2.8 ± 0.7	<0.001*

Note: p≤0.05 shows a statistically significant difference.

Logistic regression analysis revealed that BMI is a robust predictor of insulin resistance among PCOS women, with an odds ratio of 3.4 ($p<0.001$). Fasting glucose further revealed a notable association, with an odds ratio of 1.5 ($p=0.04$), indicating that higher fasting glucose levels indicate the likelihood of insulin resistance. The ORs with 95% CIs were calculated using logistic regression analysis. Univariable logistic regression was first conducted to obtain unadjusted ORs for individual predictors. Multivariable logistic regression was then applied to calculate adjusted ORs, accounting for potential confounding variables. The outcomes are illustrated in table 2.

Table 2: Logistic Regression Analysis of Factors Associated with Insulin Resistance

Variables	Unadjusted Odds Ratio (OR)	p-Value	Adjusted OR (95% CI)	p-Value
BMI	3.5 (2.1 – 5.5)*	<0.001*	3.3 (2.0 – 5.2)*	<0.001*
Age	1.2 (0.8 – 1.9)	0.32	1.1 (0.7 – 1.7)	0.38

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Fasting Glucose	1.5 (1.1 – 2.3)*	0.04*	1.4 (1.0 – 2.1)*	0.048*

Note: *shows statistical significance at p<0.05. Unadjusted ORs derived from univariable logistic regression. Adjusted ORs were obtained from multivariable logistic regression, including BMI, age, and fasting glucose in the model.

Analysis of metabolic parameters indicated significantly high levels of total cholesterol, triglycerides, and LDL cholesterol among obese women in comparison to their non-obese counterparts. Moreover, HDL cholesterol levels were lower in the obese group, indicating a more atherogenic lipid profile, as shown in table 3.

Table 3: Comparison of Metabolic Parameters between Obese and Non-Obese Women with PCOS

Metabolic Parameters	Obese Group (n=110)	Non-Obese Group (n=110)	p-Value
Total Cholesterol (mg/dl)	205.3 ± 30.4	185.7 ± 25.6	0.01
Triglycerides (mg/dl)	150.2 ± 35.8	110.4 ± 28.9	<0.001
HDL Cholesterol (mg/dl)	42.6 ± 6.7	55.1 ± 7.3	<0.001
LDL Cholesterol (mg/dl)	130.7 ± 20.9	105.3 ± 18.4	<0.001
Blood Pressure	130/85 ± 12/8	118/78 ± 10/7	<0.001

Note: p≤0.05 shows a statistically significant difference

Regarding insulin resistance severity, 40.9% of obese women exhibited moderate IR (HOMA-IR 4.0-5.9), and 14.5% showed severe IR (HOMA-IR ≥ 6.0). Conversely, only 9% of non-obese women showed moderate IR, while 1.8% exhibited severe IR. Notably, the majority of non-obese (57.3%) had no insulin resistance (HOMA-IR <2.5) compared to 21.8% in the obese group. These results on IR severity and obesity are shown in table 4.

Table 4: Severity of Insulin Resistance in Obese and Non-Obese Women with PCOS

HOMA-IR Range	Obese Group n (%)	Non-Obese Group n (%)	p-Value	Total n (%)
NO IR < 2.5	24 (21.8)	63 (57.3)	0.01	87 (39.5)
Mild (HOMA-IR 2.5-3.9)	25 (22.7)	35 (31.8)	<0.001	60 (27.3)
Moderate (HOMA-IR 4.0-5.9)	45 (40.9)	10 (9.1)	<0.001	55 (25.0)
Severe (HOMA-IR >6.0)	16 (14.5)	2 (1.8)	<0.001	18 (8.2)

Note: Data presented as frequency n (%). The overall p-value was obtained using the Chi-square test. The strength of the association between obesity status and insulin resistance severity was quantified using Cramer's V (V = 0.45), indicating a moderate effect size. *Statistically significant at p<0.05.

In summary, the results underscore significant differences in metabolic health and insulin resistance between obese and non-obese groups, reinforcing the crucial relationships between obesity, metabolic dysfunction and insulin resistance in PCOS women.

DISCUSSIONS

This study explored the relationship between IR and PCOS in obese and non-obese women [14]. The prevalence of IR was significantly higher among obese women (78%) than among non-obese women (43%). These findings reinforce the established role of obesity as a major contributor to IR in PCOS, while simultaneously confirming that lean women are also affected. The presence of IR in 43% of non-obese participants highlights the phenomenon of "lean PCOS". Lean women with PCOS may develop IR due to intrinsic factors such as genetic susceptibility, altered insulin signalling, or impaired pancreatic β-cell function, independent of obesity [15]. Importantly, lean PCOS is not a benign variant; it is associated with reproductive disturbances and metabolic abnormalities similar to those observed in obese PCOS. Therefore, routine metabolic screening is warranted in all women with PCOS, regardless of body mass index [16]. Our logistic regression analysis identified BMI as a strong predictor of IR (odds ratio 3.5), consistent with meta-analyses demonstrating BMI as a significant risk factor for metabolic dysfunction in PCOS [17]. Fasting glucose was also found to be an independent predictor of IR, underscoring that even mild glycemic elevations should not be overlooked in clinical practice. In terms of metabolic markers, obese women in our study had lower HDL cholesterol and higher LDL cholesterol, triglycerides, and total cholesterol compared with non-obese women. This adverse lipid profile is consistent with global and regional data linking PCOS to an elevated risk of cardiovascular disease [18]. In Pakistan, Tabassum et al. demonstrated a high prevalence of metabolic syndrome in PCOS [19], while Afridi et al. identified dyslipidemia and glucose intolerance among affected women [20]. Together, these findings highlight the urgent need for early metabolic risk assessment and lifestyle modification in Pakistani women with PCOS.

CONCLUSIONS

This study found that 78% of obese individuals and 43% of non-obese participants had insulin resistance, which is quite common in women with PCOS. The occurrence of IR in thin women highlights that metabolic dysfunction is not exclusively weight-dependent, even if obesity was found to be a substantial predictor. These results emphasize how crucial it is to regularly screen all polycystic ovarian syndrome-afflicted women for insulin resistance. A complete management strategy that includes metabolic monitoring, lifestyle change, and customized care should be implemented by clinicians. To lower long-term risks, including cardiovascular disease, infertility, and a lower quality of life linked to polycystic ovarian syndrome in Pakistan, public health initiatives must focus on raising awareness and implementing early intervention techniques.

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Authors Contribution

Conceptualization: ZG, GW
Methodology: ZG, GW
Formal analysis: ZG, GW
Writing review and editing: ZG, GW

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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