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ORIGINAL ARTICLE

Assessment of the Relationship Between Polycystic Ovary Syndrome and Insulin Resistance in Adolescent Females

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders among adolescent females, characterized by hyperandrogenism, menstrual irregularity, and polycystic ovarian morphology.

Objective: To assess the relationship between polycystic ovary syndrome and insulin resistance among adolescent females.

Methodology: This cross-sectional multicentered study was conducted at Sir Ganga Ram Hospital, Lahore and Sahiwal Teaching Hospital, Sahiwal from December 2022 to June 2023. 255 adolescent females aged 13–19 years diagnosed with PCOS according to the modified Rotterdam criteria. Data were collected through detailed history, physical examination, and biochemical analysis. Fasting glucose and fasting insulin levels were measured, and insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR).

Results: The mean age of participants was 16.8 ± 1.7 years, and the mean BMI was 27.6 ± 4.3 kg/m². Menstrual irregularity (85.1%) and acne (71.0%) were the most frequent clinical findings. The mean fasting glucose was 92.8 ± 9.4 mg/dL, fasting insulin was 17.5 ± 6.2 µU/mL, and mean HOMA-IR was 3.9 ± 1.4. Insulin resistance (HOMA-IR > 2.5) was present in 67.8% of participants. A significant association was observed between insulin resistance and BMI ($p < 0.001$), acanthosis nigricans ($p < 0.001$), menstrual irregularity ($p = 0.002$), and elevated testosterone levels ($p = 0.004$). A positive correlation was also noted between HOMA-IR and BMI ($r = 0.54$) as well as fasting insulin ($r = 0.72$).

Conclusion: It is concluded that insulin resistance is highly prevalent among adolescent females with PCOS and is significantly associated with both metabolic and reproductive abnormalities. Since IR can occur even in non-obese individuals, routine screening should be performed in all adolescents with PCOS.

Keywords: Polycystic ovary syndrome, Insulin resistance, Adolescents, HOMA-IR, Hyperandrogenism.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) represents a multifaceted endocrinological and metabolic condition that affects approximately 6–15% of women of reproductive age, with evidence suggesting that its onset may begin during adolescence[1]. More young females recognized with PCOS has created concern about the condition's early metabolic and psychological effects. Adolescence is sensitive period developmentally with important hormonal changes, including gonadotropins and insulin sensitivity which may hide or intensify PCOS symptoms. To avoid metabolic dysfunction and compromised reproductive health later, early treatment and diagnosis in these years is necessary[2]. The development of PCOS has many intertwining and interacting components, notably genetic, environmental and lifestyle factors. One of the most critical of these components is insulin resistance (IR) which is central in the development of the metabolic and reproductive sequelae of PCOS[3]. In this situation, IR connotes a reduction of the biological action of insulin and, therefore, results in compensatory hyperinsulinemia. This excess insulin is a key factor of ovarian hyperandrogenism since insulin, along with luteinizing hormone (LH), stimulates theca cell androgen production[4]. Furthermore, hyperinsulinemia inhibits the liver's production of sex hormone binding globulin (SHBG) and results in free testosterone which aggravates symptoms of PCOS[5]. Diagnosing PCOS in adolescents is difficult owing to features that overlap with normal pubertal physiology such as temporary irregularities in menstruation and the development of acne[6]. This highlights the importance of using evidence-based diagnosing criteria. The National Institutes of Health (NIH) and the Rotterdam criteria have been the benchmark although adaptations are necessary in the younger population[7]. The diagnostic framework that is most reliable in adolescents is the persistence of hyperandrogenism (either clinical or biochemical) and menstrual irregularity for more than two years post-menarche with other potential causes excluded. There is new evidence that suggests insulin resistance may occur before the full-blown picture of PCOS develops[8]. This is most likely in individuals who have a family history of the metabolic syndrome and type 2 diabetes mellitus[9]. Compared with age- and BMI-matched control without PCOS, adolescent girls with PCOS have been documented to have higher fasting insulin levels, elevated HOMA-IR scores, and glucose intolerance[10]. This is not to say however that obesity is the sole contributor to insulin resistance as even in adolescents who are not obese, insulin resistance is a significantly prevalent condition[11]. This reinforces the need for metabolic screening to all adolescents who have clinical features of PCOS and not only those who are overweight or obese[12]. Insulin resistance in PCOS entails more than just the dysregulation of glucose metabolism. It also fuels the development of an atherogenic lipid profile, potentiation of inflammation, and endothelial dysfunction, factors that act synergistically to heighten the risk of developing cardiovascular disease[13]. Hyperinsulinemia is also associated with an increase in appetite, thereby accreting more body fat, and exacerbating the insulin resistance that is already present. The combination of insulin resistance and hyperinsulinemia in adolescents with PCOS is a considerable risk factor for the development of cardiovascular disease, type 2 diabetes, and metabolic syndrome in later life[14].

Objective: To assess the relationship between polycystic ovary syndrome and insulin resistance among adolescent females.

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METHODOLOGY

This was a cross-sectional multicentered study conducted at Sir Ganga Ram Hospital, Lahore and Sahiwal Teaching Hospital, Sahiwal from December 2022 to June 2023. In this study, a total of 255 adolescent females participated. The study took a non-probability consecutive sampling approach. Participants falling between the age of 13 to 19 and female adolescents, diagnosed with PCOS based on modified Rotterdam criteria, were considered. The diagnosis needed to fulfill at least two of the

following criteria: oligo/anovulation (irregularity of the menstrual cycle), clinical or biochemical hyperandrogenism, and/or polycystic ovary morphology on ultrasound. Recruitment considered only those participants eligible where two or more years had lapsed since menarche was attained. Known endocrine disorder (e.g., Cushing's syndrome, thyroid disorders, congenital adrenal hyperplasia, diabetes mellitus) was a disqualifier. Also, disqualification was made for those using hormonal contraceptives, corticosteroids, insulin sensitizers, or other medicinal agents that modulate the glucose or insulin axis in the last three months.

Data Collection: After obtaining informed consent from each participant and her guardian, detailed demographic and clinical information was collected through a structured questionnaire. The questionnaire covered age, menstrual history, duration of symptoms, and family history of metabolic or reproductive disorders. Physical examination included measurements of height, weight, and calculation of body mass index (BMI). Clinical signs of hyperandrogenism such as acne, hirsutism, and acanthosis nigricans were recorded. Venous blood samples were collected after an overnight fast. Fasting plasma glucose and fasting insulin levels were analyzed in a certified laboratory using standard enzymatic and immunoassay techniques. The degree of insulin resistance was determined using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) formula:

HOMA-IR = (Fasting Insulin [μU/mL] × Fasting Glucose [mg/dL]) / 405

A HOMA-IR value greater than 2.5 was considered indicative of insulin resistance.

Data Analysis: Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 21. Quantitative variables such as age, BMI, fasting glucose, fasting insulin, and HOMA-IR were presented as mean ± standard deviation (SD). Categorical variables such as presence of menstrual irregularity, acne, and hirsutism were presented as frequencies and percentages. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

Data were collected from 255 patients, mean age was 16.8 ± 1.7 years, indicating that most participants were in the mid to late adolescent range. The average body mass index (BMI) was 27.6 ± 4.3 kg/m², with 151 participants (59.2%) classified as overweight or obese. Menstrual irregularity was the most common clinical feature, seen in 217 participants (85.1%). Acne and hirsutism were also frequent, reported in 181 (71.0%) and 164 (64.3%) participants respectively. Acanthosis nigricans, an external sign of insulin resistance, was found in 119 participants (46.7%). A positive family history of diabetes or metabolic syndrome was present in 101 participants (39.6%).

The mean fasting plasma glucose level was 92.8 ± 9.4 mg/dL, while the mean fasting insulin level was 17.5 ± 6.2 μU/mL. The average HOMA-IR value was 3.9 ± 1.4, indicating that a large proportion of adolescents had insulin resistance. A total of 173 participants (67.8%) had insulin resistance (HOMA-IR > 2.5), while 82 participants (32.2%) had normal insulin sensitivity.

The mean BMI was significantly higher in insulin-resistant participants (29.1 ± 3.8 kg/m²) compared to those without resistance (24.7 ± 2.9 kg/m², $p < 0.001$). Menstrual irregularity was more frequent in the insulin-resistant group (90.8%) than in the non-resistant group (73.1%, $p = 0.002$). Acne was also slightly more common among insulin-resistant participants (75.1% vs 62.2%, $p = 0.048$), whereas hirsutism did not differ significantly between groups ($p = 0.09$). Acanthosis nigricans was strongly associated with insulin resistance, being present in 61.3% of insulin-resistant participants compared to only 18.3% of those with normal insulin sensitivity ($p < 0.001$). Fasting glucose, fasting insulin, and HOMA-IR levels were all significantly higher in the insulin-resistant group ($p < 0.001$ for all).

Insulin resistance was found in 54.3% of normal-weight participants, 73.1% of overweight participants, and 89.5% of obese participants, showing a significant upward trend with increasing BMI ($p < 0.001$). Participants with a family history of diabetes had a higher prevalence of insulin resistance (78.4%) compared to those without a family history (61.1%, $p = 0.01$).

A strong positive correlation was observed between HOMA-IR and fasting insulin ($r = 0.72$, $p < 0.001$), followed by BMI ($r = 0.54$, $p < 0.001$). Fasting glucose ($r = 0.39$, $p < 0.001$) and total testosterone ($r = 0.29$, $p = 0.004$) also showed significant positive correlations with HOMA-IR.

Table 1: Baseline Demographic and Clinical Characteristics of Adolescent Females with PCOS (n = 255)

Variable	Mean ± SD / n (%)
Age (years)	16.8 ± 1.7
Body Mass Index (BMI, kg/m²)	27.6 ± 4.3
Overweight/Obese	151 (59.2%)
Menstrual irregularity	217 (85.1%)
Acne	181 (71.0%)
Hirsutism	164 (64.3%)
Acanthosis nigricans	119 (46.7%)
Family history of diabetes/metabolic syndrome	101 (39.6%)

Table 2: Biochemical Parameters of Study Participants (n = 255)

Parameter	Mean ± SD
Fasting plasma glucose (mg/dL)	92.8 ± 9.4
Fasting insulin (μU/mL)	17.5 ± 6.2
HOMA-IR	3.9 ± 1.4
Participants with insulin resistance (HOMA-IR > 2.5)	173 (67.8%)
Participants with normal insulin sensitivity	82 (32.2%)

Table 3: Comparison of Clinical and Biochemical Features Between Participants with and Without Insulin Resistance

Parameter	Insulin Resistance (n = 173)	No Insulin Resistance (n = 82)	p-value
Age (years)	16.9 ± 1.6	16.6 ± 1.8	0.34
BMI (kg/m²)	29.1 ± 3.8	24.7 ± 2.9	<0.001*
Menstrual irregularity	157 (90.8%)	60 (73.1%)	0.002*
Acne	130 (75.1%)	51 (62.2%)	0.048*
Hirsutism	118 (68.2%)	46 (56.1%)	0.09
Acanthosis nigricans	106 (61.3%)	15 (18.3%)	<0.001*
Fasting glucose (mg/dL)	94.6 ± 8.9	89.1 ± 7.4	<0.001*
Fasting insulin (μU/mL)	19.8 ± 5.9	12.1 ± 4.2	<0.001*
HOMA-IR	4.7 ± 1.3	2.0 ± 0.4	<0.001*
Total testosterone (ng/mL)	0.92 ± 0.24	0.66 ± 0.21	0.004*

*Statistically significant ($p \leq 0.05$)

Table 4: Stratified Analysis of Insulin Resistance According to BMI and Family History of Diabetes

Variable	Total (n)	Insulin Resistance n (%)	p-value
BMI Category			
Normal weight	81	44 (54.3%)	
Overweight	93	68 (73.1%)	
Obese	81	72 (89.5%)	<0.001*
Family History of Diabetes			
Present	101	79 (78.4%)	
Absent	154	94 (61.1%)	0.01*

*Statistically significant ($p \leq 0.05$)

Table 5: Correlation Between HOMA-IR and Clinical/Biochemical Variables

Variable	Correlation Coefficient (r)	p-value
BMI (kg/m²)	0.54	<0.001*
Fasting insulin (μU/mL)	0.72	<0.001*
Fasting glucose (mg/dL)	0.39	<0.001*
Total testosterone (ng/mL)	0.29	0.004*

*Statistically significant ($p \leq 0.05$)

DISCUSSION

This study investigated the relationship between polycystic ovary syndrome (PCOS) and insulin resistance (IR) among adolescent females and demonstrated a strong association between the two conditions. According to HOMA-IR value metrics, 67.8% of adolescents diagnosed with PCOS also have insulin resistance. The early metabolic risk associated with PCOS, even among

adolescents, is serious, and routine metabolic screening should be performed on young females with presentations of the disorder that are reproductive or dermatologic. This study, along with countless others, confirms that insulin resistance is one of the most significant components of PCOS's clinical manifestations. In the case of PCOS, insulin resistance fuels permanent hyperinsulinemia, increased androgen production, and the clinical manifestations of androgen excess through the suppression of sex hormone binding globulin (SHBG), such as hirsutism, acne, and menstrual irregularities[15]. In the current study, menstrual irregularity and acne were observed to be predominantly present among insulin-resistant individuals, demonstrating the relationship between metabolic dysregulation and the reproductive manifestations of PCOS. HOMA-IR value (3.9 ± 1.4) in this group of adolescents was also similar to other adolescent literature. This confirms that insulin resistance begins early with PCOS and sometimes even in the absence of obesity. Although obesity was significantly associated with insulin resistance ($p < 0.001$), more than half of the normal-weight participants also demonstrated IR[16]. This aligns with the evidence that the insulin resistance in PCOS is likely to include some intrinsic genetic or molecular component rather than solely an effect related to obesity. The presence of intrinsic post-receptor insulin signaling pathway anomalies and the excess visceral fat of PCOS are proposed explanations for the insulin insensitivity and resistance in the disorder irrespective of the obesity component. The study also demonstrated a strong positive correlation of HOMA-IR and BMI ($r = 0.54$) and an even stronger HOMA-IR correlation with fasting insulin ($r = 0.72$). These observations endorse the use of simple fasting criteria to identify early metabolic disturbances in low-resourced contexts where advanced testing methods like the euglycemic clamp are unfeasible[17]. Clinical evidence further supports this by the highly significant association of acanthosis inus and insulin resistance ($p < 0.001$). This highlights the potential importance of this easily identifiable skin marker in clinical practice and in the screening of adolescents with PCOS for hyperinsulinemia[18]. The situation where screening and near relatives of the patients are clinically more likely to be insulin resistant (78.4% vs. 61.1%, $p = 0.01$) corresponds to the connection of insulin resistance, along with the genetic and family- clustering of Weak PCOS. This insight emphasizes the value of gathering a thorough family history in the assessment of adolescents with probable PCOS[19]. In this study, clinically and biochemically manifested hyperandrogenism was associated with insulin resistance. In this research, the insulin-resistant group had higher total testosterone than the non-resistant group. It demonstrates the driving the hyperinsulinemic state, facilitating the ovarian theca cells to produce androgens and escalates the steroidogenesis activated by LH[20]. These mechanisms sustain the hyperandrogenic environment, disrupting the ovulatory cycles and exacerbating PCOS symptoms. These findings point to significant clinical consequences, considering the fact that PCOS, along with the early, possibly undetected, insulin resistance in adolescents, escalates the risk of type 2 diabetes, dyslipidemia, metabolic syndrome, and cardiovascular diseases in the child during adulthood. Management of IR is improved with early recognition allowing for lifestyle changes and initiation of medication such as metformin, which is effective for metabolic and reproductive issues as well. Since many adolescents might first present with cosmetic and menstrual issues, and even when patients are non-obese, clinicians must remain highly vigilant for possible insulin resistance[21].

Limitations: This study has certain limitations. Being cross-sectional in design, it could not establish a causal relationship between PCOS and insulin resistance. The reliance on HOMA-IR as the sole indicator of IR, while practical, may not fully capture dynamic insulin sensitivity. Additionally, the study was hospital-based, which may limit generalizability to the broader adolescent population. Despite these limitations, the relatively large sample size and inclusion of both obese and non-obese participants enhance the reliability of the findings.

CONCLUSION

It is concluded that insulin resistance is highly prevalent among adolescent females with polycystic ovary syndrome (PCOS), affecting nearly two-thirds of the study population. This finding establishes insulin resistance as a core component of PCOS pathophysiology, evident even in non-obese adolescents, thereby emphasizing that metabolic disturbances in PCOS are not solely dependent on body weight. The significant association of insulin resistance with menstrual irregularities, acne, acanthosis nigricans, and elevated androgen levels reflects its multifactorial impact on both reproductive and metabolic health. Early detection and management of insulin resistance in adolescents with PCOS are essential to prevent long-term complications such as type 2 diabetes mellitus, dyslipidemia, and cardiovascular disease. Routine metabolic screening, including assessment of BMI, fasting insulin, and HOMA-IR, should be integrated into clinical evaluation protocols for all adolescent PCOS patients.

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