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Association of Insulin Resistance and Elevated Androgen Levels in Patients with Polycystic Ovarian Syndrome (PCOS)

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ABSTRACT

Background: *Polycystic ovary syndrome (PCOS) is a common endocrine disorder in reproductive-aged women characterized by hyperandrogenism, ovulatory dysfunction, and insulin resistance. Insulin resistance and elevated androgen levels are closely interrelated in PCOS, though their association varies across populations.* ***Objective:*** *The aim of the study was to* determine the association between insulin resistance and elevated androgen levels in patients with polycystic ovarian syndrome (PCOS). ***Methods & Materials:*** *This cross-sectional observational study was conducted at the Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, from January to December 2024, including 110 women with PCOS selected by purposive sampling. After excluding other endocrine disorders and hormonal therapy, BMI and fasting insulin, glucose, and testosterone were measured; insulin resistance was assessed using HOMA-IR and elevated androgens by laboratory reference ranges. Statistical analysis was performed with $p < 0.05$ considered significant.* ***Results:*** *The study included 110 women with PCOS, with most aged 21–30 years (61.8%) and a mean age of 26.5 ± 7.2 years. Overweight/obesity was present in 68.2% of participants. Insulin resistance and elevated androgen levels were observed in 65.5% and 70.9% of patients, respectively. A significant association was found between insulin resistance and elevated androgen levels ($p < 0.001$), with higher fasting insulin, HOMA-IR, and total testosterone in insulin-resistant patients ($p < 0.001$).* ***Conclusion:*** *Insulin resistance is significantly associated with elevated androgen levels in women with polycystic ovarian syndrome, highlighting a strong interrelated metabolic–endocrine link in its pathophysiology.*

Keywords: *Insulin Resistance, Elevated Androgen Levels, Polycystic Ovarian Syndrome*

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is widely recognized as one of the most common endocrine disorders affecting women of reproductive age and is a major cause of menstrual irregularities, anovulatory infertility, and clinical hyperandrogenism [1]. It is a heterogeneous condition characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, often accompanied by metabolic disturbances such as insulin resistance (IR) and obesity. Clinical manifestations frequently include menstrual disturbances, features of androgen excess, and increased body weight, reflecting the complex nature of the disorder [2].

Depending on the criteria applied, PCOS is considered the most prevalent endocrine disorder among women of reproductive age, affecting approximately 6–22% of women worldwide [3]. Current evidence suggests that its global prevalence ranges from 6 to 21%, varying according to diagnostic definitions [4-6]. During the reproductive years, the occurrence of PCOS is estimated to be around 6–10%, with some reports indicating that this prevalence may be even higher [7–10].

Insulin resistance is regarded as a central pathophysiological factor in PCOS. IR and compensatory hyperinsulinaemia (HI) play a crucial role in the underlying mechanisms of the disorder and contribute significantly to the development of hyperandrogenaemia and reproductive abnormalities through multiple pathways [11]. Furthermore, a large proportion of women with PCOS exhibit insulin resistance, which subsequently enhances androgen secretion while reducing sex hormone-binding globulin levels [12–15].

Insulin can directly stimulate ovarian theca cells and enhance luteinizing hormone (LH)-mediated androgen production, leading to elevated circulating androgen levels. Consequently, insulin resistance contributes to hyperandrogenism through several mechanisms, while androgen excess can further influence adipose tissue function, inflammatory processes, and lipid metabolism, thereby establishing a self-perpetuating cycle [16]. Additionally, insulin resistance leads to hyperinsulinemia, which promotes increased androgen production and ovarian dysfunction in women with PCOS [17]. At the same time, the hormonal and metabolic disturbances

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characteristic of PCOS may further aggravate insulin resistance, reinforcing this cyclical relationship [18].

Although numerous studies have demonstrated a link between insulin resistance and androgen excess, the strength and consistency of this association vary depending on study design and population characteristics [19]. Local factors such as obesity patterns, dietary habits, and accessibility to healthcare services may also influence the observed relationship in real-world settings. Therefore, conducting a dedicated cross-sectional study in a clinically defined PCOS population is important to better understand the metabolic–reproductive interaction and to inform integrated management approaches [20].

Understanding the interplay between metabolic and endocrine abnormalities in polycystic ovary syndrome (PCOS) is essential for improving clinical management and reducing long-term complications. Insulin resistance and elevated androgen levels are key features of PCOS that contribute to reproductive dysfunction, metabolic disturbances, and increased risk of chronic diseases, yet their exact relationship remains variable across different populations and clinical settings. Moreover, variations in diagnostic criteria, biochemical markers, and population characteristics highlight the need for focused evaluation within specific clinical contexts. A clearer understanding of this association may aid in early identification of high-risk patients and support the development of targeted therapeutic strategies, including lifestyle modification and pharmacological interventions. Therefore, this study aims to determine the association between insulin resistance and elevated androgen levels in patients with PCOS.

OBJECTIVE

To determine the association between insulin resistance and elevated androgen levels in patients with polycystic ovarian syndrome (PCOS).

METHODS & MATERIALS

This cross-sectional observational study was conducted at the Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, from January to December 2024. A total of 110 women diagnosed with polycystic ovarian syndrome (PCOS) were included in the study, selected according to predefined inclusion and exclusion criteria. Patients attending both outpatient and inpatient departments were enrolled to evaluate the association between insulin resistance and elevated androgen levels in PCOS.

Participants were selected using a purposive sampling technique. The diagnosis of PCOS was established based on standard clinical, biochemical, and/or ultrasonographic criteria. Women of reproductive age with confirmed PCOS were included, while those with known endocrine disorders such as thyroid dysfunction, hyperprolactinemia, Cushing's syndrome, diabetes mellitus, or those receiving hormonal therapy were excluded to avoid confounding effects on metabolic and hormonal parameters.

Detailed demographic and clinical information, including age and relevant medical history, was collected using a structured questionnaire. Anthropometric measurements were obtained following standard procedures; height and weight were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Based on BMI values, participants were categorized into normal and overweight/obese groups for analysis.

Fasting venous blood samples were collected after an overnight fast of at least 8–10 hours. Fasting insulin and fasting glucose levels were measured using standard laboratory methods. Insulin resistance was assessed using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated as fasting insulin (μIU/mL) × fasting glucose (mmol/L) / 22.5. A predetermined cutoff value was applied to classify participants into insulin resistance present and absent groups. Serum total testosterone levels were measured to evaluate androgen status, and participants were categorized as having elevated androgen levels or normal levels based on standard laboratory reference ranges.

All data were checked for completeness, coded, and analyzed using appropriate statistical software. Continuous variables such as age and biochemical parameters were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage. The association between insulin resistance and elevated androgen levels was analyzed using the chi-square test. Independent sample t-test was applied to compare mean biochemical parameters between insulin resistance groups. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table I presents the age distribution of the study participants. The majority of patients were in the age group of 21–30 years, comprising 68 patients (61.8%). This was followed by 30 patients (27.3%) in the >30 years age group, while 12 patients (10.9%) were aged ≤20 years. The overall mean age of the study population was 26.5 ± 7.2 years.

Table I: Age Distribution of the Study Participants (*n* = 110)

Age (years)	Frequency (n)	Percentage (%)
≤20	12	10.9
21–30	68	61.8
>30	30	27.3
Mean ± SD	26.5 ± 7.2	

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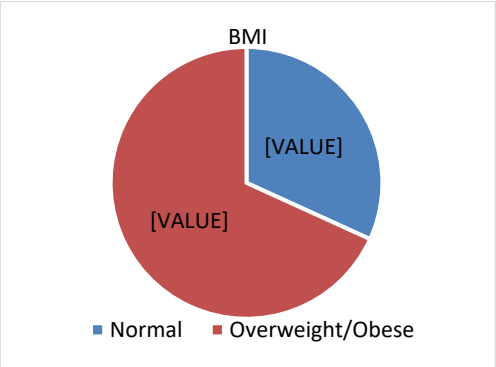

Figure I: Body Mass Index (BMI) Distribution of the Study Participants (*n* = 110)

Figure I illustrates the BMI distribution of the study participants. A higher proportion of patients were found to be overweight or obese, accounting for 75 patients (68.2%), while 35 patients (31.8%) had normal BMI.

Table II shows the prevalence of insulin resistance and elevated androgen levels among the study participants. Insulin resistance was present in 72 patients (65.5%), while 38 patients (34.5%) were insulin resistance negative. Elevated androgen levels were observed in 78 patients (70.9%), whereas 32 patients (29.1%) had normal androgen levels.

Table II: Prevalence of Insulin Resistance and Elevated Androgen Levels in the Study Population (*n* = 110)

Variable	Status	Frequency (n)	Percentage (%)
Insulin Resistance	Present	72	65.5
	Absent	38	34.5
Elevated Androgen Levels	Present	78	70.9
	Absent	32	29.1

Table III demonstrates the association between insulin resistance and elevated androgen levels among PCOS patients. A significantly higher proportion of elevated androgen levels was observed in patients with insulin resistance (60/72, 83.3%) compared to those without insulin resistance (18/38, 47.4%). The association was statistically significant ($p < 0.001$).

Table III: Association Between Insulin Resistance and Elevated Androgen Levels in PCOS Patients (*n* = 110)

Insulin Resistance	Elevated Androgen Present	Elevated Androgen Absent	Total	p-value
Present	60	12	72	<0.001
Absent	18	20	38	
Total	78	32	110	

Table IV compares biochemical parameters between patients with and without insulin resistance. Fasting insulin, HOMA-IR, and total testosterone levels were significantly higher in patients with insulin resistance compared to those without insulin resistance. The differences were statistically significant for all parameters ($p < 0.001$).

Table IV: Comparison of Biochemical Parameters According to Insulin Resistance Status (*n* = 110)

Parameters	Insulin Resistance Present (n=72) Mean±SD	Insulin Resistance Absent (n=38) Mean±SD	p-value
Fasting insulin (µIU/mL)	18.6 ± 6.2	9.4 ± 3.1	<0.001
HOMA-IR	4.8 ± 1.9	2.1 ± 0.8	<0.001
Total testosterone (ng/dL)	62.5 ± 18.3	41.2 ± 12.6	<0.001

DISCUSSION

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder among women of reproductive age and is frequently associated with a spectrum of reproductive and metabolic abnormalities, including insulin resistance and elevated androgen levels. Insulin resistance plays a central role in the pathophysiology of PCOS and contributes to increased ovarian androgen production, thereby exacerbating hormonal imbalance and clinical manifestations. The findings of this study demonstrate a high prevalence of both insulin resistance and elevated androgen levels, along with a significant association between these two factors. Furthermore, women with insulin resistance exhibited markedly higher levels of fasting insulin, HOMA-IR, and total testosterone, reflecting a pronounced metabolic–endocrine disturbance. These observations highlight the clinical importance of evaluating insulin resistance in PCOS patients, as it is closely linked with elevated androgen levels and may have important implications for early diagnosis, risk stratification, and targeted management.

The age distribution of the study participants shows that the majority of PCOS patients were in the 21–30 years age group (61.8%), followed by those aged >30 years (27.3%) and ≤20 years (10.9%), with a mean age of 26.5 ± 7.2 years. This pattern indicates that PCOS predominantly affects women in the young reproductive age group, particularly during their

twenties, which represents a critical period for reproductive health assessment and endocrine evaluation. Similar findings have been reported by Shukla et al., who described PCOS as the most common endocrine disorder among reproductive-aged women, typically diagnosed in the 20s–30s, emphasizing its peak occurrence during early reproductive life [21]. Likewise, Sharma et al. observed a mean age of 19.8 ± 1.9 years with most participants clustered between 18–25 years, further supporting the higher prevalence of PCOS among young adult females [22]. The consistency of these findings across studies suggests that PCOS is predominantly identified during early reproductive years, which may be attributed to the onset of menstrual irregularities, infertility concerns, and clinical manifestations of elevated androgen levels that prompt earlier medical evaluation in this age group.

A higher proportion of the study participants were found to be overweight or obese (68.2%), while only 31.8% had a normal BMI, indicating that increased body weight is a common characteristic among women with PCOS in the present cohort. This pattern reflects the well-established association between polycystic ovarian syndrome and increased BMI, where excess adiposity is frequently observed alongside metabolic and endocrine disturbances. A similar observation was reported by Sam et al., who noted that the prevalence of overweight and obesity among women with PCOS may be as high as 80% in some populations, particularly in Western cohorts [23]. The comparatively high proportion of overweight/obese individuals in the current study therefore aligns closely with existing evidence, reinforcing the concept that PCOS is strongly associated with increased body mass index and that obesity represents a frequent clinical phenotype in affected women, potentially contributing to both insulin resistance and androgen excess.

The present study demonstrated a high prevalence of insulin resistance (65.5%) and elevated androgen levels (70.9%) among women with PCOS, indicating that both metabolic and endocrine abnormalities are common and interrelated features in this population. These findings are consistent with previous epidemiological evidence suggesting a strong association between PCOS and insulin resistance. Su et al. reported that approximately 50–70% of PCOS cases are associated with insulin resistance, highlighting its frequent occurrence in affected women [24]. Similarly, Naeem et al. observed that insulin resistance may be present in 35–80% of women with PCOS, depending on the population and diagnostic criteria used [25]. The comparatively high prevalence of insulin resistance in the present study falls within this reported range, reinforcing its central role in the pathophysiology of PCOS. In addition, the high proportion of elevated androgen levels further supports the well-established relationship between insulin resistance and elevated androgen, where hyperinsulinemia contributes to increased ovarian androgen production. Overall, the findings of the present study are in agreement with existing literature and emphasize that insulin resistance and elevated androgen are highly prevalent and closely interrelated abnormalities in women with PCOS, underscoring a strong and clinically significant association between insulin resistance and elevated androgen levels in PCOS.

The present study demonstrated a statistically significant association between insulin resistance and elevated androgen levels among women with PCOS ($p < 0.001$), where a markedly higher proportion of elevated androgen levels was observed in patients with insulin resistance (60/72, 83.3%) compared to those without insulin resistance (18/38, 47.4%). This finding indicates a strong interrelationship between metabolic dysfunction and endocrine imbalance in PCOS. Similar observations have been reported by Yang et al., who described a bidirectional association between insulin resistance and elevated androgen levels, showing that higher testosterone levels were significantly more common in insulin-resistant PCOS patients, along with significantly increased insulin resistance markers such as HOMA-IR and fasting insulin in hyperandrogenic groups [26]. Likewise, Wang et al. reported a strong positive correlation between testosterone and HOMA-IR in hyperandrogenic PCOS phenotypes and further demonstrated that insulin resistance independently predicted elevated androgen levels even after adjustment for BMI [27]. These findings collectively reinforce the close bidirectional relationship between insulin resistance and elevated androgen levels in PCOS, suggesting that each condition may contribute to the development and progression of the other.

The present study also demonstrated significantly higher levels of fasting insulin, HOMA-IR, and total testosterone among women with insulin resistance compared to those without insulin resistance ($p < 0.001$ for all parameters), indicating a marked metabolic and endocrine disparity between the two groups. These results suggest that insulin resistance is associated not only with impaired glucose–insulin homeostasis but also with increased androgen production in women with PCOS. Similar observations have been reported by Wiweko et al., who found that the severe hyperandrogenic phenotype was associated with significantly higher insulin resistance indices across different PCOS groups, highlighting a close relationship between androgen excess and metabolic dysfunction [28]. Likewise, Li et al. reported a positive correlation between insulin resistance and intra-ovarian androgen levels and further noted that insulin resistance may be present even in some women without elevated serum testosterone, suggesting a complex and multifactorial interaction between insulin signaling pathways and androgen production [29]. Overall, the findings of the present study are consistent with existing literature and reinforce that women with insulin resistance exhibit significantly higher androgen levels and insulin resistance indices, underscoring the interconnected metabolic–endocrine nature of PCOS and highlighting the strong pathophysiological linkage between insulin resistance and elevated androgen levels.

LIMITATIONS OF THE STUDY

The study was conducted at a single center, so the results may limit the generalizability of the findings to other hospitals or regions in Bangladesh.

The small sample size limits the generalizability of the findings.

CONCLUSION

The present study was conducted to evaluate the relationship between metabolic and endocrine disturbances in women with polycystic ovarian syndrome. A significant association was observed between insulin resistance and elevated androgen levels in the study population. Elevated androgen levels were more frequently seen in patients with insulin resistance, and biochemical parameters such as fasting insulin, HOMA-IR, and total testosterone were also significantly higher in this group. These findings indicate a strong interrelationship between insulin resistance and elevated androgen levels in PCOS, suggesting that both abnormalities are closely linked in its pathophysiology.

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CONFLICTS OF INTEREST
There are no conflicts of interest.

REFERENCES

1. *Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. International evidence-based guideline for the assessment and management of polycystic ovary syndrome. Hum Reprod. 2019;34(2):388-404.*
2. *Hosain AS, Mehedi MH, Kabir IE. Pconet: A convolutional neural network architecture to detect polycystic ovary syndrome (pcos) from ovarian ultrasound images. In2022 International conference on engineering and emerging technologies (ICEET) 2022 Oct 27 (pp. 1-6). IEEE.*
3. *Li J, Wu Q, Wu XK, Zhou ZM, Fu P, Chen XH, Yan Y, Wang X, Yang ZW, Li WL, Stener-Victorin E. Effect of exposure to second-hand smoke from husbands on biochemical hyperandrogenism, metabolic syndrome and conception rates in women with polycystic ovary syndrome undergoing ovulation induction. Human reproduction. 2018 Apr 1;33(4):617-25.*
4. *The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop groxup. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod. Hum Reprod. 2004;19(1):41–7.*
5. *Dapas M, Lin FT, Nadkarni GN, Sisk R, Legro RS, Urbanek M, Hayes MG, Dunaif A. Distinct subtypes of polycystic ovary syndrome with novel genetic associations: an unsupervised, phenotypic clustering analysis. PLoS medicine. 2020 Jun 23;17(6):e1003132.*
6. *Bozdag G, Mumusoglu S, Zengin D, Karabulut E, Yildiz BO. The prevalence and phenotypic features of polycystic ovary syndrome: a systematic review and meta-analysis. Human reproduction. 2016 Dec 1;31(12):2841-55.*
7. *Conway G, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Franks S, Gambineri A, Kelestimur F, Macut D, Micic D, Pasquali R, Pfeifer M. European survey of diagnosis and management of the polycystic ovary syndrome: results of the ESE PCOS Special Interest Group's Questionnaire. European journal of endocrinology. 2014 Oct;171(4):489-98.*
8. *Jk Z. Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. Polycystic ovary syndrome. Current issues in endocrinology and metabolism. 1992:377-84.*
9. *Livadas S, Diamanti-Kandarakis E. Polycystic ovary syndrome: definitions, phenotypes and diagnostic approach. Front Horm Res. 2013 Jan 1;40:1-21.*
10. *Pasquali R, Gambineri A. Glucose intolerance states in women with the polycystic ovary syndrome. Journal of endocrinological investigation. 2013 Sep;36(8):648-53.*
11. *Cassar S, Misso ML, Hopkins WG, Shaw CS, Teede HJ, Stepto NK. Insulin resistance in polycystic ovary syndrome: a systematic review and meta-analysis of euglycaemic–hyperinsulinaemic clamp studies. Human reproduction. 2016 Nov 21;31(11):2619-31.*
12. *Mannerås-Holm L, Leonhardt H, Kullberg J, Jennische E, Oden A, Holm G, Hellström M, Lönn L, Olivecrona G, Stener-Victorin E, Lönn M. Adipose tissue has aberrant morphology and function in PCOS: enlarged adipocytes and low serum adiponectin, but not circulating sex steroids, are strongly associated with insulin resistance. The Journal of Clinical Endocrinology & Metabolism. 2011 Feb 1;96(2):E304-11.*
13. *Dunaif A, Segal KR, Shelley DR, Green G, Dobrjansky A, Licholai T. Evidence for distinctive and intrinsic defects in insulin action in polycystic ovary syndrome. Diabetes. 1992 Oct 1;41(10):1257-66.*
14. *Vgontzas AN, Trakada G, Bixler EO, Lin HM, Pejovic S, Zoumakis E, Chrousos GP, Legro RS. Plasma interleukin 6 levels are elevated in polycystic ovary syndrome independently of obesity or sleep apnea. Metabolism. 2006 Aug 1;55(8):1076-82.*
15. *Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, Janssen OE, Legro RS, Norman RJ, Taylor AE, Witchel SF. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. Fertility and sterility. 2009 Feb 1;91(2):456-88.*
16. *Barber TM, Hanson P, Weickert MO, Franks S. Obesity and polycystic ovary syndrome: implications for pathogenesis and novel management strategies. Clinical Medicine Insights: Reproductive Health. 2019 Sep;13:1179558119874042.*
17. *Singh S, Pal N, Shubham S, Sarma DK, Verma V, Marotta F, Kumar M. Polycystic ovary syndrome: etiology, current management, and future therapeutics. Journal of clinical medicine. 2023 Feb 11;12(4):1454.*
18. *De Leo V, Musacchio MC, Cappelli V, Massaro MG, Morgante G, Petraglia FJ. Genetic, hormonal and metabolic aspects of PCOS: an update. Reproductive biology and endocrinology. 2016 Dec;14(1):1-7.*
19. *Kakoly NS, Khomami MB, Joham AE, Cooray SD, Misso ML, Norman RJ, Harrison CL, Ranasinha S, Teede HJ, Moran LJ. Ethnicity, obesity and the prevalence of impaired glucose tolerance and type 2 diabetes in PCOS: a systematic review and meta-regression. Human reproduction update. 2018 Jul 1;24(4):455-67.*
20. *Mancini A, Bruno C, Vergani E, d'Abate C, Giacchi E, Silvestrini A. Oxidative stress and low-grade inflammation in polycystic ovary syndrome: controversies and new insights. International Journal of Molecular Sciences. 2021 Feb 7;22(4):1667.*
21. *Shukla A, Rasquin LI, Anastasopoulou C. Polycystic ovarian syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2026.*
22. *Sharma A, Sarwal Y, Devi NK, Saraswathy KN. Polycystic Ovary Syndrome prevalence and associated sociodemographic risk factors: a study among young adults in Delhi NCR, India. Reprod Health. 2025 Apr 28;22(1):61.*
23. *Sam S. Obesity and Polycystic Ovary Syndrome. Obes Manag. 2007 Apr;3(2):69-73.*
24. *Su P, Chen C, Sun Y. Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation. Journal of ovarian research. 2025 Feb 20;18(1):34.*
25. *Naeem I, Zehra A, Rehman F, Hussain A, Hussain A, Hussain N, Waseem M, Alqahtani RM, Shamlan G, Ahmed IA, Manzoor MF. Polycystic ovarian syndrome a risk factor for non-communicable diseases: insights into recent research and prevention approaches. Journal of Ovarian Research. 2025 Oct 10;18(1):219.*
26. *Yang Y, Ding M, Di N, Azziz R, Yang D, Zhao X. Close correlation between hyperandrogenism and insulin resistance in women with polycystic ovary syndrome-Based on liquid chromatography with tandem mass spectrometry measurements. J Clin Lab Anal. 2019 Mar;33(3):e22699.*
27. *Wang X, Nie H, Cui R, Ye G, Tan Y, Zhang J, Zhang B, Zhong X. Divergent pathophysiological drivers of polycystic ovary syndrome: insulin resistance independently fuels the hyperandrogenic phenotype whilst neuroendocrine factors dominate non-hyperandrogenic presentations. Frontiers in Endocrinology. 2026 Feb 4;17:1758861.*
28. *Wiweko B, Indra I, Susanto C, Natadisastra M, Hestiantoro A. The correlation between serum AMH and HOMA-IR among PCOS phenotypes. BMC Res Notes. 2018 Feb 9;11(1):114.*
29. *Li A, Zhang L, Jiang J, Yang N, Liu Y, Cai L, Cui Y, Diao F, Han X, Liu J, Sun Y. Follicular hyperandrogenism and insulin resistance in polycystic ovary syndrome patients with normal circulating testosterone levels. J Biomed Res. 2017 Nov 1;32(3):208–14.*