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Unveiling metabolic syndrome in polycystic ovarian syndrome: Hyperandrogenism as a key marker and implications for timely intervention

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Abstract
Introduction: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder in women of reproductive age with notable variability in prevalence globally and within Asia. PCOS is associated with metabolic and cardiovascular complications, prominently metabolic syndrome (MetS), due to its ties to insulin resistance and hyperandrogenism. This study aimed to assess MetS characteristics in women with PCOS and investigate the correlation between hyperandrogenism and MetS.
Materials and Methods: This prospective cross-sectional study was conducted over 18 months at the Post-Graduate Department of Obstetrics and Gynaecology, Govt. Medical College Srinagar, Jammu and Kashmir. A total of 200 participants aged 14-39, diagnosed with PCOS per Rotterdam criteria, were recruited. Exclusion criteria included recent hormonal or psychiatric medication use, and conditions like hyperprolactinemia or thyroid dysfunction. Participants underwent detailed history-taking and physical examination to assess MetS and hyperandrogenism.
Results: Among the 200 participants, hyperandrogenic (HA) and normoandrogenic (NA) groups were established, each comprising 100 individuals. The HA group exhibited significantly higher body mass index (BMI) and waist circumference compared to the NA group ($p<0.05$). MetS prevalence was notably higher in the HA group (33%) than in the NA group (9%), aligning with MetS's known association with hyperandrogenism. Significant disparities in fasting blood sugar, HDL, triglycerides, and blood pressure were also observed, with HA participants demonstrating elevated cardiovascular risk markers compared to their NA counterparts.
Conclusion: The findings underscore the heightened metabolic risk in HA PCOS patients, evidenced by elevated BMI, waist circumference, and MetS prevalence. The strong association between hyperandrogenism and metabolic disturbances emphasizes the need for focused management strategies in HA PCOS phenotypes.

Keywords: Polycystic ovary syndrome, metabolic syndrome, hyperandrogenism, insulin resistance, cardiovascular risk, body mass index

Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age, with a highly variable global prevalence. Reports indicate rates of 4.0% in the USA, 8.5% in Brazil, 16.6% in Denmark, and as high as 19.9% in Turkey [1]. Within Asian populations, recent studies reveal similar variability, with PCOS prevalence estimated at 7.1% using NIH criteria and 11.2% by Rotterdam criteria in China, and 4.8% by NIH criteria and 14.1% by Rotterdam criteria in Iran [2]. Among Kashmiri women of reproductive age, prevalence rates are notably high, with findings of 28.9% based on the NIH criteria, 35.3% with Rotterdam criteria, and 34.3% using the Androgen Excess PCOS Society (AEPCOS) criteria [3].

PCOS is not limited to reproductive symptoms but extends into metabolic and systemic health [4]. Characterized by reproductive irregularities, psychological burdens, cosmetic concerns, and a heightened risk of oncologic complications, PCOS is also intrinsically linked to metabolic dysfunction. Notably, women with PCOS exhibit a nearly double prevalence of metabolic syndrome (MetS) compared to the general female population, resulting in a sevenfold increase in cardiovascular disease risk [5]. Metabolic syndrome is defined by the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATPIII) criteria, which requires the presence of three or more of the following: waist circumference greater than 88 cm, triglyceride levels of at least 150 mg/dl, HDL-C lower than 50 mg/dl, blood pressure equal to or exceeding

130/85 mmHg, or fasting blood glucose between 100 and 126 mg/dl [6]. The strong correlation between PCOS and MetS highlights the need for comprehensive management strategies in women affected by this disorder.

Central to the pathophysiology of PCOS is insulin resistance, which affects an estimated 50-70% of PCOS patients but is not included in the formal diagnostic criteria for MetS [7]. Insulin resistance, a condition where normal insulin levels fail to produce the expected physiological response in fat, muscle, and liver cells, prompts the pancreas to compensate by secreting excess insulin, leading to hyperinsulinemia [8]. This elevated insulin level further complicates the metabolic profile of women with PCOS. Insulin resistance is frequently associated with abnormal fat distribution, particularly an increase in upper body fat that may be subcutaneous, intraperitoneal, or retroperitoneal. Among these, intraperitoneal fat accumulation correlates most strongly with insulin resistance and type 2 diabetes. Women with PCOS, regardless of their body mass index, often have a disproportionately large waist circumference compared to non-PCOS women within the same ethnic group, underscoring the syndrome's impact on body composition independent of obesity [9].

Hyperandrogenism, a hallmark feature of PCOS, is exacerbated by insulin resistance and hyperinsulinemia. Insulin resistance plays a critical role in stimulating ovarian androgen production, as elevated insulin levels activate insulin receptors, insulin-like growth factor (IGF-I), and luteinizing hormone (LH) receptors in the ovaries [10]. This activation initiates a cascade of hormonal effects that include the secretion of gonadotrophin-releasing hormone, which in turn stimulates LH production. Elevated insulin levels also contribute to the hyperandrogenic state by inhibiting hepatic production of sex hormone-binding globulin (SHBG), resulting in higher free testosterone levels in circulation [11]. The interplay between insulin resistance and hyperandrogenism not only exacerbates the clinical manifestations of PCOS but also significantly increases the risk factors for atherosclerotic cardiovascular disease and type 2 diabetes among these patients.

The relationship between hyperandrogenism and metabolic syndrome in PCOS underscores the urgency for early intervention and risk stratification in this population. Hyperandrogenism, driven by insulin resistance, amplifies both metabolic and reproductive symptoms, establishing it as a key marker for identifying patients at increased risk of metabolic syndrome and associated cardiovascular complications [12]. Considering the systemic nature of PCOS, targeted therapeutic approaches that address both insulin resistance and androgen excess are crucial in mitigating long-term health risks. Identifying hyperandrogenism as a predictive marker for metabolic syndrome in PCOS offers a pathway for timely and personalized intervention, particularly for managing cardiometabolic risk in affected women.

The present study was conducted in order to assess features of metabolic syndrome based on NCEP-ATP III guidelines, and correlate hyperandrogenism with the presence of metabolic syndrome in women with PCOS.

Materials and methods

Study type and design: The present study was a prospective cross-sectional study conducted in the Post-Graduate Department of Obstetrics and Gynaecology, Govt. Medical College Srinagar, Jammu and Kashmir over a period of eighteen months (April 2021-September 2022).

Study population: The study population consisted of patients aged 14-39 years who were diagnosed with PCOS according to the 2003 Rotterdam consensus. The exclusion criteria included recent use of hormonal contraception (within the past 6 months) or psychiatric medications, presence of hyperprolactinemia, thyroid dysfunction, other causes of hyperandrogenism (such as congenital adrenal hyperplasia, Cushing's syndrome, adrenal neoplasia, or virilizing ovarian tumors), and pregnancy.

Sampling methodology and sample size: A consecutive sampling methodology was utilized to recruit patients for the present study. All patients presenting to the study institution during the period of study and meeting the inclusion criteria and not excluded as per the exclusion criteria were recruited into the present study after obtaining written informed consent. Over the period of the study, a total of 200 patients were assessed.

Study methodology: A comprehensive history and general physical examination were conducted to assess the presence and severity of metabolic syndrome and hyperandrogenism in each patient, followed by appropriate laboratory tests. The history-taking process included inquiries about the age of thelarche, adrenarche, and menarche; any off-label use of anabolic steroids or testosterone; menstrual patterns regarding frequency and duration; and the timing and progression of acne and hirsutism along with previous treatments. Additionally, details of virilization symptoms, such as voice deepening or frontal balding, were recorded, as well as information on obesity onset, family history of relevant conditions like hirsutism, severe acne, PCOS, obesity, type 2 diabetes mellitus, hypertension, and cancer. Hirsutism was evaluated using the modified Ferriman-Gallwey scoring system across nine body areas, with scores of 8 or above indicating hirsutism. Systemic examination covered the respiratory, cardiovascular, and central nervous systems, and genital examination ruled out clitoromegaly. Blood samples were collected after an overnight fast within days 3-5 of the menstrual cycle, and hormone and metabolic markers, including total testosterone (TT), fasting blood sugar, HDL cholesterol, triglycerides, LH, FSH, TSH, serum prolactin, DHEAS, and 17-OH progesterone levels, were measured using Abbott ARCHITECT systems through chemiluminescent microparticle immunoassay methods.

Statistical analysis: The recorded data was compiled and entered in a spreadsheet (Microsoft Excel). Continuous variables were expressed as Mean ± SD and categorical variables were summarized as frequencies and percentages. Graphically the data was presented by bar and pie diagrams. Statistical significance was evaluated using the Student's unpaired t-test and the $\chi 2$-test. All statistical analysis was performed using SPSS® statistical package, version 11.0 (SPSS Inc., Chicago, IL, USA) for Windows®. A p-value ≤ 0.05 was considered to be statistically significant.

Ethical clearance: Consent was obtained or waived by all participants in this study. The Institutional Ethics Committee of Government Medical College Srinagar provided the required approval for this study under IRB No IRBGMC-SGR/Gynae789.

Results

The study comprised 200 participants, divided evenly between hyperandrogenic (n=100) and normoandrogenic (n=100) groups. Age distribution was fairly consistent across groups, with no statistically significant difference observed (p=0.113). In the hyperandrogenic group, 11 participants were aged 18-22, 37

were aged 23-27, 41 were in the 28-32 age range, and 11 were between 33-37 years. In the normoandrogenic group, there were 20 participants aged 18-22, 28 in the 23-27 range, 46 between 28-32, and 6 participants aged 33-37. The largest age category for both groups was 28-32 years, highlighting a predominant age range for PCOS diagnosis within the sample.

Significant differences in Body Mass Index (BMI) were observed between groups (p=0.002). In the normoandrogenic group, 59% of participants had a BMI within the range of 18.5-22.9, while only 39% of hyperandrogenic participants fell into this category. Conversely, in the 23-29.9 BMI range, a greater proportion of hyperandrogenic participants (56%) was noted compared to 41% in the normoandrogenic group. Furthermore, a small subset (5%) of hyperandrogenic participants exhibited a BMI above 30, indicative of obesity, whereas no participants in the normoandrogenic group had a BMI in this category. These findings suggest a trend toward higher BMI among hyperandrogenic individuals, which may reflect the influence of metabolic abnormalities associated with hyperandrogenism.

Key indicators of metabolic syndrome showed significant disparities between hyperandrogenic and normoandrogenic participants. Waist circumference, an important metric in metabolic syndrome evaluation, was notably greater among hyperandrogenic individuals (31.3±1.5 inches) than among normoandrogenic participants (28.8±0.9 inches, $p<0.05$). This increase aligns with previous findings linking abdominal adiposity to insulin resistance, which is often exacerbated by hyperandrogenic states.

Fasting blood sugar (FBS) levels were significantly elevated in the hyperandrogenic group (92.9±5.9 mg/dl) compared to the normoandrogenic group (86.2±3.9 mg/dl, $p<0.05$). This higher FBS level reflects a potential predisposition to impaired glucose tolerance, commonly associated with insulin resistance, a central component of metabolic syndrome in hyperandrogenic individuals.

Serum lipid profiles further emphasized differences in metabolic risk. HDL cholesterol levels, protective against cardiovascular disease, were lower among hyperandrogenic participants (46.2±2.2 mg/dl) versus their normoandrogenic counterparts (55.4±2.9 mg/dl, $p<0.05$). Conversely, triglyceride levels were significantly higher in the hyperandrogenic group (151.5±6.9 mg/dl) compared to the normoandrogenic group (137.7±6.5 mg/dl, $p<0.05$), indicating an elevated cardiovascular risk.

Blood pressure readings were higher in the hyperandrogenic group. The average systolic blood pressure (SBP) was 122.2±6.7 mmHg, compared to 118.4±3.3 mmHg in the normoandrogenic group ($p<0.05$), while the diastolic blood pressure (DBP) averaged 81.9±3.2 mmHg for hyperandrogenic participants versus 78.7±1.9 mmHg in normoandrogenic individuals ($p<0.05$). Elevated blood pressure is a hallmark of metabolic syndrome, further linking hyperandrogenism with increased metabolic risk.

Metabolic syndrome was significantly more prevalent in the hyperandrogenic group, with 33% of participants meeting the criteria compared to only 9% in the normoandrogenic group ($p<0.05$). This finding underscores a strong association between hyperandrogenism and metabolic syndrome components, suggesting that hyperandrogenic individuals with PCOS may face heightened metabolic and cardiovascular risks compared to normoandrogenic individuals.

Discussion

In the present study, the majority of patients in both the hyperandrogenic (HA) and normoandrogenic (NA) groups were observed to belong to the 28-32 age group. The mean age of the HA group was found to be 27.59±1.93 years, while that of the NA group was 26.64±2.27 years. These findings were in alignment with those reported by Tripathy *et al*. (2018), where the mean age for the HA group was documented as 27±5.30 years, similar to the present study, although the mean age of the NA group in Tripathy's study was reported to be slightly younger at 25.88±4.19 years [13]. In contrast, a study by Jamil *et al*. (2015) documented lower mean ages in both groups, with the HA group having a mean age of 26.78±4.95 years and the NA group 25.76±5.19 years, indicating possible demographic or regional variations between study populations [14].

Significant differences in body mass index (BMI) were observed between the HA and NA groups in this study. The mean BMI of the HA group was recorded as 24.13±1.75, whereas the NA group showed a lower mean BMI of 22.48±1.30. Within the HA group, 56% of participants were classified as overweight, with BMIs ranging from 23-29.9, while 59% of NA participants had normal BMIs within the 18.5-22.9 range. Additionally, 5% of patients in the HA group were classified as obese with BMIs exceeding 30. These results were consistent with findings by Huang *et al*. (2015), who reported a mean BMI of 22.89±5.07 in the NA group, closely matching those in this study [15]. However, the BMI values reported by Jamil *et al*. (2015) for both the HA (31.79±5.9) and NA (30.36±5.88) groups, as well as those by Tang *et al*. (2020) (HA: 28.1±5.1; NA: 23.1±4.3), were notably higher than those in the present study, likely reflecting BMI variations due to lifestyle, genetic influences, and dietary habits across populations [14, 16].

The prevalence of metabolic syndrome (MetS) was observed to be significantly higher among HA participants (33%) compared to NA participants (9%), with a p-value <0.05, demonstrating a strong association between hyperandrogenism and MetS. This prevalence was in agreement with Tavares *et al*. (2019) and Tripathy *et al*. (2018), who reported similar rates, ranging from 30.8% to 33.3% in hyperandrogenic phenotypes.**Error! Bookmark not defined.**, [17] Additionally, Tang *et al*. (2020) observed a wide prevalence range of 30.6% to 64.2% among South Asian hyperandrogenic populations, while Jamil *et al*. (2017) reported even higher prevalence rates, ranging from 58.3% to 80% among Middle Eastern populations.**Error! Bookmark not defined.**, **Error! Bookmark not defined.** In contrast, studies by Yildirim *et al*. (2017) and Hosseinpanah *et al*. (2014) documented much lower prevalence rates, ranging from 9.1% to 20%, in hyperandrogenic individuals [18, 19]. These variations were likely attributable to genetic, ethnic, and environmental factors influencing MetS risk profiles.

Tables and figures

Table 1: Sociodemographic characteristics of study participants (n=200)

Parameters	Hyperandrogenic (n=100)	Normoandrogenic (n=100)	Total (n=200)	p-value
Age				
18-22	11	20	31	0.113
23-27	37	28	65	
28-32	41	46	87	
33-37	11	6	17	
BMI				
18.5-22.9	39	59	98	0.002*
23-29.9	56	41	97	
30 and above	5	0	5	

*Statistically significant

Table 2: Metabolic syndrome related characteristics of the patients (n=200)

Parameters	Hyperandrogenic (n=100)	Normoandrogenic (n=100)	p-value
Waist circumference (inches)	31.3±1.5	28.8±0.9	<0.05*
FBS (mg/dl)	92.9±5.9	86.2±3.9	<0.05*
HDL (mg/dl)	46.2±2.2	55.4±2.9	<0.05*
Triglycerides (mg/dl)	151.5±6.9	137.7±6.5	<0.05*
SBP (mmHg)	122.2±6.7	118.4±3.3	<0.05*
DBP (mmHg)	81.9±3.2	78.7±1.9	<0.05*
Metabolic syndrome	33	9	<0.05*

*Statistically significant

Conclusion

In conclusion, the findings of this study highlighted a higher BMI and significant prevalence of metabolic syndrome in HA patients, supporting existing research that indicates a strong association between hyperandrogenism and metabolic risk factors. Nonetheless, observed regional differences in BMI and MetS prevalence among HA and NA groups suggested that lifestyle, dietary habits, and genetic factors may have influenced these associations. It was implied that hyperandrogenic PCOS phenotypes could be at an elevated risk for metabolic complications, highlighting the need for tailored management and preventive approaches. Further research accounting for genetic and lifestyle influences across diverse populations was recommended to enhance the understanding of the impact of hyperandrogenism on metabolic health in PCOS patients.

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