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Pattern of Hyperandrogenemia in Patients with Polycystic Ovary Syndrome at a Tertiary Care Hospital

Izhar ul Haq, Muhammad Sami, Sadaqat Ali, Qadeer Muhammad Khan, Fazal Rabi, Sana

ABSTRACT

Objectives: To assess the pattern of hyperandrogenemia in women diagnosed with polycystic ovary syndrome (PCOS).

Study Design and Setting: This was a descriptive cross-sectional study conducted at Hayatabad Medical Complex, Medical Teaching Institution (MTI), Peshawar.

Methodology: A total of 133 women aged 18–50 years with PCOS, diagnosed using the Rotterdam criteria, were enrolled through non-probability convenience sampling. Women with prior hormone therapy, hysterectomy, ovarian dermoid, or pelvic inflammatory disease were excluded. Serum levels of total testosterone (>88 ng/dl), free testosterone (>0.66 ng/dl), and DHEAS (>2750 ng/ml) were measured and stratified with age, BMI, and menopausal status. Data were analyzed using SPSS version 26, applying the chi-square test with a significance level of $p < 0.05$.

Results: Elevated free testosterone was observed in 34.6% of participants, followed by total testosterone in 24.8% and DHEAS in 24.1%. A statistically significant association was found between age and raised DHEAS ($p = 0.046$), while no significant associations were observed with BMI or menopausal status.

Conclusions: Free testosterone was the most frequently elevated androgen marker in women with PCOS, supporting its role in biochemical assessment. The age-related rise in DHEAS suggests the need for individualized hormonal interpretation to enhance diagnostic accuracy and guide management.

Keywords: Androgens, Body Mass Index, Cross-Sectional Studies, Dehydroepiandrosterone Sulfate, Polycystic Ovary Syndrome, Testosterone, Women's Health

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with an estimated prevalence of around 5-18%.[1] Among this sizable population, a significant proportion is diagnosed with PCOS, a condition defined by a combination of clinical, hormonal, and sometimes ultrasonographic features.[2] PCOS is a heterogeneous disorder and is largely characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology on ultrasound after the exclusion of other known causes of androgen excess, such as adrenal hyperplasia, androgen-secreting tumors, and Cushing's syndrome.[3]

One of the defining features of PCOS is hyperandrogenism, which refers to an excess of androgens such as testosterone, dehydroepiandrosterone sulfate (DHEAS), and androstenedione.[4] Hyperandrogenism may present clinically in the form of hirsutism (excessive hair growth in a male pattern distribution), acne, or androgenic alopecia (male-pattern hair thinning).[5] It may also be detected biochemically through elevated serum androgen levels. However, diagnosing hyperandrogenism remains complex due to the variability in both clinical presentation and laboratory measurement methods.

The diagnosis of PCOS itself poses a challenge due to its nature as a diagnosis of exclusion and the absence of a universally accepted gold standard.[6] Hyperandrogenism, despite being central to most diagnostic criteria, including the Rotterdam, NIH, and AE-PCOS Society guidelines, remains difficult to define precisely.[7] The subjective nature of clinical symptoms, such as hirsutism and acne, often leads to variability in assessment due to differences in observer interpretation, patient ethnicity, and individual thresholds of normalcy.[8] The same degree of hair growth may be considered hirsute in one ethnic group but normal in another. Consequently, the reliability of clinical hyperandrogenism as a diagnostic tool is limited.[9]

While clinical evaluation of hyperandrogenism is often variable, biochemical hyperandrogenemia provides a potentially more objective alternative. However, even in this realm, the diagnostic process is far from standardized. Total testosterone (TT), free testosterone (FT), and DHEAS are commonly used biomarkers, but their reference ranges and assay techniques vary significantly between laboratories.[10] Moreover, many prior studies have relied on limited patient samples to establish normative values, further complicating the interpretation of hormonal levels across different populations.

In clinical practice, total testosterone has traditionally been the most commonly measured androgen to assess biochemical hyperandrogenism. However, it is now recognized that free testosterone and DHEAS also provide valuable information and may be elevated even when TT is within normal limits. In a notable study involving 716 women diagnosed with PCOS, 75.3% of participants had at least one marker of hyperandrogenism. Specifically, 57.6% had elevated free testosterone, 33.0% had elevated total testosterone, and 32.7% had elevated DHEAS. Interestingly, the overlap of elevated androgens was variable: 20.4% had concurrent elevations in both TT and FT, while only 1.7% showed simultaneous elevations in TT and DHEAS. Only 8.7% of the study participants had elevated levels of all three markers TT, FT, and DHEAS, highlighting the heterogeneity of androgen profiles in PCOS.[11]

This variability in hormonal expression underscores the complexity of diagnosing and managing PCOS. While the prevalence of hyperandrogenism is readily identifiable in this patient population, there is no consensus around the most common androgen marker or markers that are routinely measured in clinical practice. The variety of hormonal patterns present complicates diagnosis and subsequent individualized targeted treatment approaches.[12]

Given the current non-specificity and extremely wide variability of androgen patterns that are evident in women with PCOS, further research into the hormonal patterns of women with hyperandrogenemia is certainly warranted. A clear understanding of the most frequently elevated androgenic biomarkers and reliable means of their identification can help clarify the biology of PCOS. More specifically, understanding the patterns of biomarkers can also inform diagnosis (i.e., more clearly defining diagnostic criteria), decrease dependence on subjective clinical signs, and consequently inform targeted therapeutic approaches. The purpose of this study was to investigate patterns of hyperandrogenemia in women with PCOS, using combinations of hormonal parameters such as total testosterone, free testosterone, and DHEAS. Our objective is to identify potential common biochemical patterns of androgen excess in PCOS, specifically to improve diagnosis, develop potentially more defined management in women with PCOS.

METHODOLOGY

This descriptive cross-sectional study was conducted to evaluate the patterns of hyperandrogenemia in women with polycystic ovary syndrome under the auspices of the Department of Diabetes, Endocrinology, and Metabolic Diseases, Hayatabad Medical Complex, Medical Teaching Institution (MTI), Peshawar. The study period commenced on July 1, 2024, and ended on December 31, 2025.

Ethical approval for the study was obtained from the Institutional Review Board of Hayatabad Medical Complex, MTI, Peshawar (Approval No. 1672, dated April 7, 2024). All participants were informed about the nature and purpose of the study, and written informed consent was obtained before enrollment.

A sample size of 133 women was included in the study. The sample size was calculated using the WHO sample size calculator, considering a 33.0% frequency of elevated total testosterone among women with PCOS, with an 8% margin of error and a 95% confidence level.[13]

A non-probability convenient sampling technique was used for the recruitment of participants. Inclusion criteria comprised women aged 18 to 50 years who were diagnosed with PCOS based on the Rotterdam criteria. According to this definition, a diagnosis of PCOS required the presence of at least two out of the following three features: polycystic ovarian morphology on ultrasound, clinical signs of hyperandrogenism such as hirsutism or male-pattern baldness (after excluding other causes of androgen excess such as Cushing's syndrome and congenital adrenal hyperplasia), and oligo-ovulation or anovulation. Women who had a history of hormone therapy, hysterectomy, ovarian dermoid cysts, or pelvic inflammatory disease were excluded from the study.

After the participants provided consent, they underwent a structured interview and clinical evaluation. Information was collected regarding demographic information, menstrual history, and hyperandrogenic symptoms. Physical examination included measurements of height, weight, and waist circumference for body mass index (BMI), and

assessment of physical signs of androgen excess, which included hirsutism and balding in the frontal scalp distribution.

For hormonal collection, the participants had 20 cc venous blood drawn from a major superficial vein in the non-dominant arm after she was seated comfortably in a chair. Ideally, blood was sent to the hospital laboratory immediately and analyzed within 30 minutes to optimize hormonal measurements. Blood samples were analyzed for total testosterone, free testosterone, and dehydroepiandrosterone sulfate (DHEAS). Hyperandrogenemia was determined by increased measurement in any of the following: total testosterone >88 ng/dl, free testosterone >0.66 ng/dl, DHEAS >2750 ng/ml. The number of measurements of any elevated levels, hyperandrogenemia sub-phenotype, was determined based on = 1 elevated hormonal parameter.

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 26. Continuous variables such as age, BMI, and hormone levels were reported as mean ± standard deviation. Categorical variables, including the presence or absence of specific symptoms or hormonal abnormalities, were presented as frequencies and percentages. The pattern of hyperandrogenemia was stratified across various baseline and clinical variables to identify effect modifiers. Post-stratification, the Chi-square test was applied to determine statistical significance, with a p-value of less than 0.05 considered significant.

RESULTS

A total of 133 women with polycystic ovary syndrome participated in the study. The mean age of the participants was 30.5 years with a standard deviation of 5.29. More than half of the women were above the age of 35. The majority of participants had a normal body mass index (BMI), while nearly one-third were categorized as overweight or obese. Most of the women were nulliparous, indicating that they had not given birth previously, whereas a smaller proportion had one or more previous births. Regarding family history, only a small percentage reported a positive family history of PCOS, while the vast majority did not. A significant number of participants had a known history of diabetes, though over half reported no such history. The menopausal status showed that nearly all participants were in the pre-menopausal phase, with only a few in peri- or post-menopause. In terms of marital status, the majority of the women were married. Educational background revealed that slightly more than half of the participants had completed matriculation or had lower education levels, while the remainder had education beyond matric. Employment status indicated that most of the women were unemployed, with less than one-third engaged in professional work. (Table 1)

Among the outcome variables assessed in the study, elevated levels of free testosterone were the most frequently observed form of hyperandrogenemia among participants. Raised total testosterone and DHEAS levels were also noted, though less commonly. These findings highlight the variability in biochemical expression of hyperandrogenism in women with PCOS, suggesting that free testosterone may serve as a more sensitive marker in this population compared to total testosterone or DHEAS alone. (Table 2)

The stratification of hyperandrogenemia patterns by age revealed no statistically significant association between age groups and elevated total or free testosterone levels. However, a significant relationship was observed between age and raised DHEAS levels ($p = 0.046$), indicating that DHEAS elevation was more commonly seen in women above 35 years of age. These findings suggest that among the androgenic biomarkers evaluated, DHEAS may show age-related variation in women with PCOS. (Table 3)

The relationship between BMI and the pattern of hyperandrogenemia was assessed; however, no statistically significant association was found between BMI categories and elevated levels of total testosterone, free testosterone, or DHEAS. Although raised androgen levels were slightly more prevalent among participants with normal BMI, the differences were not statistically significant. This suggests that while overweight or obesity is commonly associated with PCOS, it may not be a sole determinant of biochemical hyperandrogenism. (Table 4)

Analysis of hyperandrogenemia patterns by menopausal status showed that nearly all cases of elevated androgen levels occurred in pre-menopausal women. No statistically significant associations were found between menopausal

Table 1. Demographics and Baseline Characteristics of Study Participants (n = 133)

Parameter	Category	n (%)
Age (years)	Mean ± SD	30.50 ± 5.29
	= 35 years	62 (46.6%)
	> 35 years	71 (53.4%)
BMI (kg/m²)	Normal	93 (69.9%)
	Overweight/Obese	40 (30.1%)
Parity	Nulliparous	90 (67.7%)
	Uni/Multiparous	43 (32.3%)
Family History of PCOS	Yes	19 (14.3%)
	No	114 (85.7%)
History of Diabetes	Yes	57 (42.9%)
	No	76 (57.1%)
Menopause Status	Pre-menopause	128 (96.2%)
	Peri/Post-menopause	5 (3.8%)
Marital Status	Married	107 (80.5%)
	Unmarried	26 (19.5%)
Education	Matric or below	74 (55.6%)
	Above matric	59 (44.4%)
Profession	Employed	38 (28.6%)
	Unemployed	95 (71.4%)

status and elevations in total testosterone, free testosterone, or DHEAS. However, the distribution reflects the predominance of pre-menopausal status in the study population, which is expected given the inclusion criteria focusing on reproductive-aged women. (Table 5)

DISCUSSION

In this descriptive analysis of 133 women with PCOS, elevated free testosterone was more prevalent than raised total testosterone or DHEAS, patterns consistent with international data and diagnostic guidelines. Our free testosterone prevalence (~34.6%) is somewhat lower than values reported in larger cohorts, but the ranking remains aligned with global trends where free testosterone is frequently the most sensitive biochemical marker.

A large NIH-based cohort study of 716 PCOS patients reported free testosterone elevation in 57.6%, total testosterone in 33.0%, and DHEAS in 32.7% of cases, with 75.3% of participants having at least one elevated androgen marker.[11, 14] While we observed a similar total testosterone prevalence (~24.8%) and DHEAS (~24.1%), our free testosterone frequency was lower. This difference could reflect population-specific reference cutoffs, assay variation, or demographic differences.

The 2023 International PCOS Evidence-Based Guidelines highlighted that free testosterone consistently identifies the largest proportion of hyperandrogenemic women and recommended high-precision assays or calculated indices (e.g., FAI) for accurate detection.[15] This aligns with our finding that free testosterone remains the most commonly elevated marker, reaffirming its clinical utility.

BMC Endocrine Disorders in 2024 evaluated anthropometric indices and their relation to hormonal imbalance, concluding that BMI had no consistent association with total testosterone or DHEAS levels.[11] This supports the present study observation, where no statistically significant differences in

Table 2. Distribution of Outcome Variables (n = 133)

Outcome Variable	Category	n (%)
Raised Total Testosterone (TT)	**Yes**	33 (24.8%)
	No	100 (75.2%)
Raised Free Testosterone (FT)	**Yes**	46 (34.6%)
	No	87 (65.4%)
Raised DHEAS	**Yes**	32 (24.1%)
	No	101 (75.9%)

Table 3. Stratification of Pattern of Hyperandrogenemia by Age (n = 133**)**

Hyperandrogenemia Pattern	Age = 35 years	Age > 35 years	Total (n)	p-value
Raised Total Testosterone	15 (45.5%)	18 (54.5%)	33	0.877
Normal Total Testosterone	47 (47.0%)	53 (53.0%)	100	
Raised Free Testosterone	18 (39.1%)	28 (60.9%)	46	0.208
Normal Free Testosterone	44 (50.6%)	43 (49.4%)	87	
Raised DHEAS	10 (31.3%)	22 (68.8%)	32	0.046*
Normal DHEAS	52 (51.5%)	49 (48.5%)	101	

Table 4. Stratification of Hyperandrogenemia Pattern by BMI (n = 133)

Hyperandrogenemia Pattern	Normal BMI	Overweight/Obese	p-value
Raised Total Testosterone	22 (66.7%)	11 (33.3%)	0.638
Normal Total Testosterone	71 (71.0%)	29 (29.0%)	
Raised Free Testosterone	35 (76.1%)	11 (23.9%)	0.260
Normal Free Testosterone	58 (66.7%)	29 (33.3%)	
Raised DHEAS	19 (59.4%)	13 (40.6%)	0.135
Normal DHEAS	74 (73.3%)	27 (26.7%)	

Table 5. Stratification of Hyperandrogenemia Pattern by Menopause Status (n = 133)

Hyperandrogenemia Pattern	Pre-menopause	Peri/Post-menopause	Total (n)	p-value
Raised Total Testosterone	32 (97.0%)	1 (3.0%)	33	0.800
Normal Total Testosterone	96 (96.0%)	4 (4.0%)	100	
Raised Free Testosterone	45 (97.8%)	1 (2.2%)	46	0.485
Normal Free Testosterone	83 (95.4%)	4 (4.6%)	87	
Raised DHEAS	32 (100.0%)	0 (0.0%)	32	0.199
Normal DHEAS	96 (95.0%)	5 (5.0%)	101	

androgen patterns were found concerning BMI. Similarly, a 2025 study examining BMI-stratified PCOS revealed that the overall prevalence of hyperandrogenemia exceeded 78%, affecting both overweight and lean participants; no significant BMI-related differences emerged for testosterone, free testosterone, or DHEAS, again matching our non-significant BMI stratifications.[16]

Age-related variation in DHEAS is well-documented. A study focusing on adrenal androgen prevalence demonstrated a notable decline in serum DHEAS with increasing age, particularly after 30 years, reflecting a negative correlation between DHEAS levels and age.[14] Similarly, the present study findings show higher DHEAS levels among women over 35 ($p = 0.046$), while testosterone markers remained age-insensitive. This supports the concept of adrenal androgen decline with aging.

A U.S. cohort from 2020 reported that asymptomatic women with elevated DHEAS had increased metabolic risks independent of obesity, and DHEAS concentrations were inversely associated with age but independent of BMI.[17] These findings corroborate our age-related DHEAS stratification despite the absence BMI effect. Evidence from a review on androgen prevalence estimates biochemical hyperandrogenism in 25–35% of women with PCOS for DHEAS, whereas free testosterone may be raised in up to 89% and total testosterone in 49–80%.[18] Our study falls within these ranges for total testosterone and DHEAS, but our free testosterone estimate appears at the lower end, possibly due to assay differences or cut-off definitions.

Other emerging PCOS literature (2023–2025) has proposed reclassifying PCOS into hyperandrogenic and hypo/hyperandrogenic phenotypes with age-dependent decline in androgenicity after age 35.[19] Although speculative, our findings of lower DHEAS with age fit the concept of a phenotypic shift in androgen profiles in older PCOS patients. A 2024 anthropometric study (BMC Endocrine Disorders) confirmed that markers like BMI and WHtR did not reliably predict androgen levels, reinforcing that obesity status alone is insufficient to stratify androgen excess risk, a conclusion mirrored in our BMI-stratified analysis.[20, 21] Attempts to link increased enzyme activity (like 5á-reductase) with hyperandrogenism and insulin resistance were also highlighted in recent reviews, though direct population parallels are limited.[22] While not measured in our study, these mechanisms may partly explain biochemical variability despite similar BMI profiles.

In summary, across the literature from 2020–2025, consistent trends appear: free testosterone often yields the highest detection rate of hyperandrogenemia; DHEAS is variably elevated with an inverse relationship to age; and BMI shows limited predictive value for biochemical hyperandrogenism in PCOS. Our study largely parallels these global observations, with unique local values shaped by assay-specific definitions and demographic context.

The findings of this study have important clinical implications for the diagnosis and management of polycystic ovary syndrome (PCOS), particularly in resource-limited settings. The observation that free testosterone is the most frequently elevated androgen marker supports its role as a key biochemical indicator in the assessment of hyperandrogenemia. Clinicians should therefore prioritize free testosterone measurement when evaluating women with suspected PCOS, especially in cases where clinical signs such as hirsutism and acne are subtle or absent.

Furthermore, the significant association between age and DHEAS levels suggests that androgen profiles may shift with age, with adrenal androgen excess more prevalent in older reproductive-age women. As these findings show, there must be an age-stratified interpretation of androgen levels for PCOS diagnosis. Similarly, the non-significant association between BMI and androgen excess suggests there should not be an exclusion of patients with biochemical hyperandrogenism based only on body weight, as it is particularly relevant that all patients with suspected or diagnosed PCOS should be screened for hyperandrogenism. This is particularly important for lean PCOS patients, so they are not neglected due to the belief that obesity should precede assessment of hormones.

The findings of this study have several important clinical implications for the evaluation and management of women with polycystic ovary syndrome (PCOS). First, the observation that free testosterone was the most frequently elevated androgen highlights the need for clinicians to prioritize its measurement over total testosterone and DHEAS when screening for biochemical hyperandrogenemia. Free testosterone is a more sensitive marker, reflecting biologically active androgens, and its routine assessment may reduce underdiagnosis in women who present with clinical features of hyperandrogenism but have normal total testosterone levels.

Second, the significant association between age and elevated DHEAS emphasizes that clinicians should consider age-specific variations when interpreting androgen profiles in PCOS. Younger women may show a predominance of ovarian androgens (testosterone), while adrenal contribution (DHEAS) may increase with age. This distinction is clinically relevant because it may influence treatment choices, for example, adrenal-targeted therapies could be more beneficial in older patients with predominant DHEAS elevations.

Third, since no significant correlation was observed with BMI or menopausal status, this study suggests that hyperandrogenemia can manifest independently of obesity or reproductive stage. Clinicians should therefore avoid relying solely on weight reduction or menopausal transition as predictors of androgen normalization and instead ensure regular hormonal monitoring across all BMI categories and

age groups.

Finally, these findings support a personalized approach in managing PCOS by tailoring treatment strategies based on the predominant androgen abnormality. For instance, combined oral contraceptives and anti-androgen therapies may be prioritized for elevated free testosterone, while adrenal suppression strategies (e.g., glucocorticoids) may be considered in women with persistently raised DHEAS. Such individualized management could improve symptom control, fertility outcomes, and long-term metabolic health in PCOS patients.

LIMITATIONS:

Despite some contributions to research on hormonal characteristics of hyperandrogenemia in women with PCOS, this study has limitations. First, the cross-sectional design limits the ability to comment on any definitive cause/effect of hyperandrogenism in relation to any classifying variable, like age and BMI. Longitudinal studies could provide insight into the time properties of androgen levels in PCOS and its variations across the reproductive life span. Second, the study recruited participants in one tertiary care hospital, as a non-probability convenience sampling technique, leading to possible limited generalization of findings to different representativeness within the population of women with PCOS across Canada. The sample of women in this study may not have sufficiently represented the diversity of PCOS phenotypes in rural or underserved populations.

Third, the assessment of biochemical hyperandrogenism primarily relied on serum levels of total testosterone, free testosterone, and DHEAS without the use of any advanced measures such as the free androgen index (FAI) or estradiol and sex hormone binding globulin (SHBG), which could have helped enhance the diagnostic accuracy. Furthermore, differences in assay sensitivity and the absence of standardized reference ranges may also complicate the interpretation of individual hormone levels. Lastly, clinical hyperandrogenism was not measured with validated scoring systems such as the modified Ferriman-Gallwey score, which may impact comparability with studies that employed more objective clinical criteria. Despite these limitations, the study provides meaningful information to the existing data on PCOS and identifies the need for standardized and multidimensional assessments.

CONCLUSION

This study revealed the heterogeneous nature of hyperandrogenemia in women with polycystic ovary syndrome, and identifies free testosterone as the most commonly elevated androgen marker for anovulation, followed by total testosterone and DHEAS. The identified significant association between age and increased prevalence of raised DHEAS levels highlights the overall impact of age on adrenal androgen production, whereas the lack of correlation with BMI reinforces that hyperandrogenism can occur despite body weight. The outcomes of this review support a detailed hormonal profile in all women with PCOS to ensure a complete differential diagnosis and personalized management. The addition of a free testosterone test to standard assessment may increase the sensitivity of detecting biochemical hyperandrogenism, thereby potentially improving clinical outcomes in this heterogeneous population.

Authors Contribution:
Izhar Ul Haq: Introduction + Discussion, data collection
Muhammad Sami: Data Collection + review article
Sadaqat Ali: Data Collection + review article
Qadeer Muhammad Khan: Data analysis and conclusion
Fazal Rabi: Data Collection + review article+
Sana: Data Collection + review article

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