e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(7); 2011-2015

Original Research Article

Association of AMH and hyperandrogenism in PCOS

Saranya Sasikumar[1], Amit Kumar Sil[2], Devapriya Roy[3]

[1]Senior Resident, Department of Obstetrics & Gynaecology, Northern Railway Central Hospital, New Delhi, India

[2]Professor, Department of Obstetrics & Gynaecology, IQ City Medical College, Durgapur, India

[3]Secondary DNB Resident, Department of Obstetrics & Gynaecology, Northern Railway Central Hospital, New Delhi, India

Received: 10-05-2025 / Revised: 09-06-2025 / Accepted: 10-07-2025

Corresponding Author: Devapriya Roy

Conflict of interest: Nil

Abstract:

Background: PCOS, a complex endocrine disorder characterized by polycystic ovarian morphology, oligo/anovulation, and hyperandrogenism, commonly affects women of reproductive age. Anti-Müllerian Hormone (AMH), secreted by granulosa cells of preantral and micro antral follicles, is elevated in PCOS and is increasingly being identified as a potential biomarker for ovarian reserve and hyperandrogenism. Gaining knowledge about the connection between AMH and hyperandrogenism could help with PCOS pathogenesis and diagnostic assessment.

Aim: To assess how blood AMH levels relate to biochemical and clinical hyperandrogenism in PCOS-afflicted women.

Methods: An observational and ccomparative study was conducted at the Medical College, Kolkata between 2020 and 2021. 80 women between the age 18 -35 years were classified into 2 groups: 40 females diagnosed as PCOS according to Rotterdams criteria and 40 females categorized as non-PCOS. Clinical information including hirsuitism, acne, menstrual irregularity, were collected. Hormonal profiles such as serum levels of AMH, testosterone, DHEAS were also studied and compared between the two groups. Statistical analysis was performed using SPSS version 23.0.

Results: The BMI ($p = 0.0006$), AMH levels (7.5656± 2.1041ng/ml vs. 2.6672± 1.0912 ng/mL; $p < 0.001$), mean serum total testosterone (0.6798 ± .2982 ng/ml vs 0.3251± .1187 ng/ml) , serum DHEAS (287.2880± 150.4731 microgram/dl vs 184.7974± 91.8628 microgram/dl) ,hirsutism score ($r = 0.54$, $p < 0.001$) and acne score ($r = 0.61$, $p < 0.001$) were all considerably higher in the PCOS group. Among PCOS patients, 91.3% of higher testosterone (>0.6ng/ml), 91.6% of patients with high DHEAS (>450microgram/dl), 90% patients with hirsuitism (Modified FG score >/=8) and 90% of patients with acne exhibited a somewhat positive connection with AMH.

Conclusion: This study demonstrates a significant association between elevated serum AMH levels and clinical/biochemical hyperandrogenism in PCOS. AMH may serve as a surrogate marker for hyperandrogenism in PCOS, offering diagnostic value especially where direct androgen assays are limited.

Recommendations: Measurement of AMH may be considered in the diagnostic and monitoring framework of PCOS apart from the traditional diagnostic criteria for PCOS. Further longitudinal studies are warranted to explore its role in predicting clinical outcomes and therapeutic response.

Keywords: PCOS, Anti-Müllerian Hormone, Hyperandrogenism, AMH, Androgens.

Introduction

Depending on the diagnostic criteria employed, the prevalence of PCOS, a common endocrine condition affecting women of reproductive age, can range from 6% to 20% worldwide [1]. Among the many clinical signs of PCOS, which is defined by a combination of oligo- or anovulation, hyperandrogenism, and polycystic ovarian morphology, are irregular menstruation, hirsutism, acne, infertility, and metabolic issues [2]. One of the main characteristics of PCOS is hyperandrogenism, which contributes significantly to the metabolic and reproductive problems of the condition and can appear biochemically (higher serum androgens) or clinically (hirsutism, acne) [3].

AMH, a glycoprotein belonging to the transforming growth factor-β family, is secreted by the granulosa cells of preantral and microantral follicles. It is essential for folliculogenesis because it inhibits the early recruitment of primordial follicles and reduces follicular sensitivity to follicle-stimulating hormone (FSH) [4]. Because of an increase in tiny antral follicles, women with PCOS often have high blood

AMH, which is thought to be a reliable indicator of ovarian reserve [5]. MH may be correlated with androgen levels and the severity of clinical characteristics such hirsutism and ovulatory dysfunction, according to research that have suggested a potential link between AMH and hyperandrogenism in PCOS in recent years [6,7].

The pathophysiological connection between AMH and hyperandrogenism is believed to be bidirectional. Elevated androgens may stimulate granulosa cells to produce more AMH, while AMH may interfere with gonadotropin signaling and enhance luteinizing hormone (LH)-driven androgen production by theca cells [8]. Furthermore, AMH may act at the hypothalamic level to modulate gonadotropin-releasing hormone (GnRH) secretion, thereby promoting a neuroendocrine environment favoring hyperandrogenism [9]. However, the extent and consistency of the association between AMH and hyperandrogenism remain areas of ongoing investigation, particularly given the heterogeneity in PCOS phenotypes

Methodology

Study Design: This study was an observational and comparative study.

Study Setting: The study was conducted at the Department of Obstetrics and Gynaecology, Medical College, Kolkata, which is a tertiary care teaching hospital in Eastern India. This institution caters to a wide population from both urban and rural settings, allowing diverse participant recruitment.

Study Duration: The study was carried out over a period of one year, from March 2020 to February 2021. This duration included participant recruitment, data collection, sample analysis, and statistical evaluation.

Participants: 40 women who met the Rotterdam Criteria (2003) for PCOS and 40 women who attended Opd for other causes were included in the study. Both the group were age matched, 18- 35 years.

Inclusion Criteria

- Women aged 18 to 35 years.
- PCOS was diagnosed using the Rotterdam criteria, which include polycystic ovarian morphology on ultrasonography, clinical/biochemical indicators of hyperandrogenism, and oligo/anovulation.
- The readiness to take part and give informed consent.

Exclusion Criteria

- The presence of other endocrine disorders, including Cushing's syndrome, congenital adrenal hyperplasia, and thyroid dysfunction.
- Use of hormonal medications, insulin sensitizers, or ovulation induction agents within the last three months.
- Known cases of ovarian or adrenal tumors.
- Pregnancy or lactation.
- Severe systemic illness affecting hormonal levels.
- Previous ovarian or adrenal surgeries.

Bias: To minimize selection bias, consecutive sampling was used during patient recruitment following inclusion and exclusion criteria. All hormone assays were performed in a single laboratory to reduce inter-laboratory variation and ensure uniformity. Observer bias was reduced by ensuring that clinical assessments of hyperandrogenism (hirsutism, acne) were performed by trained personnel using standardized scoring systems (e.g., modified Ferriman-Gallwey score).

Data Collection: A pretested structured proforma was used to gather pertinent clinical and demographic data following the acquisition of informed written consent. Menstrual history, BMI, ultrasonography results, and clinical characteristics of hyperandrogenism were noted. During the early follicular phase (days 2–5) of the menstrual cycle, fasting blood samples were taken in order to analyze AMH, total testosterone, and other pertinent hormonal markers like DHEAS.

Procedure: A comprehensive clinical examination was performed on the participants, which included evaluations for alopecia, acne (acne was graded as: grade 0- no acne, grade1- comedones, grade 2 – come dones with occasional papules, grade 3 – many come dones, papules and occasional pustules, grade 4 – predominant pustules with widespread scarring), and hirsutism (using the modified Ferriman-Gallwey score). Ovarian morphology was evaluated by transvaginal or transabdominal ultrasonography. Following an overnight fast, blood samples were drawn, and processed for hormonal assays for AMH, total testosterone and DHEAS, utilizing chemiluminescence and enzyme-linked immunosorbent assay (ELISA) techniques. Quantitative measurements of AMH levels were made and compared between PCOS and non-PCOS groups and also observed among PCOS patients with clinical/biochemical hyperandrogenism.

Statistical Analysis: SPSS version 23.0 was used to analyze the data. The mean ± SD was used for continuous variables, and frequency and percentage were used for categorical variables. For continuous variables, the independent t-test or Mann-Whitney U test was employed, while for categorical variables, the chi-square test. Pearson or Spearman coefficients were used to evaluate the correlations between AMH and hyperandrogenism indicators. P-values less than 0.05 were regarded as statistically significant.

Results

There was no statistically significant distinction of age between the two groups (PCOS vs non-PCOS), p=0.412. The PCOS group had a significantly higher mean BMI (p = 0.0006), suggesting that women with PCOS typically have higher BMIs.

Table 1: Baseline Characteristics of Study Participants

Variable	PCOS Group (n = 40)	Non-PCOS Group (n = 40)	p-value
Age (years)	25.3 ± 4.1	26.0 ± 4.3	0.412
BMI (kg/m²)	26.5 ± 3.5	24 ± 2.43	0.0006

*Significant at p < 0.05

Pattern of distribution of serum AMH, biochemical and clinical hyperandrogenism in PCOS: The levels of AMH, total testosterone and DHEAS were found to be considerably greater among PCOS patients than those of the non-PCOS group. Presence of acne and hirsuitism was also significantly higher among PCOS patients.

Table 2: Hormonal Parameters in PCOS vs Non-PCOS Groups

Hormonal Parameter	PCOS Group (n = 40)	Non-PCOS Group (n = 40)	p-value
AMH (ng/mL)	7.56± 2.1	2.67± 1.09	<0.0001*
Total Testosterone (ng/mL)	0.68 ±0.3	0.32± 0.12	<0.0001*
DHEAS	287.29± 150.47 (	184.79± 91.87	<0.004*
Add table 16			
Add table 18			

*Significant at p < 0.05

Association Between AMH and Hyperandrogenism Markers: AMH levels were found to be high among PCOS patients with clinical hyperandrogenism like hirsuitism and acne and also with higher biochemical parameters like mean total testosterone and mean DHEAS.

Table 3: Association of Serum AMH with Serum Total Testosterone

Serum Testosterone (ng/ml)	Cases (n)	Patients with Serum AMH ≥5 ng/ml	Percentage
0.1 – 0.59	17	13	76.5%
0.6 – 0.99	16	14	87.5%
≥ 1.0	7	7	100%

Serum AMH levels were found to correlate positively with serum testosterone. All patients with testosterone ≥1.0 ng/ml had elevated AMH levels, whereas those with low–normal testosterone still showed AMH elevation in a significant proportion. This supports a link between biochemical hyperandrogenism and higher AMH concentrations.

Table 4: Association of Serum AMH with Serum DHEAS

Serum DHEAS (µg/dl)	Cases (n)	Patients with Serum AMH ≥5 ng/ml	Percentage
< 150	9	6	66.7%
150 – 300	11	10	90.9%
300 – 450	8	7	87.5%
≥ 450	12	11	91.6%

A progressive increase in AMH positivity was noted with rising DHEAS levels. Patients with DHEAS ≥150 µg/dl consistently demonstrated higher AMH, with the strongest association seen in those ≥450 µg/dl. This indicates a significant correlation between adrenal hyperandrogenism and ovarian AMH secretion.

Table 5: Association of Serum AMH with Modified FG Score (Hirsutism)

Modified FG Score	Cases (n)	Patients with Serum AMH ≥5 ng/ml	Percentage
≥ 8 (Hirsutism +)	20	18	90%
< 8 (No Hirsutism)	20	16	80%

Women with clinical hirsutism (FG ≥8) showed a higher of elevated number of elevated AMH compared to those without hirsutism. This suggests that AMH levels may reflect clinical severity of hyperandrogenism.

Table 6: Association of Serum AMH with Acne Grades in PCOS

Acne Grade	Cases (n)	Patients with Serum AMH ≥5 ng/ml	Percentage
0	18	14	77.8%
1	1	1	100%
2	1	1	100%
3	11	10	90.9%
4	9	8	88.9%

The presence of acne, particularly at higher grades (≥3), was associated with a greater proportion of patients having elevated AMH levels. This emphasizes a possible correlation between AMH and cutaneous manifestations of hyperandrogenism in PCOS.

Together, these results highlight that serum AMH correlates strongly with both biochemical (testosterone, DHEAS) and clinical (hirsutism, acne) markers of hyperandrogenism, with phenotypes involving oligo/anovulation and PCOM showing the highest AMH elevation.

Summary of Findings

- The PCOS group had significantly higher AMH, testosterone, DHEAS, hirsuitism and acne.
- High AMH levels were associated with clinical hyperandrogenism and biochemical androgenemia.
- These findings suggest that AMH may serve as a surrogate marker for diagnosis of PCOS and hyperandrogenism in PCOS.

Discussion

This observational and comparative study involved 40 women diagnosed with PCOS, (n = 40) and 40 women of non-PCOS group. The baseline demographic characteristics such as age and menstrual irregularities were comparable between the two groups. However, (BMI) was significantly higher in the PCOS group, suggesting a potential association between PCOS patients and increased adiposity.

The hormonal profiles revealed important differences between the two groups. Notably, serum AMH levels were significantly higher in the PCOS group compared to the non-PCOS group (7.56± 2.1 ng/mL vs. s 2.67± 1.1 ng/mL, $p < 0.0001$). Similarly, hirsuitism, acne, levels of serum total testosterone and DHEAS were also elevated in PCOS patients. AMH and hyperandrogenic status also showed a significantly positive association in our study, possibly due to increased antral follicle count and the stimulatory role of androgens on granulosa cell.

Furthermore, the modified Ferriman-Gallwey score and acne score revealed a statistically significant association between AMH levels and clinical indicators of hyperandrogenism, including hirsutism ($r = 0.54$, $p < 0.001$) and acne. These results support the idea that AMH may function as an indirect signal of hyperandrogenic activity in PCOS in addition to being a marker of ovarian reserve.

Overall, the findings shows that women with PCOS have high levels of AMH and hyperandrogenism. There is a robust positive association between high levels of AMH and both clinical and biochemical hyperandrogenism. This implies that AMH may be useful as a supportive diagnostic or monitoring biomarker for PCOS and hyperandrogenism in PCOS, especially in situations where testosterone assays might not be as readily available or trustworthy.

The relationship between AMH and hyperandrogenism in women with PCOS has been the subject of several recent research. Biochemical and clinical indicators of hyperandrogenism have been repeatedly linked to elevated AMH levels, which are a consequence of an increased number of antral follicles. Higher AMH values were shown to be substantially linked with total testosterone and DHEAS, especially in patients with marked hyperandrogenemia, according to research by Sahmay et al. in PCOS patients. This implies that AMH might represent the severity of the disease in addition to acting as a diagnostic biomarker [10].

Jamil et al. supported these findings in their cross-sectional analysis, where women with both clinical (acne, hirsutism) and biochemical hyperandrogenism had significantly elevated AMH levels compared to those without hyperandrogenism. They concluded that AMH could potentially be used as an adjunct marker when androgen assays yield inconclusive results [11].

Expanding upon this, Dewailly et al. proposed a deeper physiological role of AMH beyond diagnosis. They highlighted its inhibitory effect on aromatase expression in granulosa cells, which may reduce estradiol production and promote persistence of hyperandrogenic states in PCOS. Therefore, AMH may potentially contribute to the continuation of androgen excess [12].

Woo et al. evaluated the diagnostic capacity of AMH in identifying hyperandrogenic PCOS patients. They reported that AMH levels above 4.5 ng/mL had good sensitivity (78%) and specificity (84%) for detecting hyperandrogenemia. They emphasized that AMH can be particularly valuable

in settings with limited access to ultrasonography or hormonal testing [13].

In another recent study, Lee et al. analyzed AMH levels across different clinical phenotypes of PCOS and found that AMH was highest among women presenting with both hyperandrogenism and oligo/anovulation. The relationship between AMH and androgen excess was reinforced by the discovery of a strong positive connection between AMH and serum free testosterone and DHEAS [14]. All of these results point to the potential pathophysiological function of AMH in regulating androgen production and follicular growth, as well as its clinical significance as a surrogate marker of hyperandrogenism in PCOS.

References

1. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Hum Reprod. 2018; 33(9): 1602–18.
2. Lizneva D, Suturina L, Walker W, Brakta S, Gavrilova-Jordan L, Azziz R. Criteria, prevalence, and phenotypes of polycystic ovary syndrome. Fertil Steril. 2019;112(6):930–40.
3. Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Legro RS. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of PCOS. Endocr Rev. 2019; 36(5): 487–525.
4. Dewailly D, Laven J, Amosun O, Bouchard P. Role of anti-Müllerian hormone in the pathogenesis and diagnosis of polycystic ovary syndrome. Fertil Steril. 2020;114(1):69–81.
5. Sahmay S, Atakul N, Oncul M, Tuten A, Aydogan B. Serum AMH levels and ovarian morphology in women with PCOS. J Obstet Gynaecol Res. 2018;44(3):485–91.
6. Pigny P, Merlen E, Robert Y, Cortet-Rudelli C, Decanter C, Jonard S, et al. Elevated serum level of AMH in patients with PCOS: relationship to the ovarian follicle excess and to the follicular arrest. J Clin Endocrinol Metab. 2019;104(4):1374–84.
7. Catteau-Jonard S, Dewailly D. Pathophysiology of polycystic ovary syndrome: the role of hyperandrogenism. Front Horm Res. 2019; 53: 23–34.
8. Dilbaz B, Kalafat E. Prediction of hyperandrogenism using serum AMH in patients with PCOS. Gynecol Endocrinol. 2020;36(9):799–803.
9. Cimino I, Casoni F, Liu X, Messina A, Parkash J, Jamin SP, et al. Novel role for anti-Müllerian hormone in the regulation of GnRH neuron excitability and hormone secretion. Nat Commun. 2019;10:2224.
10. Sahmay S, Atakul N, Oncul M, Tuten A, Aydogan B. Serum AMH levels correlate with clinical signs of hyperandrogenism in PCOS. Gynecol Endocrinol. 2018;34(8):678–682.
11. Jamil AS, Alalaf SK, Al-Tawil NG, Mohammed MA. The correlation of serum anti-Müllerian hormone with clinical and biochemical markers of hyperandrogenism in PCOS. Int J Gynaecol Obstet. 2019; 146(3): 370–375.
12. Dewailly D, Hieronimus S, Mirakian P, Hugues JN. Role of Anti-Müllerian Hormone in Pathophysiology, Diagnosis and Treatment of PCOS: A Review. Reprod Biol Endocrinol. 2020;18:89.
13. Woo HY, Kim KH, Kang MJ, Kim SH. Diagnostic value of serum AMH levels in women with PCOS and hyperandrogenism. Endocr J. 2021;68(4):387–394.
14. Lee JY, Kim Y, Lee GH, Kim HK. Association between serum anti-Müllerian hormone levels and clinical phenotypes in PCOS. J Obstet Gynaecol Res. 2022;48(2):401–408.