

AMH Levels in Different Phenotypes of Infertile Women with PCOS: A Comparative Analysis

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a complex endocrine disorder presenting with variable features such as hyperandrogenism, oligo/anovulation, and polycystic ovarian morphology. Anti-Müllerian hormone (AMH), secreted by granulosa cells of preantral and small antral follicles, is consistently elevated in PCOS and serves as an essential marker of ovarian reserve. However, its role in differentiating PCOS phenotypes among infertile women remains inadequately defined.

Methods: This cross-sectional analytical study was conducted among 180 infertile women diagnosed with PCOS as per the Rotterdam criteria. Participants were categorized into four phenotypes based on the presence of hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM). Serum AMH levels were estimated using an enzyme-linked immunosorbent assay (ELISA). A comparative analysis of clinical, biochemical, and ultrasonographic parameters among phenotypes was conducted using ANOVA or the Kruskal–Wallis test for continuous variables and the chi-square test for categorical variables, with $p < 0.05$ deemed significant.

Results: All PCOS phenotypes showed significantly elevated mean AMH levels compared to controls, with the highest concentrations observed in Phenotype A (HA + OD + PCOM), followed by Phenotypes B, C, and D ($p < 0.001$). Higher AMH levels correlated with greater disease severity, including increased hyperandrogenism and antral follicle count.

Conclusion: Serum AMH levels vary across PCOS phenotypes and correlate with clinical and biochemical severity. Phenotype A exhibits the highest AMH values, reflecting greater ovarian reserve and follicular activity. AMH can serve as a valuable adjunct diagnostic marker to refine phenotypic classification and guide individualized fertility treatment strategies.

Key-words: Anti-Müllerian hormone, PCOS; Infertility, Phenotypes, Hyperandrogenism, Ovarian reserve

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with an estimated prevalence ranging from 6% to 20% depending on the diagnostic criteria used ^[1]. It is a multifactorial condition characterized by a combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (PCOM) on ultrasonography ^[2].

The wide heterogeneity in clinical and biochemical presentation has led to the recognition of distinct phenotypic subgroups within the syndrome. Based on the Rotterdam criteria, PCOS can be classified into four phenotypes according to the presence or absence of hyperandrogenism (HA), ovulatory dysfunction (OD), and PCOM ^[3]. This phenotypic diversity underscores the need for reliable biomarkers that can help delineate disease subtypes, assess severity, and guide individualized management strategies. Among candidate biomarkers, anti-Müllerian hormone has emerged as a promising marker of ovarian reserve and follicular activity ^[4]. AMH is secreted by granulosa cells of preantral and small antral follicles and plays a key role in inhibiting excessive follicular recruitment ^[5]. Elevated AMH concentrations in women with PCOS reflect an increased number of small

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antral follicles and are often associated with greater disease severity and hyperandrogenic features [6]. Infertility is a major clinical manifestation of PCOS, frequently requiring medical or assisted reproductive interventions for management [7]. While AMH is widely used in fertility practice to assess ovarian reserve, its potential utility in differentiating PCOS phenotypes among infertile women remains inadequately explored. Understanding the relationship between AMH levels and specific PCOS phenotypes could enable clinicians to develop more precise and individualized treatment protocols. In particular, phenotypes associated with markedly elevated AMH levels may require lower gonadotropin doses during ovarian stimulation to reduce the risk of ovarian hyperstimulation syndrome (OHSS) [8]. The present study was undertaken to evaluate differences in serum AMH concentrations across the four phenotypic subgroups of PCOS in infertile women. By correlating hormonal, clinical, and ultrasonographic parameters, this study aims to assess the diagnostic and prognostic value of AMH in phenotype-based disease stratification. Establishing AMH as a phenotype-specific biomarker could facilitate the development of more tailored fertility management strategies, optimizing outcomes while minimizing unnecessary medical interventions.

MATERIALS AND METHODS

This cross-sectional observational study was conducted over a period of 24 months (January 2021 to December 2022) in the Department of Reproductive Endocrinology and the Assisted Reproduction Unit of a tertiary care teaching hospital. The study aimed to compare serum anti-Müllerian hormone (AMH) levels across different phenotypes of infertile women diagnosed with polycystic ovary syndrome (PCOS). Ethical approval was obtained from the Institutional Ethics Committee, and written informed consent was secured from all participants before enrollment.

Study Population and Design- A total of 180 infertile women, aged 20–40 years, diagnosed with PCOS as per the Rotterdam criteria (2003), were enrolled. Diagnosis required at least two of the following criteria:

- (i) clinical or biochemical hyperandrogenism (HA),
- (ii) oligo- or anovulation (OD), and

(iii) polycystic ovarian morphology (PCOM) on ultrasonography.

Women with infertility due to other causes (tubal, uterine, or male factor), as well as those with thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, or Cushing's syndrome, were excluded from the study.

Phenotypic Classification is classified into four phenotypes as follows:

- Phenotype A (Classic PCOS): HA + OD + PCOM
- Phenotype B: HA + OD
- Phenotype C: HA + PCOM
- Phenotype D: OD + PCOM

Each participant underwent a detailed clinical assessment, including measurement of body mass index (BMI), waist-to-hip ratio (WHR), and modified Ferriman–Gallwey (mFG) score for hirsutism. Venous blood samples were collected during the early follicular phase (days 2–3) of the menstrual cycle. Serum levels of AMH, FSH, LH, and total testosterone were estimated using standard enzyme-linked immunosorbent assay (ELISA) and chemiluminescent immunoassay methods. All assays were performed in duplicate, and intra- and inter-assay coefficients of variation were maintained below 10%.

Transvaginal ultrasonography was performed by a single experienced sonographer using a 7.5 MHz transducer. Polycystic ovarian morphology was defined as the presence of ≥ 12 follicles (2–9 mm in diameter) and/or an ovarian volume $> 10 \text{ cm}^3$ in at least one ovary.

Statistical Analysis- Statistical analyses were performed using IBM SPSS Statistics (Version 25.0). Normality was assessed with the Shapiro–Wilk test. Normal data are expressed as mean $\pm$ SD, non-normal data as median (IQR), and categorical variables as frequency (percentage). Group comparisons among the four PCOS phenotypes used one-way ANOVA or Kruskal–Wallis tests, with Bonferroni correction where applicable. Chi-square or Fisher's exact tests were applied for categorical data. Pearson or Spearman correlations assessed associations. Multivariate linear regression identified independent predictors of serum AMH after adjusting for age and BMI. A p -value < 0.05 was considered significant. Non-parametric tests and ranges were used for small samples.

Ethical Approval- The study was approved by the Institutional Ethics Committee of King George's Medical University, Lucknow (Approval No: IEC/OBG/2020/951). All participants provided written informed consent before participation. The study adhered to the Declaration of Helsinki (2013 revision) for research involving human subjects.

RESULTS

A total of 180 infertile women with PCOS were analyzed, with 45 subjects in each phenotype group. The mean age was 28.6 ± 4.1 years, and the mean BMI was 27.2 ± 3.8 kg/m². Central obesity (waist–hip ratio >0.85) was present in 65% of participants. Baseline demographic and hormonal parameters are summarized in Table 1.

Table 1: Baseline characteristics of the study population

Variable	Mean $\pm$ SD
Age (years)	28.6 $\pm$ 4.1
BMI (kg/m ²)	27.2 $\pm$ 3.8
Waist-to-Hip Ratio	0.89 $\pm$ 0.07
Duration of Infertility (years)	3.2 $\pm$ 1.8
FSH (mIU/mL)	6.1 $\pm$ 1.3
LH (mIU/mL)	9.3 $\pm$ 2.5
Total Testosterone (ng/dL)	68.1 $\pm$ 21.4

Values are mean $\pm$ SD unless otherwise specified. BMI: body mass index; FSH: follicle-stimulating hormone; LH: luteinizing hormone. Sample size: $n=180$ ($n=45$ per phenotype).

Serum AMH levels differed significantly among the four PCOS phenotypes ($p<0.001$). Phenotype A (HA + OD + PCOM) demonstrated the highest mean AMH level (7.8 $\pm$ 1.9 ng/mL), followed by Phenotypes B, C, and D (Table 2, Fig. 1).

Table 2: AMH levels among PCOS phenotypes.

Phenotype	AMH (ng/mL)
A: HA + OD + PCOM	7.8 $\pm$ 1.9
B: HA + OD	6.5 $\pm$ 1.7
C: HA + PCOM	5.9 $\pm$ 1.6
D: OD + PCOM	5.2 $\pm$ 1.4

AMH: anti-Müllerian hormone; PCOM: polycystic ovarian morphology; HA: hyperandrogenism; OD: ovulatory dysfunction. p -value for between-group difference: $p<0.001$. Sample size: $n=45$ per phenotype.

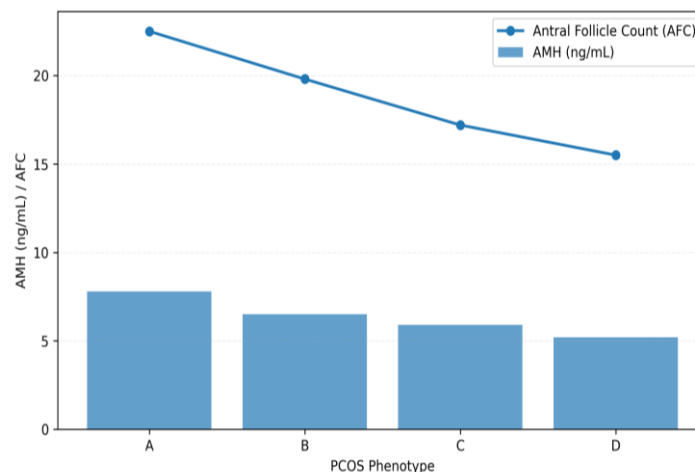


Fig. 1: Mean AMH and antral follicle count among PCOS phenotypes.

AMH levels showed a positive correlation with both the modified Ferriman–Gallwey (mFG) score ($r=0.49$, $p<0.01$) and total testosterone levels ($r=0.45$, $p<0.01$).

Phenotype A showed the most significant degree of hyperandrogenism and the highest LHs: FSH ratio compared with other groups (Table 3).

Table 3: Clinical and biochemical findings by phenotype.

Variables	Phenotype A	Phenotype B	Phenotype C	Phenotype D
mFG Score (mean±SD)	14.2±3.5	12.8±3.1	10.5±2.9	9.6±2.4
Total Testosterone (ng/dL)	78.3±22.4	72.1±20.2	65.7±21.9	60.3±19.8
LH: FSH Ratio	1.5±0.4	1.3±0.3	1.2±0.4	1.1±0.4

mFG: modified Ferriman–Gallwey score. Sample size: $n=45$ per phenotype. (Report exact p -values in manuscript text/tables.)

Transvaginal ultrasonography revealed the highest antral follicle count (AFC) in Phenotype A, averaging 22.5 ± 4.2 . This was followed by Phenotypes B, C, and D. Figure 1 illustrates the distribution of mean Anti-Müllerian Hormone (AMH) and AFC values among the four phenotypes. Among the 50 participants who underwent

controlled ovarian stimulation, those classified as Phenotype A required lower doses of gonadotropins but also experienced a higher incidence of ovarian hyperstimulation syndrome (OHSS). In contrast, Phenotypes C and D required higher gonadotropin doses for adequate follicular development. (Table 4, Fig. 2).

Table 4: Gonadotropin dose and ovulation induction response

Phenotypes	Avg Dose of FSH (IU)	Mature Follicles (mean±SD)	OHSS Incidence (%)
A	900±180	3.8±1.2	10
B	1050±210	3.0±1.1	6
C	1200±240	2.7±0.8	4
D	1350±260	2.4±0.9	2

OHSS: ovarian hyperstimulation syndrome. Avg doses reported as mean±SD. Sample for ovulation induction subset: $n=50$ (state phenotype-wise counts if available).

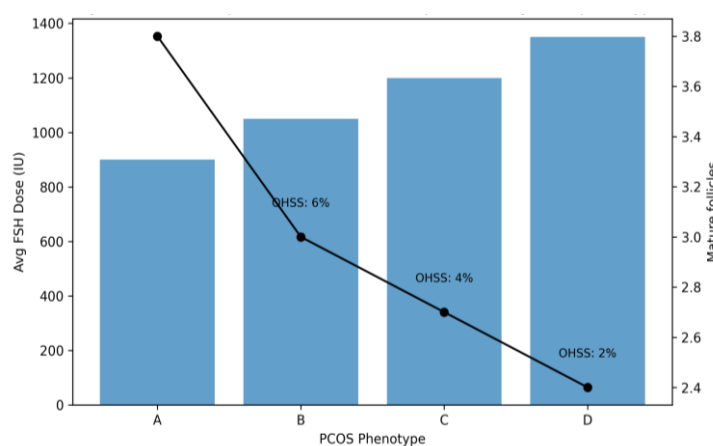


Fig. 2: Gonadotropin dose and follicular response among PCOS phenotypes.

After adjusting for age and BMI, phenotype classification remained independently associated with serum AMH levels ($\beta=0.32$, $p=0.001$). This indicates that AMH reflects the biological and clinical heterogeneity among PCOS subtypes and may serve as a marker of disease severity.

DISCUSSION

This study demonstrates significant variation in serum AMH concentrations across different PCOS phenotypes among infertile women, consistent with previous literature linking elevated AMH levels to increased

follicular activity and disease severity in PCOS [9]. The finding that Phenotype A (HA + OD + PCOM) exhibited the highest AMH concentrations aligns with prior reports associating this phenotype with more pronounced clinical manifestations, including hyperandrogenism, anovulation, and polycystic ovarian morphology [10]. The positive correlations observed among AMH levels, total testosterone, and modified Ferriman–Gallwey (mFG) scores further support the hypothesis that AMH may serve as a biomarker of the severity of hyperandrogenic states.

The relationship between AMH and androgen excess offers important insights into PCOS pathophysiology. Hyperandrogenism disrupts folliculogenesis, leading to the accumulation of small antral follicles that secrete AMH at higher levels [9,10]. Genetic and molecular determinants may jointly influence both androgen synthesis and AMH production pathways, suggesting that these hormones share common regulatory mechanisms. Understanding these molecular links could facilitate the identification of new therapeutic targets for restoring normal follicular development and ovulatory function [11]. Clinically, the heterogeneity among PCOS phenotypes has important implications for fertility management. Women with higher AMH levels—particularly those in Phenotype A—tend to exhibit robust ovarian responses to gonadotropin stimulation but are also at increased risk for ovarian hyperstimulation syndrome (OHSS) [12,13]. Conversely, phenotypes with lower AMH concentrations (C and D) may require higher gonadotropin doses to achieve adequate follicular maturation. Tailoring ovulation induction protocols according to AMH level and phenotype could therefore optimize treatment efficacy while minimizing complications [11,14].

These findings emphasize that PCOS is a varied disorder with differences in severity and reproductive potential. While the Rotterdam criteria guide diagnosis, measuring AMH levels can enhance phenotypic classification and inform personalized treatments, potentially improving assisted reproduction outcomes [11,15]. However, the study has limitations. Its cross-sectional design precludes causal inference, and the small sample size may limit generalizability. Larger, multicentric studies are needed to confirm AMH's role in different populations [16,17].

In summary, elevated AMH levels in Phenotype A reflect a more pronounced hyperandrogenic and follicular profile, underscoring its potential as a surrogate marker

of disease severity in PCOS [12,17]. Recognition of phenotypic variation based on AMH concentration enables a more personalized approach to fertility management, enhancing clinical outcomes and reducing the risks of overstimulation [17].

CONCLUSIONS

Serum anti-Müllerian hormone (AMH) levels showed significant variation among the four phenotypes of polycystic ovary syndrome (PCOS) in infertile women. The highest AMH concentrations were found in Phenotype A (HA + OD + PCOM), reflecting greater disease severity and hyperandrogenic activity. These results underscore the importance of AMH as an adjunct biomarker for phenotypic classification and for predicting ovarian response in PCOS. Incorporating AMH assessment into fertility evaluation can support individualized treatment strategies, optimize ovulation induction, and improve clinical outcomes.

Future multicentric studies are crucial to confirm anti-Müllerian hormone (AMH) as a predictor of reproductive outcomes and metabolic risks in polycystic ovary syndrome (PCOS). AMH-guided stimulation protocols may customize gonadotropin dosing and lower the risk of ovarian hyperstimulation syndrome. Further research into the genetic and molecular pathways of AMH could enhance our understanding of PCOS and help develop targeted therapies.

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