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Research Article

Diagnostic Utility Of Anti-Müllerian Hormone (AMH) In Polycystic Ovary Syndrome (PCOS): A Hospital Based Case Control Study.

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Abstract: Background: Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women, presenting with several possible combinations of signs and symptoms and a range of phenotypes, which may include reproductive, endocrine, and metabolic alterations. Anti-Müllerian hormone (AMH) is an important regulator of folliculogenesis in the ovaries. The levels of AMH could be altered in PCOS, and may have diagnostic value. Objectives: To evaluate the diagnostic utility of anti-Müllerian hormone (AMH) in identifying polycystic ovary syndrome (PCOS) and across different PCOS phenotypes. ***Materials and Methods***: This hospital-based case–control study included 60 women with PCOS, diagnosed using the Rotterdam 2013 criteria, and 30 age-matched healthy controls. Clinical assessment, anthropometry, biochemical profiling, hormonal assays, and pelvic ultrasonography were performed. Serum AMH levels were measured using ELISA. Insulin resistance was calculated using HOMA-IR, and free androgen index (FAI) was derived. PCOS cases were classified into phenotypes A–D. Statistical analyses included Mann–Whitney U test, Chi-square test, Kruskal–Walli's test, Spearman correlation, and ROC curve analysis. ***Results***: Women with PCOS showed significantly higher prevalence of hyperandrogenism (90%), ovulatory dysfunction (80%), and polycystic ovarian morphology (70%). Obesity and metabolic abnormalities, including dyslipidaemia and IR (HOMA-IR ≥2 in 68.3%), were significantly more common in the PCOS group ($p<0.001$). AMH levels were markedly elevated in PCOS (median 8.23 ng/mL) compared with controls, with the highest levels observed in phenotype A. AMH demonstrated excellent diagnostic performance, with an optimal cut-off of 3.59 ng/mL yielding 95% sensitivity and 96.7% specificity (AUC=0.988). AMH showed a strong positive correlation with IR ($\rho=0.638$, $p<0.001$) and a moderate correlation with serum testosterone ($\rho=0.398$, $p=0.002$). ***Conclusion***: AMH is a highly accurate biomarker for diagnosing PCOS, with significant correlations to hyperandrogenism and insulin resistance. AMH levels vary across PCOS phenotypes, being highest in phenotype A. A cut-off of 3.59 ng/mL is clinically useful for diagnosing PCOS in the studied population.

Keywords: Anti-Müllerian Hormone; Hyperandrogenism; Insulin Resistance; PCOS; Running Title: AMH in PCOS Diagnosis.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women, presenting with several possible combinations of signs and symptoms and a range of phenotypes, which may include reproductive, endocrine, and metabolic alterations. PCOS is characterized by hypothalamic–pituitary–ovarian axis dysfunction and anovulation but, unlike other causes of ovulatory failure that feature insufficient ovarian follicle growth or suppressed gonadotropin secretion (or both), PCOS typically includes androgen excess and subtle alterations (not detected by routine tests) in serum levels of gonadotropins and estrogens. PCOS has the potential for serious consequences, including increased risk for the development of endometrial hyperplasia and neoplasia. Furthermore, extra-reproductive manifestations of PCOS include insulin resistance (IR), metabolic syndrome (MS), and low-grade chronic inflammation.1 Based on the limited data, the prevalence of PCOS in India ranges from 3.7 to 22.5%.2 The clinical manifestations of PCOS include Oligoamenorrhoea, hirsutism, excessive acne and hair loss. The metabolic consequences include impaired glucose tolerance, type 2 diabetes, obesity and increased risk of cardiovascular diseases. Clinicians now have these three sets of criteria to choose from, though the Rotterdam criteria3 are found to be more preferred.

Anti-Müllerian hormone (AMH) is an important regulator of folliculogenesis in the ovaries.4 It is secreted by granulosa cells of the ovarian follicles and its serum levels are elevated two to three fold in women with PCOS in comparison with normo-ovulatory women, consistent with the increased number of small antral follicles in PCOS.5,6 However, it is unclear whether AMH is simply a marker which is increased in PCOS, or actually an important contributing factor to its pathophysiology. Some

researchers have recently found a relationship between insulin resistance and AMH, and more recently between antral follicles and free androgen index (FAI).7 Studies concluded that obesity, insulin resistance, and Hyperandrogenism play major roles in the increasing level of AMH.8 However, a significant relationship between AMH and androgens could not be found. This study aimed to evaluate the diagnostic utility of AMH in the diagnosis of PCOS and to correlate find the correlation between AMH and Hyperandrogenism as well as insulin resistance in patients with PCOS.

MATERIALS AND METHODS

The present hospital-based case control study (N=90) was conducted in the Department of Endocrinology, S. Nijalingappa Medical College and H.S.K. hospital for a duration of one year. All the eligible women presenting clinically diagnosed PCOS based on Rotterdam 2013 criteria, during the study period were enrolled and 30 apparently healthy females were selected as controls. As controls, apparently healthy females without endometriosis, cysts, or other ovarian gynecological disorders, with regular menstrual cycles (26–35days) without any endocrine abnormalities and with morphologically normal ovaries detected on ultrasound examination were included. Women with non-classical Congenital adrenal hyperplasia, Cushing's syndrome, thyroid dysfunction, hyperprolactinemia, and androgen-producing tumors patients were excluded from the study Patients at presentation receiving medications that could alter the endocrine and metabolic parameters under investigation for the above disorder s were excluded from the study. Prior to the commencement, ethical clearance was obtained for the study from the Institutional Human Ethics Committee, S. Nijalingappa Medical College and H.S.K. hospital, Bagalkot, Karnataka. The selected women were briefed about the nature of the study and a written informed consent was obtained from the selected women prior to the enrolment.

Data collection

Women were interviewed for demographic and clinical presentation were noted along with detailed medical and obstetric history. Anthropometry measurements including BMI, waist to hip ratio (WHR), vitals, systemic examination and clinical signs including acne, acanthosis nigricans, skin tag and hirsutism.

Clinical and biochemical tests were conducted using standard assays. A total of 8 ml was withdrawn and put equally in 2 plain vials on day 2–3 of menses or after withdrawal bleeding. Samples were then centrifuged at 3000 rpm in centrifugation machine at the biochemistry laboratory for serum analysis. One vial of centrifuged sample was stored at - 80 degrees in deep freezer for batch analysis of AMH. Analysis for AMH levels was done using Serolisa Human AMH ELISA Kit. ELISA for the quantitative measurement of human AMH level in serum or plasma (Ref KT-804).9 -The other vial was used to test for additional parameters as follows: Fasting Blood Sugars (OGTT) and Fasting Insulin, were estimated using standard biochemical tests. Hormonal assays for T3, T4 and TSH, FSH, LH, prolactin and total testosterone was performed using chemiluminescence immunoassay on the ECiQvitros from Johnson's and Johnson's. TAS was performed for all the women using 3 MHz Medison model—SONACE X1 to diagnose PCOM. Estimation of SBHG levels was done using Diametra direct immunoenzymatic colorimetric method for the quantitative determination of SHBG concentration in human serum or plasma (Ref DK087).10 Menstrual irregularity11, Hirsutism12, clinical Hyperandrogenism (modified Ferriman-Gallwey score ≥8), and biochemical Hyperandrogenism by increased FT24 were used for diagnosis of PCOS.

Free androgen index (FAI)= total testosterone *100/SHBG. HOMA-IR were calculated.
Imaging of USG pelvis was performed and the observations were documented.
Patients were classified into four PCOS phenotypes as below.

Statistical analysis

The data obtained was coded and entered into Microsoft Excel Worksheet (Annexure III). The data was analyzed using statistical software SPSS version 20.0. Continuous variables were presented as mean ± standard deviation (SD) or median and interquartile (IQR) range and analyzed for normality by the Shapiro-Wilk test. Categorical variables were compared using the Chi-square or Fisher's exact test while continuous variables were compared using Mann-Whitney U tests. The continuous data pertaining to different PCOS phenotypes was compared using Kruskal Wallis test. To determine cut off value of AMH in predicting PCOS was by determined by receiver operating characteristic curve, sensitivity, specificity, positive predictive value (PPV) negative predictive value (NPV), All tests were two- tailed and a p-value of less than 0.05 was considered significant. All tests were two-tailed and a p-value of less than 0.05 was considered significant.

RESULTS

Patients were classified into four PCOS phenotypes as below.[3,13]

	Phenotype A	Phenotype B	Phenotype C	Phenotype D
Hyperandrogenism and hirsutism	Present	Present	Present	Absent
Ovulatory dysfunction	Present	Present	Absent	Present
Polycystic ovarian morphology	Present	Absent	Present	Present

Figure 1. Classification of PCOS phenotypes. Phenotypes A to D according to the presence or absence of hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology (Rotterdam criteria).

A total of 90 participants that is, 60 patients with PCOS (Cases) and 30 apparently healthy females (Controls) were studied.

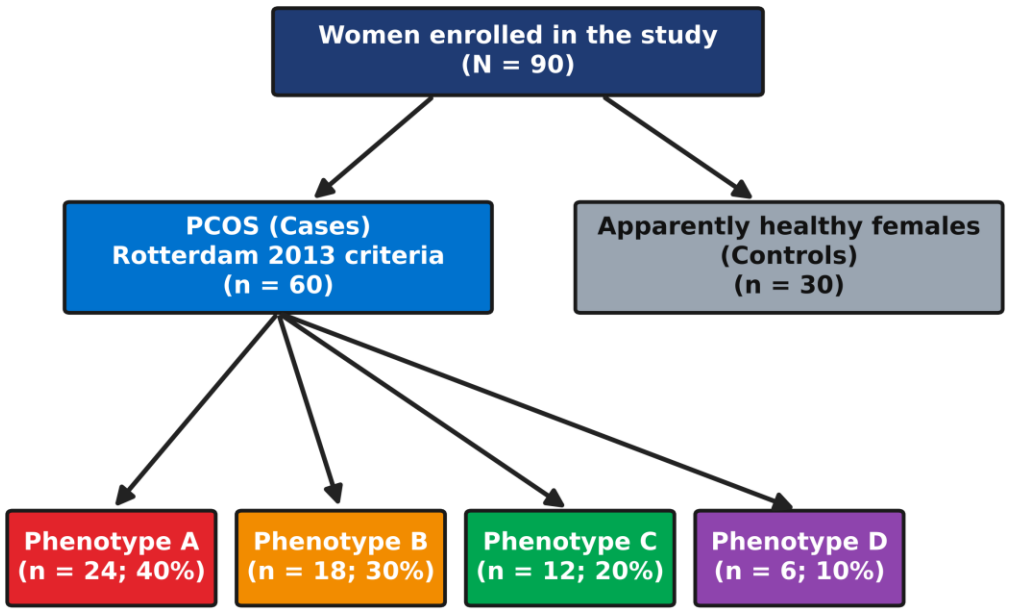


Figure 2. Flow diagram of study participants. A total of 90 women were studied: 60 women with PCOS diagnosed by the Rotterdam 2013 criteria (cases) and 30 apparently healthy females (controls). Cases were classified into phenotypes A to D.

The comparison of clinical characteristics of the women with PCOS and women in control group is as depicted in table 2. About one-third of the PCOS women were between 21 to 25 years of age or obese. Most common clinical features were hyperandrogenism (90%) followed by irregular cycles and ovulatory dysfunction (80% each) and USG morphology of PCOS (70%). Prevalence of obesity and waist circumference of ≥ 80 cm was significantly higher (P<0.001) in PCOS women compared to controls.

Table 1. Clinical profile of the patients with PCOS

	Case (N=60)	**Control (N=30)**	**p-value**
Age in years distribution, N(%)			
18 to 20	11 (18.3)	7 (23.3)	0.865
21 to 25	22 (36.7)	11 (36.7)	
26 to 30	24 (40.0)	10 (33.3)	
31 to 35	3 (5.0)	2 (6.7)	
BMI			
< 18.50	0 (0)	6 (20.0)	<0.001
18.50 to 22.99	11 (18.3)	12 (40.0)	
23.00 to 24.99	5 (8.3)	3 (10.0)	
25.00 to 29.99	27 (45.0)	7 (23.3)	
≥ 30	17 (28.3)	2 (6.7)	
WC			

≥ 80	52 (86.7)	11 (36.7)	<0.001
< 80	8 (13.3)	19 (63.3	
WHR			
≥ 0.81	51 (85.0)	10 (33.3)	<0.001
< 0.81	9 (15.0)	20 (66.7)	

Table 2. Comparison of clinical characteristics of the study population

Parameters					
	Median	**IQR**	**Median**	**IQR**	p
Age (Years)	25.00	6.00	23.50	6.50	0.447
No. of cycles per year	4.00	4.00	12.00	0.00	**<0.001**
Length of cycle (days)	70.00	45.00	30.00	2.00	**<0.001**
Age at menarche (Years)	13.00	2.00	13.00	2.00	0.268
Hirsutism (FG score)	6.00	4.00	-	-	**-**
Height (Cms)	153.00	8.75	154.50	9.50	0.387
Weight (Kg)	63.50	18.75	52.00	14.00	**<0.001**
Body mass index (Kg/m^2)	27.45	6.33	21.90	6.35	**<0.001**
Waist circumference(cm)	87.50	17.00	78.00	8.25	**<0.001**
Waist hip ratio	0.90	0.10	0.79	0.05	**<0.001**
Pulse rate (/Minute)	83.00	6.00	84.00	6.00	0.367
Systolic blood pressure (mm Hg)	110.00	8.00	110.00	6.50	0.972
Diastolic blood pressure (mm Hg)	80.00	10.00	80.00	4.50	0.790
Antimullerian hormone (ng/ml)	6.80	6.60	2.04	1.02	**<0.001**
Fasting blood sugar (mg/dL)	86.00	25.00	82.00	8.50	0.054
Oral glucose tolerance test (2hrs)	119.00	32.50	111.00	16.50	**0.043**
Fasting insulin (microU/ml)	18.00	23.80	3.40	1.33	**<0.001**
HOMA IR	3.68	5.11	0.72	0.33	**<0.001**
McAuley index	17.25	23.84	2.80	1.34	**<0.001**
Serum Testosterone (Fasting) ng/dl	57.32	22.03	12.03	3.98	**<0.001**
Serum Testosterone (nmol/l)	1.99	0.76	0.42	0.14	**<0.001**
SHBG (nmol/L)	20.51	18.58	116.00	13.75	**<0.001**
Free androgen index (FAI)	10.08	9.07	0.36	0.11	**<0.001**
Serum TSH (microU/ml)	3.22	1.88	3.70	1.42	**0.045**
Serum Prolactin (ng/ml)	10.00	8.42	10.80	4.48	0.258
Total cholesterol (mg/dL)	184.00	61.25	118.00	11.25	**<0.001**
High density lipoprotein (mg/dL)	43.00	10.75	68.00	9.00	**<0.001**
Low density lipoprotein (mg/dL)	103.50	43.75	80.00	10.50	**<0.001**
Triglycerides (mg/dL)	180.00	50.00	127.00	12.50	**<0.001**
Triglycerides (mmol/L)	10.00	2.78	7.06	0.69	**<0.001**

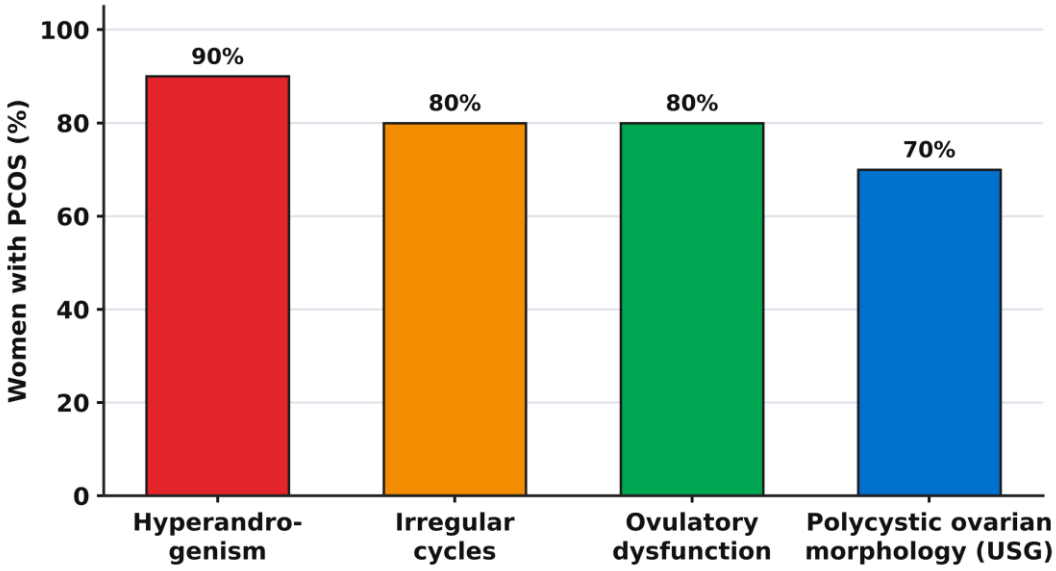


Figure 3. Prevalence of diagnostic features among women with PCOS (n = 60). Hyperandrogenism was the most common feature, followed by irregular cycles, ovulatory dysfunction and polycystic ovarian morphology on ultrasonography.

A significantly higher number of women with PCOS had fasting blood sugar levels between 100 to 125 mg/dL (20%) and ≥126 mg/dL (5%), OGTT levels between 140 to 199 mg/dL (15%) and ≥ 200 mg/dL (6.67%). Similarly, HOMA IR ≥2 (68.33%), total cholesterol ≥200 mg/dL (46.67%) HDL ≤50 mg/dL (63.33%) LDL ≥100 mg/dL (53.33%) was observed only among women with PCOS. Further triglyceride levels of ≥150 mg/dL were also significantly high in women with PCOS compared to women in control group (73.33% vs 13.33%). (p<0.001).

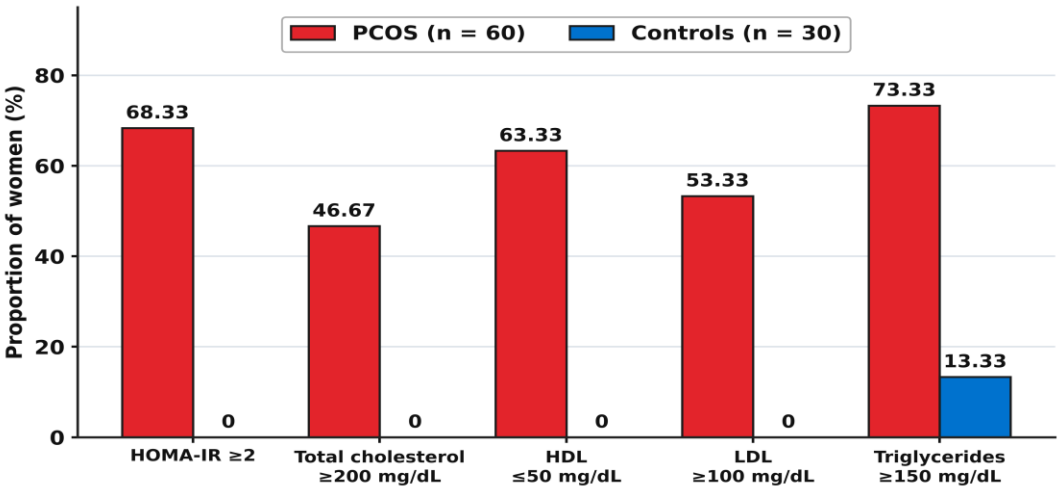


Figure 4. Metabolic abnormalities in women with PCOS and controls. Proportion of women with HOMA-IR ≥2, total cholesterol ≥200 mg/dL, HDL ≤50 mg/dL, LDL ≥100 mg/dL and triglycerides ≥150 mg/dL in the PCOS (n = 60) and control (n = 30) groups (p<0.001).

In the present study the common PCOS phenotype was phenotype A (40%) followed by B (30%), C (20%) and D (10%). The median AMH levels was significantly higher in phenotype A (12 [6.97 IQR] ng/mL) compared to other phenotypes. The comparison of clinical characteristics in different PCOS phenotypes is as depicted in table 3

Table 3: AMH levels in PCOS phenotypes.

		AMH (ng/mL)	
PCOS phenotypes	**N (%)**	**Median**	**IQR**
Phenotype A (Classic PCOS)	24 (40)	12.00	6.97
Phenotype B (Non PCOD PCOS)	18 (30)	6.25	0.98
Phenotype C (Ovulatory PCOS)	12 (20)	4.11	1.05
Phenotype D (Non-Hyperandrogenic PCOS)	6 (10)	4	1.83

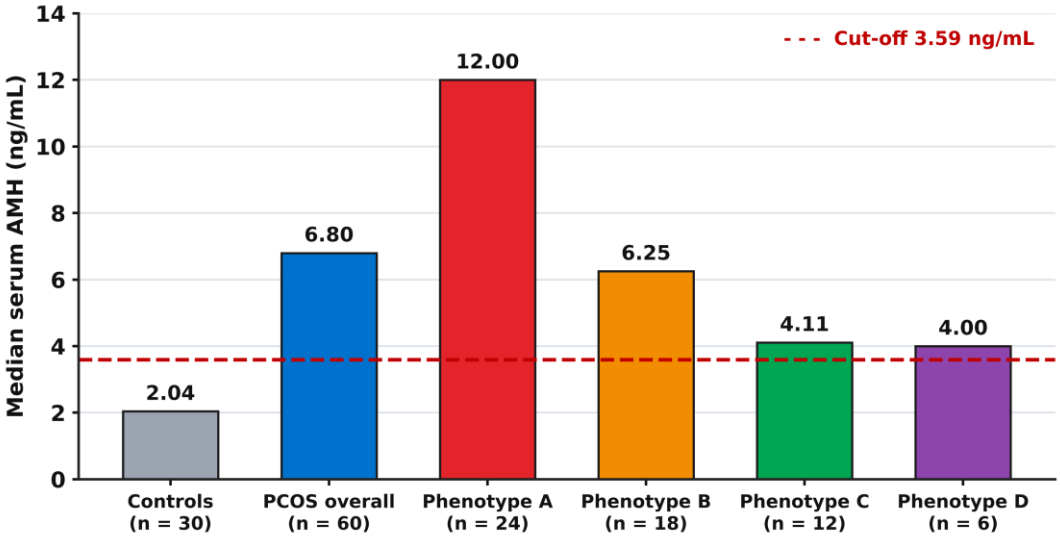


Figure 5. Median serum AMH levels in controls, overall PCOS and PCOS phenotypes. AMH was highest in phenotype A, followed by phenotype B, with similar lower levels in phenotypes C and D. The dashed line marks the AMH cut-off of 3.59 ng/mL.

The Area under the receiver operating characteristics curve in predicting AMH cut-off indicated a value of 3.59 ng/mL yielded maximum sensitivity (95%) and specificity (96.66%) with AUC=0.988; SE=0.008; p<0.001; 95% CI=0.972 to 1.000. In the present study PCOS was diagnosed in 60 cases. Of them, significantly higher number of women had AMH levels of ≥ 3.59 ng/mL (95%). Accuracy of predicting PCOS and the different phenotypes are depicted in table --. There is a 95% sensitivity of predicting overall PCOS and 100% for phenotypes A and B. Only 3 cases of CPOS phenotypes C and D could not be predicted. (Table 4)

Table 4 : Accuracy of AMH in predicting PCOS and all phenotypes of PCOS

PCOS and types	AMH ≥ 3.59ng/ml n/N	sensitivity	specificity
Overall	57/60	95	96.67
Phenotype A	24/18	100	48.48
Phenotype B	18/18	100	44.44
Phenotype C	11/12	91.67	39.74
Phenotype D	4/6	66.67	35.71

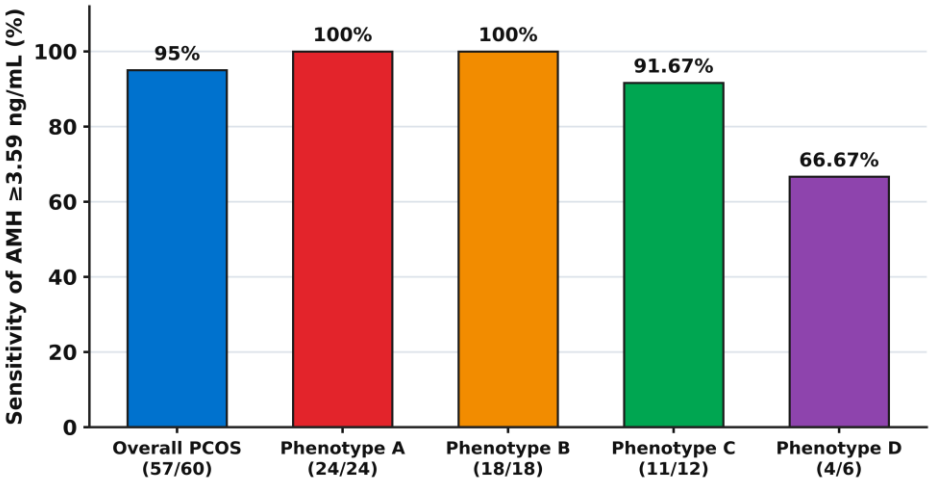


Figure 6. Sensitivity of AMH ≥3.59 ng/mL in predicting PCOS and its phenotypes. Values in parentheses are the number of women with AMH ≥3.59 ng/mL out of the total in each group (AUC = 0.988; overall specificity 96.67%).

DISCUSSION

Anti-Müllerian hormone is an important regulator of folliculogenesis in the ovaries.4 Our study findings suggest that AMH with a cut-off value of 3.59 ng/mL in highly accurate in discriminating PCOS among the women presenting with features of PCOS. There is significant strong linear relationship between AMH with hyperandrogenism as well as insulin resistance in women with PCOS. However, it is unclear whether AMH is simply a marker which is increased in PCOS, or actually an important contributing factor to its pathophysiology. The pathogenesis of PCOS and the exact mechanism responsible for elevated AMH levels in PCOS remains poorly understood. Studies concluded that obesity, insulin resistance, and hyperandrogenism play major roles in the increasing level of AMH.7 Further there is, paucity of data on the use of AMH for diagnosis of PCOS in women of Indian ethnicity.

In our study population with PCOS, the common feature was hyperandrogenism (90%) and the mean fasting testosterone levels of 61.52±26.30 ng/dL. The second common criterion were irregular cycles (80%) with average 6.00±2.76 cycles per year and mean length of cycle being 71.85±33.22 days, and ovulatory dysfunction (80% each). Finally, USG morphology of PCOS was present in 70% of the women.

The mean AMH levels in women with PCOS in our study population was 8.23±4.33 ng/mL (Range: 3.06 - 18.10 ng/mL). There was a significant (P<0.001) three-fold increase in the median AMH levels in women with PCOS compared to the apparently healthy population control. This effect was much more pronounced (six-fold increase) in the phenotype A of PCOS. Phenotype B had a threefold and phenotype C and D indicated a two-fold rise of AMH. Earlier observations by Saxena U. et al.14 (2018) Sahmay S. et al.15 (2014) among few others have also reported that AMH levels were found to be two to three times elevated in PCOS women compared to controls.5,6,16,17

Various studies that considered AMH as diagnostic tool for PCOS reported a cut-off value int eh range of 2.8 to 7.8.18-23 with sensitivity and specificity ranging between 53-95% and 69-98%, respectively. In the present study, the diagnostic potential of AMH were determined by ROC curve and ROC curve yielded with a cut off value of 3.59 ng/mL for AMH with maximum sensitivity (95%) and specificity (96.66%). For the cut-off values of AMH ≥ 3.59 ng/mL, the accuracy varies with the phenotype of PCOS. While all the women with PCOS type A and B had AMH levels above the cut-off, majority with the C and D type had AMH levels above the cut-off value. Similar to our results, studies by Saxena U. et al.14 (2018) Saikumar P. et al.24(2013) reported the best diagnostic potential of AMH at cut-off of 3.44 and 3.34 ng/ml, respectively. Dewailly D et al.19 (2011) observed a higher sensitivity and specificity of 92% and 97%, respectively, at a cut-off of 4.9 ng/ml and concluded that AMH not only reflects AFC but also the degree of hyperandrogenism making AMH a better marker than follicle numbers per ovary. In general, due to the metabolic abnormalities, PCOS women tend to be obese with higher central waist to hip ratio. Obesity is considered as one of the most important features of PCOS and its prevalence in diseased women varies between 61 and 76%. Many studies25 explain that females with PCOS have increased visceral and subcutaneous body fat distribution due to increased androgen production rates;26 this central obesity follows a masculinized body fat distribution27 where the amount of visceral fat correlates with the degree of insulin resistance.28 In our study, the prevalence of obesity (BMI ≥ 30 Kg/m2) and central obesity with a waist circumference of ≥ 80 cm was 28% and 87%, respectively. These estimates are significantly high (P<0.01) among PCOS women compared to control group.

In line with the previous observations, insulin resistance was also significantly higher number of women in with PCOS (HOMA IR ≥2 68.33%; p<0.001), compared to control group. FBS and OFTT levels indicate that among PCOS women, about 15-20% and 5% were in the state of pre-diabetes or diabetes, respectively. A few biomarkers have been used to detect insulin resistance in PCOS women. For instance, insulin restrains the release of sex hormone binding globulin (SHBG) from the liver and the production of insulin-like growth factor binding protein 1 (IGFBP-1). It also affects the homeostatic model assessment (HOMA-IR) which is based on computations of fasting glucose and levels of insulin.25 In the present study the median HOMA IR (3.68 [IQR 5.11] vs 0.72 [IQR 0.33]; p<0.001) was significantly elevated in women with PCOS compared to controls on the other hand, SHBG (20.51 [IQR 18.58] vs 116 [IQR 13.75] nmol/L; p<0.001) was significantly elevated in women with PCOS compared to controls. There was significant strong positive correlation between HOMA IR and AMH (ρ=0.638; R2=0.4276 p<0.001). Another important finding of the present study was that there was significant moderate positive correlation between serum testosterone levels and AMH (r=0.398; R2=0.1694; p=0.002) which further confirms the linear relation between AMH and serum testosterone levels which is the key component in the diagnosis of PCOS.

The findings of the present study are partly in agreement with the previous studies25,29,30 which report worsened cardiovascular profile and associated complications in women with PCOS. However, obesity may not be the independent predictor of all these features since these abnormalities are also observed among lean women with PCOS. Whether obesity leads to PCOS or whether PCOS leads to obesity is debatable.31

Overall, the present study shows that, AMH with a cut-off value of 3.59 ng/mL in highly accurate in discriminating PCOS among the women presenting with features of PCOS. There is significant strong linear relationship between AMH with hyperandrogenism as well as insulin resistance in women with PCOS. Further, women with PCOS are at significant risk of developing insulin resistance, diabetes mellitus and dyslipidemia. However these observations require further validation in larger studies.

The strength of this study was that it focused on PCOS subtypes and the relationship of AMH with IR, Hyperandrogenism and lipid abnormalities.

Limitations

Although the present study explored various determinants and attributes of PCOS quantitatively as well as qualitatively, it was derived from a subset of women presenting from urban locality in from to a single center involving relatively smaller sample size. Also the distribution of subgroup analysis was biased due to heterogeneity in the sample size. Several other parameters including hypertension, family history of diabetes mellitus and infertility were not considered.

Recommendations

Further multicentric studies involving large sample size with subgroup standardized population covering the other determinants including hypertension, family history of diabetes mellitus and infertility may extend the usefulness of AMH in the diagnosis of PCOS and predicting the outcomes with respect to fertility.

CONCLUSION

There is significant strong linear relationship between AMH with hyperandrogenism as well as insulin resistance in women with PCOS. It significantly varies with PCOS phenotypes. Phenotype A having the highest levels followed by phenotype B while phenotypes C & D have similar lower levels. An AMH with a cut-off value of 3.59 ng/mL in highly accurate in the diagnosis of PCOS

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Conflict of Interest

The authors declare no conflict of interest.

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