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Assessment of the Effect of Age and Body Mass Index on Anti-Mullerian Hormone Level in Iraqi Women with Polycystic Ovary Syndrome

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Abstract

In women with polycystic ovary syndrome (PCOS), the serum level of anti-Müllerian hormone (AMH) and its relation with body mass index (BMI) and age have become a subject of debate in many studies, and the results data were conflicting. Recent clinical guidelines underscore the increasing importance of AMH in the diagnosis of PCOS, proposing its inclusion as a formal diagnostic criterion. The study aimed to evaluate the effect of age and BMI on the serum level of AMH in PCOS patients. The bioethics committee approved the conduct of the study. The study included 108 infertile women diagnosed with PCOS and 63 infertile women without PCOS based on the Rotterdam criteria. The serum was isolated from the blood samples, and then the enzyme-linked immunosorbent (ELISA) technique was used to measure the serum level of AMH. Age and BMI information were recorded at the time the blood samples were taken. The mean of AMH level in women with PCOS was (8.1 ± 3.55 SD) ng/ml, which showed a significant difference ($p<0.05$) from (1.66 ± 1.59 SD) ng/ml in women without PCOS. The BMI was not significantly difference among the two groups, and it showed no significant correlation with AMH. The age of the PCOS group was younger (27.4 ± 4.7 SD) years than that of the non-PCOS control group (29.15 ± 6.22 SD) years. In addition, the age does not correlate with AMH significantly. The ROC curve highlights AMH as a highly reliable biomarker for PCOS diagnosis, whereas BMI and Age alone provide limited diagnostic value. PCOS is a disorder of younger reproductive-age women, with diagnosis clustering in the 20–29 age group. Women with PCOS, in general, tend to have higher values of BMI. AMH emerges as the most appropriate biomarker, which remains elevated in PCOS regardless of BMI or age, as they can be considered as contextual factors for management and prognosis of PCOS.

Keywords: Polycystic Ovary Syndrome, anti-Müllerian hormone, Body mass index, ELISA

Introduction

In women, polycystic ovary syndrome (PCOS) represents a common multidisciplinary disorder that affects women of reproductive age. About 10–15% of women can be affected by this condition in their reproductive years [1]. Significant challenges arise from the PCOS condition, such as reproductive health, dermatological conditions and psychological effects for women of reproductive age [2]. Also, PCOS is associated strongly with metabolic disturbances, like obesity and insulin resistance, which result in further pathophysiology and clinical outcomes, disease complication [3]. The most widely used standard for PCOS diagnosis is the Rotterdam criteria [4]. Anti-Müllerian hormone (AMH) is a glycoprotein secreted by the granulosa cells located in growing follicles [5]. It is commonly used to evaluate ovarian function and reserve [6]. Dysregulated AMH secretion has been linked to abnormal folliculogenesis [7]. A strong association between PCOS and AMH levels and PCOS has been reported by multiple studies, which led some experts to suggest incorporating of AMH into PCOS diagnostic criteria [8]. Recent studies suggest a direct role of AMH in PCOS pathogenesis by participating in chronic anovulation and follicular arrest [9]. The AMH levels in women correlate negatively with age and after menopause become undetectable. The peak of AMH levels is assumed to be reached in the ages of women age between 21 and 30 years [10]. Although of progressively declines of AMH with age progression, women with PCOS tend to maintain higher AMH levels in comparison with age-matched controls due to a greater pool of small follicles [3]. Body mass index (BMI), particularly in PCOS, represents an additional critical factor which can influence AMH levels, where the obesity is very prevalent. Obesity negatively affects the reproductive function by many mechanisms, such as hyperinsulinemia, insulin resistance and chronic low-grade inflammation [11]. A negative association of BMI with AMH have reported by recent studies, suggesting that reduction of AMH secretion and granulosa cell function may be impaired by adiposity [12]. Nevertheless, other studies have reported no significant association between BMI and AMH, indicating that AMH level be affected by other metabolic and hormonal factors [13]. Furthermore, the interaction between PCOS, BMI and Age in determining AMH levels has been highlighted by emerging research. These findings propose that the combination assessment of these variables may give a more precise evaluation of ovarian reserve than evaluating them independently [14]. Therefore, evaluating the effect of both BMI and age on AMH levels is important for clinical interpretation improvement and management of infertility in women with PCOS. Therefore, this study aims to assess the effect of BMI and age on serum AMH levels in infertile women with PCOS.

Materials and Methods

Study design and sample collection:

According to the Rotterdam criteria for PCOS, 108 Iraqi infertile women with PCOS and 63 infertile women without PCOS were chosen to conduct a cross-sectional analytical study to evaluate the effect of BMI and age on serum AMH levels in infertile women diagnosed with PCOS. Blood samples were collected from patients aged between 18 and 39 years old. All of the samples were collected from Kamal Al-Samarrai Hospital for Infertility Treatment and IVF, Baghdad/ Iraq, during the period from September 2025 to March 2026. Venous blood was obtained from patients on days 2-3 of the menstrual cycle. Ethical consent was obtained from the scientific committee of the College of Biotechnology / Al-Qasim Green University and from the Ministry of Health (9495 on 6/٠٨/2025). Before taking the blood samples, all of the participants were informed about the aims of this study, and their participation agreement was obtained. All of the participants have a history of infertility (≥1 year) and did not receive hormonal therapy within the last 3 months. In addition, the exclusion criteria included women with endometriosis, thyroid disorders, hyperprolactinemia, premature ovarian insufficiency, smoking status and current use of metformin or other insulin-sensitizing agents. The serum was isolated from the blood samples, and then the enzyme-linked immunosorbent assay (ELISA) technique was used to measure the serum level of AMH. Age and BMI information were recorded at the time the blood samples were taken. To gain a better understanding of the role of age and BMI in changing AMH levels, patients were divided into age groups with a 5-year difference between each group. Regarding the BMI, the groups were further divided according to the WHO classification.

Statistical analysis

A priori power analysis was conducted using G*Power to determine the adequacy of the study sample. Statistical analysis was performed using IBM SPSS Statistics and Microsoft Excel. Mean ± standard deviation was used to express data. The Shapiro–Wilk test was used to assess data normality. Differences between groups were analysed using the Mann–Whitney U test. Correlations between variables were assessed using Spearman correlation. Diagnostic performance was evaluated using the Receiver Operating Characteristic Curve. A p-value ≤0.05 was considered statistically significant.

Results and Discussion

In PCOS patients, there is a conflicting result of the BMI and age effect on AMH level. Although BMI appears to independently modulate AMH levels, aside from age and related parameters. Patients with central obesity demonstrate poorer ovarian function compared to their non-centrally obese counterparts [15].

The data normality assessment of AMH, BMI and age data was done by using the Shapiro–Wilk test as shown in Table (1). Most variables fail to meet the assumption of normality, suggesting the need for nonparametric statistical approaches in subsequent analyses.

Table (1): Shapiro–Wilk Normality Test Results for PCOS and Non-PCOS Groups

Shapiro–Wilk test *p*-value			
Group	**AMH**	**BMI**	**AGE**
PCOS	0.011	0.001	0.189 NSS
Non-PCOS	0.001	0.564 NSS	0.002

NSS: not statistically significant, *p*> 0.05

The participants were classified among age groups as shown in figure (1). The majority of cases are concentrated in the 25–34-year range. The age of the PCOS group was younger (27.4 ± 4.7 SD) years than that of the non-PCOS control group (29.15 ± 6.22 SD) years. In the PCOS group. The age distribution in figure (1) reveals that PCOS cases were predominantly clustered in younger reproductive age groups (20–29 years), on the other hand, non-PCOS control group are consistently distributed into older age groups (30–39 years). The results in Table (2) showed that the age median of PCOS women was 27 years, which was significantly lower than woman without PCOS (30 years, $p<0.0٥$). These results are in line with epidemiological findings that PCOS is diagnosed more frequently in younger reproductive-age women, especially between 20–29 years [16]. The early onset of PCOS reflects its pathophysiology, where hyperandrogenism and menstrual irregularities manifest early. The analysis of figure (1) highlights that the prevalence of PCOS is sharply declining after 30 years (only 9% in 35–39 years, which agrees with longitudinal studies showing PCOS symptoms may decrease with age, but with persistence of metabolic risks [17,18]. This pattern of age distribution suggests that metabolic conditions and gynaecological issues may explain later reproductive years challenges [1].

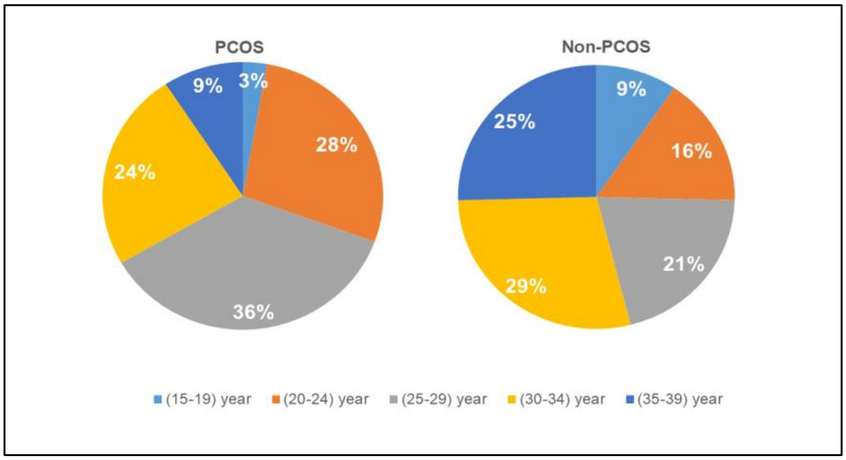

Figure (1): Age Distribution: PCOS vs. non-PCOS control groups

Table (2): Comparison of Median Age, BMI, and AMH Levels Between PCOS and non-PCOS control Groups

Parameters		**PCOS Group**	**non-PCOS control group**	***p*-value**
Age [years]	**Median**	27	30	$p<0.05^{(1)}$
BMI [kg/m^2]		29.72	27.9	NSS
AMH [ng/ml]		7.45	1.1	$p<0.05^{(2)}$

NSS= not statistically significant, *p*> 0.05; $^{(1)}$U= 2716; z= -2.199 —Mann–Whitney test; $^{(2)}$U= 265; z= -10.045 —Mann–Whitney test.

After classification of patients according to BMI, the obesity class was dominant among the two groups, as shown in figure (2). In the PCOS group, BMI was (30.4 ± 6.5 SD) Kg/m2, while it was (28.3 ± 5.08 SD) Kg/m2 in the non-PCOS control group. As shown in figure (2), women with PCOS are more founded into overweight and obese BMI categories compared with non-PCOS control group. Only 24% of PCOS women are within the BMI normal range, while about half (48%) are classified as obese (Class I–III). However, 36% of non-PCOS control group fall into the normal BMI range, and fewer (39%) are classified in obesity categories. This distribution emphasizes an association between excess adiposity and PCOS. Recent studies reported that the prevalence of obesity is significantly higher among women with

PCOS, as BMI contributes to metabolic dysfunction and hormonal imbalances [16,19,20]. Although the BMI median was somewhat higher in the PCOS group (29.72 kg/m²) vs (27.9 kg/m²) in the non-PCOS control group, as shown in Table (2), the difference was not statistically significant. These findings show that while obesity is prevalent in PCOS, lean phenotypes are still clinically relevant. Recent reviews highlight that metabolic dysfunction can arise even in normal-weight PCOS women, proposing that BMI alone cannot entirely explain disease pathogenesis [3]. However, obesity worsens PCOS symptoms and metabolic risks, supporting the lifestyle importance of interventions [1]. As shown in Table (2), the most obvious difference lies in AMH levels. In PCOS women, the median of AMH level was significantly higher (7.45 ng/ml), compared to 1.1 ng/ml non-PCOS control group ($p<0.05$). A recent meta-analysis shows that AMH levels in PCOS are consistently higher across populations, suggesting its diagnostic utility, particularly in younger women [21].

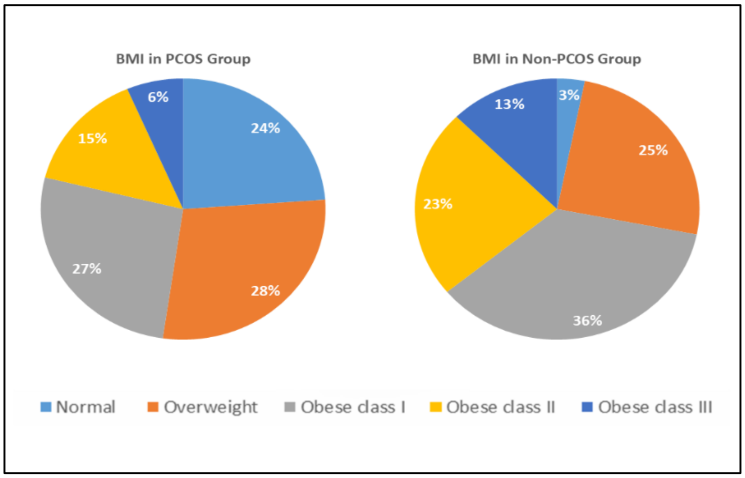

Figure (2): BMI Category Distribution in PCOS vs. non-PCOS control group

After classification of patients to five distinct age groups (15–19, 20–24, 25–29, 30–34, and 35–39 years), Table (3) show the results of BMI median comparison across age groups of women with and without PCOS, showed slightly higher of PCOS group BMI in most age categories (15–29 years), none of the differences reached statistical significance. In younger age groups (15–24 years), PCOS women exhibited higher median BMI (28.90–30.26 kg/m²) compared to non-PCOS control group (24.76–24.87 kg/m²). This result aligns with recent epidemiological studies showing that obesity often manifests earlier in PCOS [22]. Even though PCOS women tend toward higher BMI values in most age categories, Table (3) highlights that BMI differences between PCOS and non-PCOS control group across age groups are modest and not statistically significant.

Table (3): Median BMI Across Age Groups in PCOS and non-PCOS control group

Age Group (year)	BMI (Kg/m^2) (Median)		*p*-value
	PCOS	Non-PCOS	
15-19	28.90	24.87	NSS
20-24	30.26	24.76	NSS

25-29	29.96	27.91	NSS
30-34	28.86	30.38	NSS
35-39	29.93	28.04	NSS

NSS= not statistically significant, *p*> 0.05

Correlation between BMI and AMH

The scatter plots in figure (3) illustrate the relationship between AMH levels and BMI in women with and without PCOS. In both groups, the correlations are weak and statistically insignificant (PCOS: Spearman $\rho = 0.035$, $p = 0.717$; non-PCOS control group: Spearman $\rho = -0.070$, $p = 0.583$). This suggests that BMI does not exert a direct influence on AMH levels, despite the well-established role of obesity in exacerbating PCOS symptoms. This result is consistent with other studies that reported a lack of strong correlation between AMH level and BMI in PCOS women, indicating that ovarian dysfunction is not directly mediated by adiposity [12,22,23]. The weak positive slope in the PCOS group and the weak negative slope in the non-PCOS control group highlight subtle differences. In PCOS, AMH may remain elevated regardless of BMI, while in non-PCOS control group, higher BMI may be associated with slightly lower AMH, reflecting age-related ovarian reserve decline compounded by metabolic stress. Yet, these associations are too weak to be clinically meaningful, as confirmed by the non-significant p-values. However, Singh M reported that there is a strong negative relationship between blood AMH levels and BMI in females with PCOS [24]. Another study found that AMH level might be differentially regulated by BMI in obese and lean PCOS [25]. Table (4) demonstrates that AMH levels are consistently higher in women with PCOS compared to non-PCOS control group across all BMI categories, with statistically significant differences ($p<0.0$٥). This reinforces the role of AMH as a robust biomarker of PCOS, independent of body weight. Interestingly, in obese class I, AMH levels in PCOS women were slightly lower (5.72 ng/ml) compared to other BMI categories, though still significantly higher than in non-PCOS control group (0.80 ng/ml). The obesity may modestly decrease AMH levels, perhaps due to granulosa cell function alteration [26]. In obese class II, PCOS women had the highest median AMH (8.63 ng/ml), compared to 2.03 ng/ml in non-PCOS control group, suggesting heterogeneity in how obesity interacts with AMH [23]. For obese class III, PCOS women maintained elevated AMH (7.87 ng/ml), while no non-PCOS control group data were available. This highlights that even in severe obesity, AMH remains a distinguishing feature of PCOS. The result agreed with many studies that reported AMH is a reliable diagnostic across diverse populations [1].

Table (4): Median AMH Levels Across BMI Categories in PCOS and non-PCOS control groups

BMI Group (Kg/m^2)	AMH (ng/ml) (Median)				*p*-value
	No. of Cases	PCOS	No. of Cases	non-PCOS control group	
Normal	26	7.33	16	1.29	$p<0.0$٥$^{(1)}$
Overweight	30	7.46	22	1.05	$p<0.0$٥ $^{(2)}$
Obese class I	29	5.72	15	0.80	$p<0.0$٥ $^{(3)}$
Obese class II	16	8.63	10	2.03	$p<0.0$٥ $^{(4)}$
Obese class III	7	7.87		-	-

(1)U= 12.000; z= -5.299 —Mann–Whitney test; (2)U= 25.000; z= --5.649—Mann–Whitney test; (3)U= 21.000; z= -4.865—Mann–Whitney test; (4)U= 3.000; z= -3.735—Mann–Whitney test.

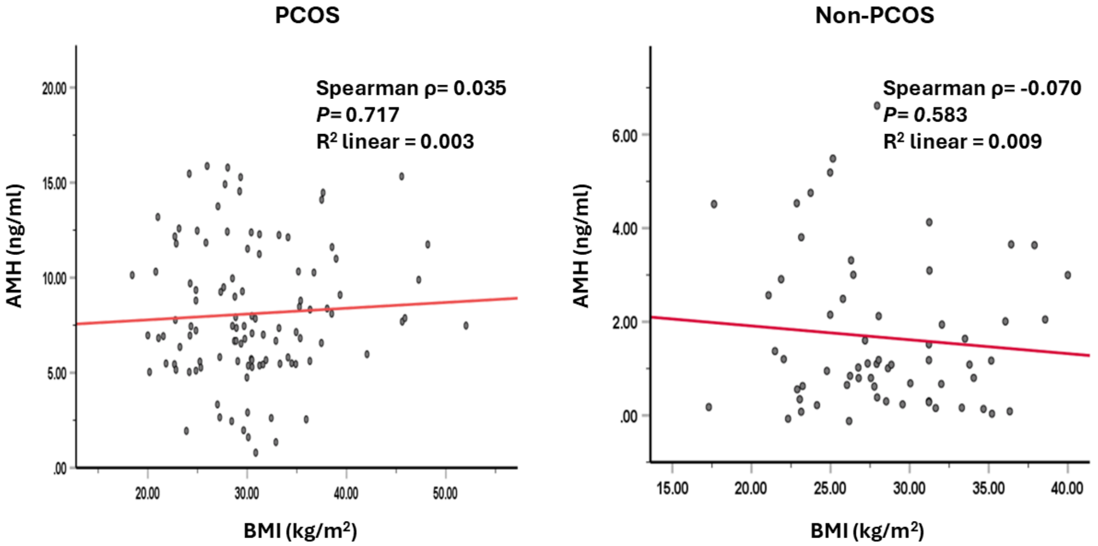

Figure (3): Correlation Between AMH and BMI in PCOS and non-PCOS control groups

Correlation between Age groups and AMH

As shown in figure (4), the correlation between AMH and age in both groups is weak and statistically non-significant (PCOS: Spearman ρ = -0.109, p = 0.260; non-PCOS control group: Spearman ρ = -0.115, p = 0.370). The negative slopes suggest a general decline in AMH with age, but the weak associations indicate that AMH variation cannot be fully explained by age alone. This result agreed with a meta-analysis study that reported that AMH levels decline gradually with age in all women, but remain significantly elevated in PCOS compared to controls, reflecting increased follicle count and disrupted folliculogenesis [21]. The age weak correlation in the PCOS group suggests that AMH levels are relatively stable across reproductive years, consistent with findings that PCOS women often maintain elevated AMH into their thirties [16]. In contrast, the slightly stronger negative slope in non-PCOS control group women reflects the expected age-related decline in ovarian reserve, which is well documented in population studies [26]. Table (5) demonstrates that AMH levels are consistently higher in women with PCOS compared to non-PCOS controls across all age groups, with statistically significant differences (p<0.0٥), which also confirms that AMH is a robust biomarker of PCOS, independent of age. In the 15–19 age group, PCOS women showed markedly elevated AMH (9.96 ng/ml) compared to non-PCOS control group (1.16 ng/ml). This finding is in line with recent evidence that AMH is particularly elevated in adolescents and young adults with PCOS [27,28]. In the 20–24 group, PCOS women maintained high AMH (8.42 ng/ml vs. 1.80 ng/ml), supporting studies that show AMH peaks in early reproductive years and remains a reliable diagnostic marker [16].

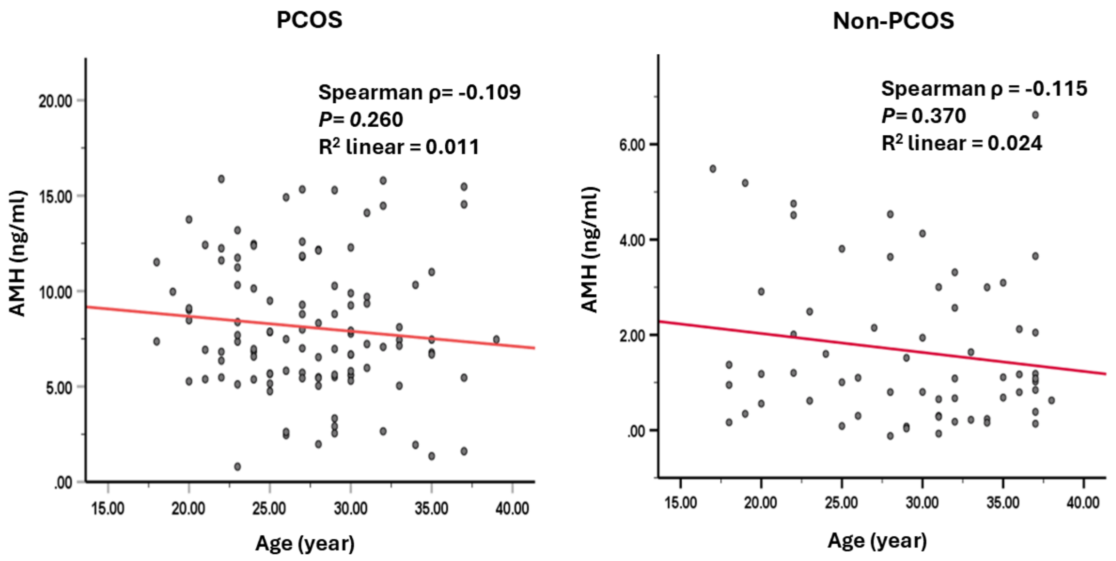

Figure (4): Association Between AMH and Age in PCOS and non-PCOS control groups

Table (5): Median AMH Levels Across Age Groups in PCOS and non-PCOS control samples

Age Group (year)	AMH (ng/ml) (Median)		*p* value
	PCOS	non-PCOS control group	
15-19	9.96	1.16	p<0.0٥ (1)
20-24	8.42	1.80	p<0.0٥ (2)
25-29	6.96	1	p<0.0٥ (3)
30-34	7.33	0.73	p<0.0٥ (4)
35-39	7.11	1.1	p<0.0٥ (5)

NSS= not statistically significant; (1)U= 0.000; z= -2.324 —Mann–Whitney test; (2)U= 8.000; z= -4.435—Mann–Whitney test; (3)U= 19.000; z= -4.956—Mann–Whitney test; (4)U= 9.500; z= -5.359—Mann–Whitney test; (5)U= 11.000; z= -3.637—Mann–Whitney test.

Assessment of AMH, BMI and age as diagnostic factors for PCOS

The ROC curve compares the discriminative ability of AMH, BMI and age in distinguishing between PCOS and non-PCOS control groups. Among the three variables, AMH demonstrates the strongest diagnostic performance, with an area under the curve (AUC = 0.961), indicating excellent predictive accuracy as shown in figure (5). Recent studies confirm these observations, which demonstrated that AMH is consistently elevated in PCOS across populations and serves as a strong diagnostic parameter, especially when combined with clinical criteria [23,27]. The limited BMI diagnostic value in this analysis reflects the PCOS heterogeneity, as this disorder can occur independently of the obesity [26]. Similarly, age is not a consistent predictor, since PCOS arise across reproductive years [16]. Teede et al. (2023) highlight that while age and BMI are important factors for management, they lack diagnostic specificity in comparison with AMH. Clinically, these results emphasise that AMH must be prioritised as an adjunct in PCOS diagnosis, particularly in younger women, where the criteria of ultrasound may be less reliable. The addition of AMH into PCOS diagnostic frameworks reduces misclassification and improves accuracy, while age and BMI remain appropriate for prognosis and treatment planning instead of diagnosis.

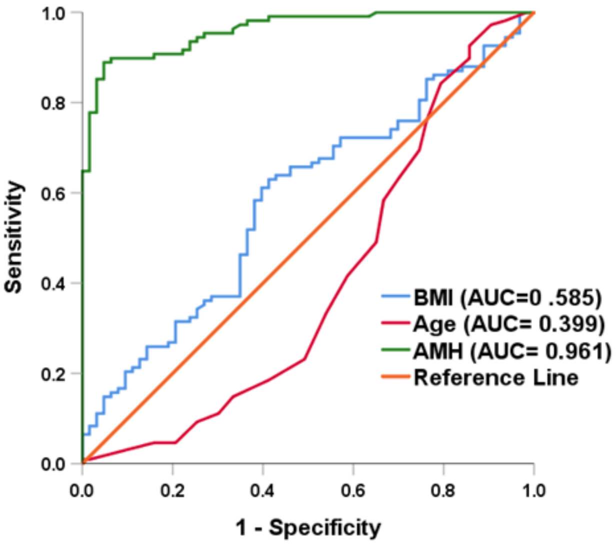

Figure (5): ROC Curve Analysis of BMI, Age, and AMH for PCOS Diagnosis

Conclusions

PCOS is a disorder of younger reproductive-age women, with diagnosis clustering in the 20–29 age group. Women with PCOS in general tend to have higher values of BMI, however the differences with non-PCOS control groups are minor and often not statistically significant. Across all analyses, AMH emerges as the most appropriate biomarker, which remains elevated in PCOS regardless of BMI or age.

The correlation analysis shows that AMH is statistically independent of these factors, in addition, the ROC analysis reveals its superior accuracy in PCOS diagnosis compared with BMI and age. These findings highlight the need for age-specific strategies of diagnosis, comprehensive assessment of metabolic risk and addition of AMH into diagnostic frameworks. Age and BMI can be considered as contextual factors for the management and prognosis of PCOS.

Acknowledgments

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Author's Declaration

We hereby confirm that all the Tables and figures in the manuscript are original and have been created by us. The study protocol was approved by the Kamal Al-Samarrai Hospital for Infertility Treatment and IVF, Ministry of Health, Baghdad/ Iraq (No. 9495 on 6/0٨/2025). Before taking the blood samples, all of the participants were informed about the aims of this study, and their participation agreement was obtained. No conflict of interest was declared by the authors

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