

■ Original Article

## Anti-Mullerian Hormone According to Polycystic Ovary Syndrome Phenotypes

### *Polikistik Over Sendromu Fenotiplerine Göre Anti-Müllerian Hormon Değerleri*

Serkan Polat\*<sup>1</sup> , Osman Nuri Erginay<sup>1</sup> 

<sup>1</sup>Department of Obstetrics and Gynecology, Ankara Etlik Zübeyde Hanım Women's Health Training and Research Hospital, Ankara, Türkiye

#### Abstract

**Objective:** The aim of this study was to investigate the level of antimullerian hormone in patients with polycystic ovary syndrome (PCOS) according to phenotypic characteristics.

**Materials and Methods:** This study was designed as a cross-sectional cohort study and included patients attending the PCOS clinic of Etlik Zübeyde Hanım Gynecology Training and Research Hospital. Anti-mullerian hormone (AMH) levels were recorded according to phenotype assessment.

**Results:** A total of 118 patients with PCOS participated in the study. Accordingly, 47 patients (39.8%) belonged to phenotype A, 15 patients (12.7%) to phenotype B, 37 patients (31.3%) to phenotype C and 19 patients (16.1%) to phenotype D. The mean age of the patients was 22.97±4.98 years. The mean body mass index was 26.1± 4.26 kg/m<sup>2</sup>. The most common reason for presentation to the PCOS outpatient clinic was irregular menstruation. The most common reason for presentation to the PCOS outpatient clinic was irregular menstruation in phenotype A (80.9%) and phenotype D (84.2%). There was a difference between the groups in the distribution of the presence of polycystic ovarian morphology (PCOM) and the frequency of ovarian dysfunction by phenotype (p<0.001, p<0.001). A statistically significant difference was found between median AMH levels (ng/ml) according to phenotype (p<0.001). The median value was 6.3 ng/ml in phenotype A, 2.4 ng/ml in phenotype B, 6.1 ng/ml in group C and 6.6 ng/ml in phenotype D.

**Conclusion:** In our study, phenotype A was the most frequently observed group and AMH levels were significantly higher in the phenotype D group than in the other groups.

**Keywords:** Phenotype; polycystic ovary syndrome; anti mullerian hormone; Ferriman Gallwey score

## Öz

**Amaç:** Bu çalışmanın amacı polikistik over sendromlu (PKOS) hastalarda fenotipik özelliklere göre antimüllerian hormon düzeyini araştırmaktır.

**Gereç ve Yöntemler:** Bu çalışma kesitsel bir kohort çalışması olarak tasarlandı ve Etlik Zübeyde Hanım Kadın Hastalıkları Eğitim ve Araştırma Hastanesi PKOS kliniğine başvuran hastalar dahil edilmiştir. Anti-müllerian hormon (AMH) düzeyleri fenotip değerlendirilmesine göre kaydedilmiştir.

**Bulgular:** Çalışmaya toplam 118 PKOS hastası katılmıştır. Buna göre 47 hasta (%39.8) fenotip A, 15 hasta (%12.7) fenotip B, 37 hasta (%31.3) fenotip C ve 19 hasta (%16.1) fenotip D'dir. Hastaların yaş ortalaması 22.97±4.98 yıldır. Ortalama vücut kitle indeksi 26.1± 4.26 kg/m<sup>2</sup>'dir. PKOS polikliniğine en sık başvuru nedeni adet düzensizliğidir. Fenotip A (%80.9) ve fenotip D'de (%84.2) PKOS polikliniğine en sık başvuru nedeni adet düzensizliğidir. Polikistik over morfolojisi (PKOM) varlığı ve over disfonksiyonu sıklığının fenotipe göre dağılımında gruplar arasında fark vardır (p<0.001, p<0.001). Fenotipe göre ortanca AMH düzeyleri (ng/ml) arasında istatistiksel olarak anlamlı bir fark bulunmuştur (p<0.001). Ortanca değer fenotip A'da 6,3 ng/ml, fenotip B'de 2,4 ng/ml, grup C'de 6,1 ng/ml ve fenotip D'de 6,6 ng/ml'dir.

**Sonuç:** Çalışmamızda fenotip A en sık gözlenen gruptur ve AMH düzeyleri fenotip D grubunda diğer gruplara göre anlamlı olarak daha yüksektir.

**Anahtar Kelimeler:** Fenotip; polikistik over sendromu; anti müllerian hormon; Ferriman Gallwey skoru

## 1. Introduction

Polycystic ovary syndrome (PCOS) is a health problem characterized by complex hormonal disorders affecting public health (1). Stein and Levinthal described PCOS in 1935 with the publication of seven cases and this was the beginning of this syndrome that affects public health today (2). PCOS has a lifelong negative impact on women's health and leads to anxiety, depression, insulin resistance, abdominal obesity, hypertension and dyslipidemia (3). Another negative effect seen in women with PCOS is the increased rate of infertility. Its prevalence is between 8 and 13% depending on the population studied (1,2). In order to standardize the diagnosis of PCOS, 3 classification systems based on phenotypic characteristics have been defined and diagnostic criteria vary according to these classifications which are still valid. The so-called Rotterdam criteria are as follows; Oligo and/or anovulation, Clinical and/or biochemical symptoms of hyperandrogenism, Polycystic ovary morphology on ultrasound, Other conditions that cause androgen increase or are associated with androgen increase should be ruled out before the diagnosis of PCOS is made.

The main phenotypic characteristics of PCOS cases are based on clinical symptoms and/or signs, laboratory findings or imaging findings. Considering the diagnostic classifications and criteria,

there are generally 3 main phenotypic features that make up the clinical picture in PCOS cases of reproductive age;

1. Ovulatory and menstrual dysfunction (OD)
2. Clinical features of hyperandrogenemia and/or hyperandrogenism (HA)
3. Morphology of polycystic ovaries (PCOM).

In PCOM, clinical manifestations such as hirsutism, oligo-anovulation, hyperandrogenemia on biochemical tests and ultrasonographic appearance of the ovaries can occur in very different combinations. Hyperandrogenemia is the common phenotypic feature found in all 3 diagnostic classifications. Some clinical and laboratory phenotypic features that are not included in the definition criteria for PCOS but complement the clinical picture and influence disease severity and morbidity have also been described. These include obesity, metabolic abnormalities (insulin resistance/hyperinsulinemia, glucose intolerance/type 2 DM, metabolic syndrome, dyslipidemia), sleep apnea, psychosocial problems and abnormal gonadotropin dynamics. The most important factors influencing the phenotype in PCOS are ethnic, racial and other cultural factors. These phenotypic traits have similar inheritance patterns and cause similar diseases. The severity of phenotypic traits is also highly variable. Another importance of phenotypic traits is that treatment

needs, types and options differ according to these traits. The OD+HA+PKOM phenotype is considered the complete (classic) phenotype according to the Rotterdam classification and the highest rate is seen in this phenotype. Clinical manifestations (phenotype A: HA + OD + PCOM; phenotype B: HA + OD; phenotype C: HA + PCOM and phenotype D: OD + PCOM). According to the Rotterdam criteria, endocrine and metabolic abnormalities are lowest in the OD+PCOM group among these 4 different phenotypes. The prevalence and distribution characteristics of metabolic abnormalities (insulin resistance, metabolic disease pattern and glucose intolerance) did not differ significantly between the 4 groups. Therefore, metabolic abnormalities and distribution characteristics are not used to distinguish between different clinical PCOS phenotypes.

Serum anti-Müllerian hormone (AMH) can be used to identify PCOS in adults (4). Serum AMH is used in accordance with the PCOS diagnostic algorithm. AMH level is not required for the diagnosis of PCOS in patients with irregular menstrual cycle and hyperandrogenism findings. AMH level is not required in adolescents. In the general population, serum AMH levels usually peak at 20-25 years of age. In the general population, serum AMH levels are lower in individuals with a higher body mass index (BMI). Studies in patients with PCOS have shown that phenotyping is necessary to better monitor clinical outcomes (5). In our study, data were grouped according to phenotype and the correlation of AMH results with phenotypes was tried to be determined.

## 2. Material and Methods

This study was designed as a cross-sectional cohort study involving patients treated at the Etlik Zübeyde Hanım Gynecology Training and Research Hospital PCOS Clinic. Approval for non-interventional studies was obtained from the Ethics Committee of Etlik Zübeyde Hanım Gynecology Training and Research Hospital prior to the start of the study (approval date: 20/03/2024, No.: 03/13). The study involved 118 subjects who agreed to participate in the study and gave their verbal and written informed consent.

The Rotterdam criteria were used for the diagnosis of PCOS (6). Hyperandrogenism was determined clinically by Ferriman-Gallwey score (>8) and biochemically by serum total testosterone (>1.5 nmol/L) and free androgen index (FAI) (>4). Ovulatory dysfunction was defined as patients with menstrual cycles lasting longer than 38 days (oligomenorrhea).

### Inclusion criteria

Between 14 and 40 years after menarche,  
No underlying metabolic disease (type 2 diabetes, hypertension, diagnosed anemia),  
We have one patient with AMH levels in our hospital,  
Female patients attending the PCOS clinic will be included.

### Exclusion criteria

Age > 40 years;  
Menopause, pregnancy or breastfeeding within the last 6 months;  
Hyperandrogenism and/or biochemical hyperandrogenemia due to secondary etiologies, including congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome, hyperprolactinemia, thyroid dysfunction and adrenal disease),  
Pre-existing systemic diseases.

Demographic characteristics, laboratory results and hospital records were obtained. Age, PCOS phenotype, age at first menstruation, menstrual cycle pattern and birth weight were routinely recorded. Body weight (kg) and height (m), body mass index (kg/m<sup>2</sup>), systolic and diastolic blood pressure (mmHg) and Ferriman & Gallwey (FG) score for hirsutism (mean hirsutism of 7 or more points) were recorded. The cycle length was recorded. The phenotypic characteristics of the PCOS patients were recorded according to the "Hyperandrogenemia and PCOS Association". They were categorized according to the PCOS criteria: Phenotype A: oligomenorrhea + hyperandrogenism + polycystic ovaries (PCO), Phenotype B: oligomenorrhea + hyperandrogenism, Phenotype C: hyperandrogenism + PCO, Phenotype D: Oligomenorrhea + PCO.

In routine practice at the PCOS clinic, around 7 ml of blood is taken in a vacuum gel tube for hormonal and biochemical analysis by medical staff. The blood samples are centrifuged by the examiners at 1000 x g for 20 minutes. In the next step, the supernatant part is separated and transferred to 3 mL Ependorfs. These samples are used to determine the levels of anti-Müllerian hormone (AMH), oestradiol, luteinizing hormone (LH) and follicle stimulating hormone (FSH), which are routinely tested at the PCOS clinic. In addition, patients attending the PCOS outpatient clinic are routinely examined by transvaginal ultrasound (TV-USG) for the number of antral follicles and ovarian volume. Patients are examined transvaginally by the same experienced gynecologist using a Samsung HS70A and the number of antral follicles in both ovaries and the volume



of the right and left ovary are recorded. The Orsini formula (length x depth x width x 0.5235)-[transverse, anteroposterior, longitudinal axes] is used to measure the volume of the ovaries.

### Statistical analysis

The data were analyzed using the IBM SPSS V23 program. The Shapiro-Wilk test was used to analyze the data. The Fisher-Freeman-Halton test was used to analyze categorical data and multiple comparisons were performed using the Bonferroni-corrected Z-test. One-way analysis of variance was used to compare the variables that fit the normal distribution with the groups. The Kruskal Wallis test was used to compare the variables that did not conform to the normal distribution with the groups. The results of the analyzes were presented as frequency (percentage) for categorical variables, mean ± standard deviation and median (minimum - maximum) for quantitative variables. The significance level was set at  $p < 0.050$ .

The power analysis was performed with the program G\*POWER 3.1 to determine the sample size. The power analysis for sample size calculation was based on the previous study by Barrea et al. (3). Participants who met the inclusion criteria were included in the study. After analyzing the 95% confidence (1- $\alpha$ ), 95% test power (1- $\beta$ ) and  $d = 1.5915486$  effect size one-sided independent samples t-test, the number of samples to be taken was set at 80. Since our sample size is above this number, we assume that the test power is higher.

### 3. Results

The number of patients admitted to our PCOS outpatient clinic between November 2023 and April 2024 was 118. The average age of the patients was  $22.97 \pm 4.98$  years. The mean body mass index was  $26.1 \pm 4.26$  kg/m<sup>2</sup>. These data are shown in Table 1, which describes the demographic characteristics. The most common reason for presentation to the PCOS outpatient clinic was irregular menstruation. The most frequently observed phenotypic group was group A. The analysis of clinical characteristics by phenotype is shown in Table 2. According to this, there was no significant difference between the phenotypic groups in terms of age, BMI and FG score median ( $p = 0.773$ ,  $p = 0.501$ ,  $p = 0.985$ ). There was a difference between groups in reasons for use when assessing reasons for use ( $p < 0.001$ ). The most common reason for use was irregular menstruation in phenotype A (80.9%) and phenotype D (84.2%). There were also differences between groups in the distribution of the presence of PCOM and the frequency of ovulatory disorders according to phenotype ( $p < 0.001$ ,  $p < 0.001$ ).

The laboratory parameters according to phenotype are shown in Table 3. No statistically significant difference was found between FSH (mIU/ml), LH (mIU/ml) and FSH/LH median values according to phenotype ( $p = 0.566$ ,  $p = 0.171$ ,  $p = 0.217$ ). A statistically significant difference was found between the median values of 17-hydroxyprogesterone (ng/ml) ( $p = 0.032$ ). The median value was 0.94 ng/ml in phenotype A, 0.45 ng/ml in phenotype B, 1.5 ng/ml in phenotype C and 0.5 ng/ml in group D. There was no statistically significant difference between the median values of dehydroepiandrosterone sulfate (DHEAS) (ng/L) by phenotype ( $p = 0.899$ ). There was a statistically significant difference between the median levels of AMH (ng/ml) according to phenotype ( $p < 0.001$ ). The median value was 6.3 ng/ml for phenotype A, 2.4 ng/ml for phenotype B, 6.1 ng/ml for phenotype C and 6.6 ng/ml for phenotype D. There was no statistically significant difference between the median values of insulin and HOMA-IR according to phenotype ( $p = 0.170$ ,  $p = 0.535$ ).

	Study Group n:118
<b>Age (years)</b>	22.97±4.98
<b>Body Mass Index (kg/m<sup>2</sup>)</b>	26.1±4.26
<b>Reason for applying n (%)</b>	
Menstrual irregularity	66 (55%)
Increased hair growth	40 (33%)
Acne	4 (3%)
Child counselling	1 (0.8%)
Failure to lose weight	7 (5.9%)
<b>Polycystic ovarian morphology n (%)</b>	
Yes	103 (87.2%)
No	15 (12.7%)
<b>Oligo/anovulation n (%)</b>	
Yes	80 (67.7%)
No	38 (32.2%)
<b>Phenotypes n (%)</b>	
A	47 (39.8%)
B	15 (12.7%)
C	37 (31.3%)
D	19 (16.1%)
<b>The Ferriman-Gallwey score</b>	15.52±6.47

**Table 2.** Clinical characteristics of patients according to PCOS phenotypes

	Phenotypes				T-test	p
	A	B	C	D		
Age (years)	23,5 ± 4,9	22,9 ± 6,2	22,6 ± 5	22,4 ± 4,3	1,119	0,773*
Body Mass Index (kg/m <sup>2</sup> )	26,8 ± 6,6	28 ± 4,7	25,6 ± 5,3	26,6 ± 5,4	2,363	0,501*
Ferriman Gallwey score	15 (5 - 28)	15 (5 - 29)	15 (5 - 34)	14 (5 - 30)	0,050	0,985**
Reason for applying n (%)						
Menstrual irregularity	38 (80,9) <sup>a</sup>	7 (46,7) <sup>ab</sup>	5 (13,5) <sup>b</sup>	16 (84,2) <sup>a</sup>	62,108	<0,001***
Increased hair growth	6 (12,8) <sup>a</sup>	7 (46,7) <sup>b</sup>	27 (73) <sup>b</sup>	0 (0) <sup>a</sup>		
Acne	2 (4,3)	0 (0)	2 (5,4)	0 (0)		
Child counselling	0 (0)	0 (0)	1 (2,7)	0 (0)		
Failure to lose weight	1 (2,1)	1 (6,7)	2 (5,4)	3 (15,8)		
Polycystic ovarian morphology n (%)						
No	0 (0) <sup>b</sup>	15 (100) <sup>a</sup>	0 (0) <sup>b</sup>	0 (0) <sup>b</sup>	78,204	<0,001***
Yes	47 (100)	0 (0)	37 (100)	19 (100)		
Oligo/anovulation n (%)						
No	0 (0) <sup>a</sup>	0 (0) <sup>a</sup>	37 (100) <sup>b</sup>	1 (5,3) <sup>a</sup>	128,029	<0,001***
Yes	47 (100)	15 (100)	0 (0)	18 (94,7)		

\*Kruskal Wallish H test, \*\*One-Way Analysis of Variance, \*\*\*Fisher Freeman Halton Test; a-b: No difference between groups with the same letter; Mean ± standard deviation, Median (minimum-maximum).

#### 4. Discussion

In our study, adolescent and adult PCOS patients were examined with regard to clinical and biochemical parameters. In summary, it was found that the most frequently observed group was phenotype A and AMH levels were significantly higher in the phenotype D group than in the other groups.

With the 2023 ESHRE guideline, elevated AMH levels in the adult group were included in the diagnostic criteria (4). Given the difficulty of ultrasound diagnosis of PCOS, even years after menarche, serum anti-Müllerian hormone (AMH) has been proposed as an alternative marker for PCOM. AMH is a polypeptide. It belongs to the transforming growth factor beta (TGF-β) family and is secreted exclusively by granulosa cells in preantral and small antral follicles. The AMH serum level is significantly higher in women with PCOS than in women with normal ovulation. A strong correlation between the circulating AMH level and the number of antral follicles has been demonstrated (7,8). When we compared AMH levels by phenotype in our study, we found that AMH levels were significantly higher in the phenotype D group compared to the other groups.

In 2009, Piouka et al. found that AMH levels reflect the severity of PCOS (9). Sahmay et al. showed that AMH levels differed between phenotypes and were significantly higher in phenotype A (10). Subsequently, many studies have shown that AMH levels were higher in the phenotype A group than in other groups (11-14). However, in our study, we found that AMH levels were high in the phenotype D group and particularly low in the phenotype B group. We believe that we determined the result in this way because we studied not only the adult group but also the adolescent group.

Bozdag et al. studied 392 women to determine diagnostic AMH levels and found that the AMH levels of women with phenotype A PCOS were significantly higher and the most appropriate AMH threshold for the diagnosis of PCOS was 4.86 ng/mL (15). Dewailly et al. proposed a simplified diagnosis of PCOS based on an AMH threshold of 5 ng/mL (16). Another study determined an AMH value of 6.095 ng/ml for phenotype-A with a sensitivity of 69.2% and a specificity of 86.7% (17). The average of our values is also close to this threshold. Since it may not be easy to perform an ultrasound and examine the ovaries in virginal and obese patients or in regions where an ultrasound is not easily accessible, the AMH level can be used to determine the





**Table 3.** Results of laboratory outputs obtained according to PCOS phenotypes

	Phenotypes				T test	p*
	A	B	C	D		
FSH (IU/L)	4,66 ± 1,35	4,69 ± 1,01	4,55 ± 2,28	4,89 ± 1,38	2,031	0,566
	4,58 (2 - 8)	5 (3 - 6)	4,56 (1 - 12)	5,13 (2 - 7)		
LH (IU/L)	9,25 ± 4,98	6,75 ± 4,56	9,85 ± 6,68	8,94 ± 6,33	5,012	0,171
	8,06 (2 - 22)	5,46 (3 - 18)	8,76 (1 - 31)	6,81 (2 - 25)		
FSH/LH	0,74 ± 0,52	0,91 ± 0,47	0,79 ± 0,66	1 ± 0,76	4,443	0,217
	0,59 (0 - 3)	0,8 (0 - 2)	0,6 (0 - 3)	0,75 (0 - 3)		
Estradiol (pg/ml)	47,82 ± 25,78	34,15 ± 19,49	50,45 ± 28,25	53,75 ± 30,45	5,394	0,145
	43 (2 - 162)	35,6 (5 - 85)	43 (5 - 115)	46,4 (5 - 112)		
TSH (IU/L)	2,1 ± 1,26	1,98 ± 0,73	2,69 ± 1,98	2,11 ± 1,48	1,945	0,584
	1,72 (0 - 6)	1,97 (1 - 4)	2,15 (0 - 9)	2 (0 - 6)		
17-hydroxyprogesterone (ng/ml)	1,69 ± 2,44	0,6 ± 0,42	1,91 ± 1,75	1,1 ± 1,26	8,835	<b>0,032</b>
	0,94 (0 - 11) <sup>ab</sup>	0,45 (0 - 1) <sup>a</sup>	1,5 (0 - 8) <sup>b</sup>	0,5 (0 - 5) <sup>ab</sup>		
Prolactin (ng/ml)	17,16 ± 7,56	22,03 ± 21,94	17,52 ± 9,08	20,17 ± 11,14	0,63	0,889
	16,7 (6 - 31)	17 (7 - 98)	18 (4 - 39)	18,6 (7 - 45)		
DHEA-SO4 (ng/ml)	250,22 ± 97,46	250,67 ± 138,25	245,76 ± 113,13	251,58 ± 98,14	0,588	0,899
	245 (83 - 461)	245 (102 - 625)	205 (97 - 587)	242 (100 - 451)		
AMH (ng/ml)	7,66 ± 4,25	2,79 ± 1,47	7,52 ± 5,19	8,03 ± 3,67	26,496	<b>&lt;0,001</b>
	6,3 (1 - 21) <sup>a</sup>	2,4 (1 - 8) <sup>b</sup>	6,1 (1 - 24) <sup>a</sup>	6,6 (3 - 17) <sup>a</sup>		
Insulin (ng/ml)	4,02 ± 4,13	6,19 ± 3,98	5,37 ± 6,94	5,23 ± 4,38	5,029	0,170
	3,03 (0,01 - 18)	7,7 (0,28 - 13,7)	3,78 (0,09 - 40,3)	4,39 (0,02 - 17,4)		
HOMA-IR	4,74 ± 4,71	4,04 ± 2,71	3,69 ± 2,63	4,64 ± 2,25	2,182	0,535
	3,96 (0,01 - 23,5)	2,86 (0,86 - 9,09)	3,14 (0,01 - 9,09)	4,14 (1,52 - 8,5)		

\*Kruskal Wallish H test, a-b: No difference between groups with the same letter; FSH: follicle-stimulating hormone; LH: Luteinizing hormone; TSH: thyroid stimulating hormone; DHEA-SO4: dehydroepiandrosterone sulfate; AMH: Anti-müllerian hormone; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance

PCOS phenotype. As women with PCOS phenotype A should be counseled about the lifelong effects on metabolism and lifestyle changes with healthy eating habits and regular exercise should be strongly recommended, such a quantitative test may be more reliable and accessible than a transvaginal ultrasound. Many morbidities occur in this patient group, from obstetric complications to complications such as cancer and heart disease, that affect menopausal life (18).

Obesity and insulin resistance (IR) are interrelated parameters in the pathophysiology of PCOS (19,20). PCOS phenotype A is usually associated with obesity and IR and is considered the most severe form of PCOS (21). Phenotype B has similar but

milder metabolic consequences. Phenotype C is milder as the predominant problem is subfertility. Phenotype D is the mildest form with less obesity, IR or metabolic side effects (22). PCOS phenotypes A and B are more prone to obesity and insulin resistance (20). Since our study only selected patients who presented to the PCOS outpatient clinic and had a diagnosis, their metabolic status was found to be similar and there was no difference between the phenotypes.

The retrospective design and limited number of patients studied is one of the limitations of the study, and the lack of grouping of comparative data by age group is another limitation. In this study, we have no data on the number of antral follicles in the

patients. Secondly, the values for the area under the curve and the ROC analysis for the AMH values according to phenotype could not be provided.

PCOS is one of the most common endocrine disorders worldwide and can affect women of any age. Although many genetic, environmental and hormonal factors are thought to be responsible, the etiopathogenesis is still not fully understood. According to the phenotypic characteristics of the patients presenting in our study, the most common group was phenotype A. The AMH level was significantly higher in the phenotype D group than in the other groups. In group B, the AMH cut-off value, which was considered high, was below 3.32 ng/ml. Our results may shed light on the etiopathogenesis of PCOS. The development of PCOS in adolescence and adulthood could be due to different mechanisms and hormonal changes. Large prospective series of studies are needed to make a definitive statement on this topic.

#### Author contribution

Study conception and design: SP; data collection: ONE; analysis and interpretation of results: SP and ONE; draft manuscript preparation: SP and ONE. All authors reviewed the results and approved the final version of the manuscript.

#### Ethical approval

The study was approved by the Ethics Committee for Non-interventional Studies of Etlik Zubeyde Hanım Women Health Education Research Hospital (Protocol no. 03/13 - 20.03.2024).

#### Funding

The authors declare that the study received no funding.

#### Conflict of interest

The authors declare that there is no conflict of interest.

#### Yazar katkısı

Araştırma fikri ve tasarımı: SP; veri toplama: ONE; sonuçların analizi ve yorumlanması: SP ve ONE; araştırma metnini hazırlama: SP ve ONE. Tüm yazarlar araştırma sonuçlarını gözden geçirdi ve araştırmanın son halini onayladı.

#### Etik kurul onayı

Bu araştırma için Etlik Zübeyde Hanım Kadın Sağlığı Eğitim ve Araştırma Hastanesi Girişimsel Olmayan Çalışmalar Etik Kurulundan onay alınmıştır (Karar no: 03/13 - 20.03.2024).

#### Finansal destek

Yazarlar araştırma için finansal bir destek almadıklarını beyan etmiştir.

#### Çıkar çatışması

Yazarlar herhangi bir çıkar çatışması olmadığını beyan etmiştir.

## References

1. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab.* 2004;89(6):2745-9. [\[Crossref\]](#)
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril.* 2004;81(1):19-25. [\[Crossref\]](#)
3. Barrea L, Arnone A, Annunziata G, et al. Adherence to the Mediterranean Diet, Dietary Patterns and Body Composition in Women with Polycystic Ovary Syndrome (PCOS). *Nutrients.* 2019;11(10):2278. [\[Crossref\]](#)
4. Teede HJ, Tay CT, Laven JJE, et al; International PCOS Network. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. *Eur J Endocrinol.* 2023;189(2):G43-64. [\[Crossref\]](#)
5. Yilmaz M, Isaoglu U, Delibas IB, Kadanali S. Anthropometric, clinical and laboratory comparison of four phenotypes of polycystic ovary syndrome based on Rotterdam criteria. *J Obstet Gynaecol Res.* 2011;37(8):1020-6. [\[Crossref\]](#)
6. Tian X, Ruan X, Du J, Cheng J, Ju R, Mueck AO. Sexual function in Chinese women with different clinical phenotypes of polycystic ovary syndrome. *Gynecol Endocrinol.* 2023;39(1):2221736. [\[Crossref\]](#)
7. Cook CL, Siow Y, Brenner AG, Fallat ME. Relationship between serum müllerian-inhibiting substance and other reproductive hormones in untreated women with polycystic ovary syndrome and normal women. *Fertil Steril.* 2002;77(1):141-6. [\[Crossref\]](#)
8. Seifer DB, Maclaughlin DT. Mullerian Inhibiting Substance is an ovarian growth factor of emerging clinical significance. *Fertil Steril.* 2007;88(3):539-46. [\[Crossref\]](#)
9. Piouka A, Farmakiotis D, Katsikis I, Macut D, Gerou S, Panidis D. Anti-Mullerian hormone levels reflect severity of PCOS but are negatively influenced by obesity: relationship with increased luteinizing hormone levels. *Am J Physiol Endocrinol Metab.* 2009;296(2):E238-43. [\[Crossref\]](#)
10. Sahmay S, Atakul N, Oncul M, Tuten A, Aydoğan B, Seyisoglu H. Serum anti-Mullerian hormone levels in the main phenotypes of polycystic ovary syndrome. *Eur J Obstet Gynecol Reprod Biol.* 2013;170(1):157-61. [\[Crossref\]](#)
11. Sova H, Unkila-Kallio L, Tiitinen A, et al. Hormone profiling, including anti-Müllerian hormone (AMH), for the diagnosis of polycystic ovary syndrome (PCOS) and characterization of PCOS phenotypes. *Gynecol Endocrinol.* 2019;35(7):595-600. [\[Crossref\]](#)
12. Jamil AS, Alalaf SK, Al-Tawil NG, Al-Shawaf T. Comparison of clinical and hormonal characteristics among four phenotypes of polycystic ovary syndrome based on the Rotterdam criteria. *Arch Gynecol Obstet.* 2016;293(2):447-56. [\[Crossref\]](#)
13. Ozay AC, Emekci Ozay O, Gulekli B. Comparison of Anti-müllerian Hormone (AMH) and Hormonal Assays for Phenotypic Classification of Polycystic Ovary Syndrome. *Ginekolo Pol.* 2020;91(11):661-7. [\[Crossref\]](#)



14. Tatar ÖB, Erginay ON, Akdaş Reis Y. Clinical and Demographic Characteristics of Patients Diagnosed with Polycystic Ovary Syndrome: A Cross-Sectional Observational Study. *Türk Kadın Sağlığı ve Neonatoloji Dergisi*. 2024;6(1):1-7. [\[Crossref\]](#)
15. Bozdag G, Mumusoglu S, Coskun ZY, Yarali H, Yildiz BO. Anti-Müllerian hormone as a diagnostic tool for PCOS under different diagnostic criteria in an unselected population. *Reprod Biomed Online*. 2019;39(3):522-9. [\[Crossref\]](#)
16. Dewailly D, Gronier H, Poncelet E, et al. Diagnosis of polycystic ovary syndrome (PCOS): revisiting the threshold values of follicle count on ultrasound and of the serum AMH level for the definition of polycystic ovaries. *Hum Reprod*. 2011;26(11):3123-9. [\[Crossref\]](#)
17. Gürsu T, Eraslan A, Angun B. Comparison of body mass index, anti-müllerian hormone and insulin resistance parameters among different phenotypes of polycystic ovary syndrome. *Gynecol Obstet Clin Med*. 2022;2(4):164-70. [\[Crossref\]](#)
18. Eralp B, Ibanoglu MC, Engin-Ustun Y. Evaluation of pregnancy and neonatal outcomes according to the phenotypic types of polycystic ovary syndrome: A prospective study. *Int J Gynaecol Obstet*. 2023;163(3):894-903. [\[Crossref\]](#)
19. Gupta M, Yadav R, Mahey R, et al. Correlation of body mass index (BMI), anti-mullerian hormone (AMH), and insulin resistance among different polycystic ovary syndrome (PCOS) phenotypes - a cross-sectional study. *Gynecol Endocrinol*. 2019;35(11):970-3. [\[Crossref\]](#)
20. Panidis D, Tziomalos K, Misichronis G, et al. Insulin resistance and endocrine characteristics of the different phenotypes of polycystic ovary syndrome: a prospective study. *Hum Reprod*. 2012;27(2):541-9. [\[Crossref\]](#)
21. Sobti S, Dewan R, Ranga S. Metabolic syndrome and insulin resistance in PCOS phenotypes. *Int J Reprod Contracept Obstet Gynecol*. 2017;6(11):5067-73. [\[Crossref\]](#)
22. Guastella E, Longo RA, Carmina E. Clinical and endocrine characteristics of the main polycystic ovary syndrome phenotypes. *Fertil Steril*. 2010;94(6):2197-201. [\[Crossref\]](#)