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Anti-Mullerian Hormone (AMH) Variability in Cases of Polycystic Ovary Syndrome (PCOS) Phenotypes

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Abstract
Background: Polycystic ovarian syndrome (PCOS) affects 8–13% of women and affects their hormones, metabolism, and mental wellbeing. The major goal of this research was to determine if anti-mullerian hormone (AMH) measurements in blood may be used to diagnose polycystic ovary syndrome (PCOS) and to predict its prognosis. **Methods:** An obstetrician-gynecologist at Benha University Hospital and one hundred of her patients took part in the investigation. The patients were divided into two equal groups. In Group I, 50 women, which was called the PCOS group, were found to have PCOS according to the Rotterdam criteria. In Group II, 50 women were used as controls. **Results:** Impressively, the AMH was able to predict PCOS with an 88% sensitivity and a 72% specificity. **Conclusions:** PCOS women had higher hormonal levels of LH, FSH, testosterone and androstenedione, and higher FG score, while had lower ovarian volume. Additionally, the current study concluded that AMH is a significant predictor of PCOS with a sensitivity of 88% and a specificity of 72%.

Keywords: Polycystic ovary syndrome, phenotypes, and anti-mullerian hormone.

Introduction

Polycystic ovarian syndrome (PCOS) affects 8–13% of women and affects on their hormones, metabolism, and mental wellbeing. The majority of cases of amulatory infertility also stem from this. This illness encompasses a wide range of phenotypes and affects women of all ages. Although gynecologists Irving Stein and Michael Leventhal were the first to diagnose polycystic ovarian syndrome in 1935, there is currently no conclusive test for the condition [1].

Irregular gonadotropin production, ovulatory dysfunction, and aberrant gonadotropin-releasing hormone pulsatility are additional risk factors for polycystic ovarian syndrome, along with hyperandrogenism and insulin resistance. Polycystic ovarian syndrome (PCOS) pathophysiology is complicated by endothelial dysfunction, high levels of oxidative stress, low-grade chronic inflammation with raised C-reactive protein levels, and other variables [2].

The anti-mullerian hormone (AMH) resembles a glycoprotein dimer in structure and is a member of the transforming growth factor-β family. On the short arm of the chromosome is where you may find the AMH-encoding gene. The cells that encase a woman's preantral and antral follicles—the ovarian granulosa—secrete AMH. Her levels naturally decline with age and become undetectable once menopause hits. Oral contraceptive pill usage has little effect on hormone levels, but there is a slight but noticeable fluctuation in concentration over the menstrual cycle [3].

During human folliculogenesis, the expression of AMH protein begins in the primary follicle stage and peaks in follicle-stimulating hormone (FSH)-dependent pre-antral and small antral follicles, which are 4 mm or less in diameter. However, it gradually decreases in subsequent stages and disappears in follicles (8 mm or greater in diameter). [4]

Polycystic ovarian syndrome (PCOS) is characterized by elevated serum AMH levels, which may indicate a role for AMH in the pathophysiology of PCOS [1]. AMH is generated by the pre-antral and small antral ovarian follicles.

After years of dismissal, Bakeer et al. [5] showed that AMH might be helpful in diagnosing polycystic ovary syndrome (PCOS) in infertile Egyptian women and reported that it is now being used more often.

Blood AMH levels were used by El-Omda et al. [6] to reach the conclusion that PCOS and serum AMH are directly related. Polycystic ovarian syndrome (PCOS) may be diagnosed and its effects predicted using AMH level, according to research by Singh et al. [4]. Since an increase in AMH value indicates the existence of hyperandrogenism, serum AMH levels provide insight into the phenotype and severity of polycystic ovary syndrome.

In order to better understand polycystic ovarian syndrome (PCOS), this research set out to investigate if blood AMH measurement may serve as a diagnostic and predictive tool.

Patients and Methods

One hundred women who visited the OB/GYN clinic at Benha University Hospitals participated in this prospective case-control study. Each patient was asked to sign a document indicating their fully informed consent. After receiving clearance from the Faculty of Medicine, Benha University Hospitals' Ethics Committee, the research was conducted.

Hyperandrogenism, defined as hirsutism (Ferriman-Gallwey score > 8), oligoand/or

anovulation, acne, seborrhea, and ultrasound-confirmed polycystic ovary (minimum of 12 follicles with 2-9 mm diameters in each 10 mm3) were all considered for inclusion in the study. Women in the control group did not have endometriosis, cysts, or any other ovarian gynecological disorders. They also had typically 26–35 day menstrual cycles, no endocrine abnormalities (normal levels of prolactin, FSH, and basal estradiol; no hyperandrogenism), and ultrasound-confirmed morphologically normal ovaries.

Those who had taken hormone replacement treatment in the three months before the start of the trial were not eligible to participate.

Grouping:

Two equal groups of patients were formed:

Using the Rotterdam criteria, 50 women were identified as having polycystic ovary syndrome (PCOS) in Group I.

Women served as controls in Group II (N=50).

We took a thorough medical history from each patient, noting [Personal history: detailed menstrual history, hirsutism, acne, infertility, galactorrhea, oligomenorrhea, amenorrhea, a patient's current complaint and how long it has lasted], current symptoms (including how long the patient has been experiencing them), past medical history (including any autoimmune diseases, diabetes, hypertension, thyroid abnormalities, or PCOS in the family), and surgical history (including any previous operations). Laboratory tests include complete blood count (CBC), erythrocyte sedimentation rate and C-reactive protein, liver and kidney functions, PT, PTT and INR, LH, FSH, testosterone, and AMH; physical examinations include a general examination that includes vital signs like blood pressure, temperature, jaundice, and signs of pallor, cyanosis, jaundice, and enlarged lymph nodes; and a pelvic and abdominal examination that includes abdominal inspection, palpation, percussion, and auscultation; and a pelvic and abdominal examination that includes pelvic and abdominal exams.

Anti-mullerian hormone (AMH)

Day 2 of menstruation was the day for basal hormone analysis, which included AMH. The ELISA kit was used to determine the level of AMH. After allowing the samples to coagulate for 2 hours at ambient temperature or overnight at 4 °C in a serum separator tube, we centrifuged them for 20 minutes at approximately 1000 × g. Either the freshly prepared serum was tested immediately or separated into portions and stored at -20 °C for future use. No repeated freezing and thawing was done. Efficient samples were not included. We took care to prevent hemolysis in the samples, as it could affect the findings. At a concentration of ovaryand/or increasing ovarian volume with a minimum size of

0.014 ng/mL, this test demonstrated analytical sensitivity. The coefficients of variance within each test were 12.3% and between each assay they were 14.2%.

AMH ELISA Kit for Anti-Mullerian Hormone Research

The notion of sandwich ELISA constituted the basis for this test. The provided microtiter plate includes a target-specific capture antibody pre-coated onto each well. The target antigen binds to the capture antibody after standards or samples are introduced to the wells. A sample or unbound standard is rinsed away. Afterwards, a detection antibody that binds to the trapped antigen was added, which is biotin-conjugated. Nothing remained of the unbound biotinylated detection antibody after washing. After that, a biotin-binding Avidin-Horseradish Peroxidase conjugate was introduced. It washed away the unbound Avidin-HRP conjugate. After that, the HRP enzyme reacted with a TMB substrate, resulting in the formation of color. Before measuring the optical density (OD) of the well at a wavelength of 450 nm ± 2 nm, a sulfuric acid stop solution was applied to end the color development process. Using known antigen concentrations, an optical density (OD) standard curve was created. By comparing the OD of an unknown sample to the standard curve, the antigen concentration can be determined.

Plasma and serum were the target samples for this test. For your convenience, we have included the sample collecting methods below. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Avoid cycles of freezing and thawing. Genomic Extracts: Gather the cells by centrifugation, then discard the liquid above them. After three washes with PBS, resuspend the cells in PBS. Four times, use an ultrasonicator to lyse the cells. Another option is to immerse the cells in -20°C water three times before thawing them to room temperature. To spin out any cell remnants, spin the mixture at 1500×g for 10 minutes at 2 - 8°C. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Keep away from cycles of freezing and thawing.

To prepare erythrocyte lysates, centrifuge whole blood at 1000×g for 20 minutes to separate the cells, and discard the supernatant. Resuspend the cells in PBS after washing them three times. Three times, freeze the cells at -20°C and then defrost them at room temperature. To extract cellular remnants, spin the mixture at 5,000×g for 10 minutes at 2-8°C. To conduct the test, separate the supernatant. Prior to running, erythrocyte lysates should be diluted using the reference

standard and the sample diluent. Keep undiluted samples at a temperature of -20°C or below. Avoid cycles of freezing and thawing.

Gather plasma via anticoagulant collection using EDTA or heparin. Perform a 15-minute

Plasma Depleted of Platelets—As an anticoagulant, collect plasma using EDTA. Perform a 15-minute centrifugation run at 1000×g at 2-8°C no later than 30 minutes after sample collection. In order to remove all platelets, it was suggested that the samples be spun in a centrifuge at 10,000×g for 10 minutes. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Avoid cycles of freezing and thawing.

To prepare serum, place samples in a serum separator tube and let them coagulate for two hours at room temperature or overnight at 4°C. Then, spin the tubes at around 1000×g for 20 minutes. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Avoid cycles of freezing and thawing. Users should independently explore tissue-specific circumstances while working with tissue homogenates, since the preparation procedures might differ based on the kind of tissue. This is only one example among many. Prior to homogenization, rinse tissues in PBS* to eliminate any extra blood. Weigh the tissues. Use a glass homogenizer set on ice to finely mince tissues and then homogenize them in 5- 10 mL of PBS. Use ultrasonication or freeze/thaw cycles of -20°C and room temperature three times to lyse the cells. Spin the mixture at 5000×g for 5 minutes to ensure homogeneity. To conduct the test, separate the supernatant. Retain undiluted samples at a temperature of -20°C or below. Keep away from cycles of freezing and thawing. Pee: Voided first pee of the day into a clean container after aseptic collection (midstream). Use a centrifuge to separate the solid from the liquid, and then save the liquid for testing. Keep undiluted samples at a temperature of -20°C or below. Keep away from cycles of freezing and thawing. Saliva, perspiration, tears, cerebral fluid, follicular fluid, lung lavage fluid, and other bodily fluids: The samples should be spun at 1000×g for 20 minutes in order to extract any particles. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Keep away from cycles of freezing and thawing. 1x PBS (0.02mol/L pH 7.0-7.2)

Before pipetting (to prevent foaming), bring all samples and reagents to room temperature without adding any further heat. Stir gently to combine. Following the instructions in the preceding sections, get all of the necessary reagents, standards, and samples ready. Before incubating for 90 minutes at 37°C, add 100µl of Standard, Blank, or Sample to each well and cover

centrifugation run at 1000×g at 2-8°C no later than 30 minutes after sample collection. To conduct the test, separate the supernatant. Keep undiluted samples at a temperature of -20°C or below. Avoid cycles of freezing and thawing.

with a plate sealer. Do not wash; instead, aspirate the contents of each well. Before covering with a plate sealer, carefully stir each well to mix thoroughly after adding 100µl of 1x Biotinylated Detection Antibody. Allow to sit at 37°C for 30 minutes. Rinse three times after aspirating the contents of each well. Wash the mixture by adding around 350 µl of Wash Buffer using a squirt bottle, multi-channel pipette, manifold dispenser, or automated washer. Before aspirating thoroughly, let each wash rest for one to two minutes. To get rid of any leftover Wash Buffer after the final wash, aspirate the plate, turn it upside down, and tap it on some clean absorbent paper.

Including 100µl of 1x HRP. Before incubating for 30 minutes at 37°C, fill up each well with the conjugate working solution and cover with a fresh plate sealer. After drawing out the contents of each well, repeat steps 4 and 5 to wash. Before incubating for 15 minutes at 37°C, fill each well with 90µl of TMB Substrate solution, and cover with a fresh plate sealer. Keep it out of direct sunlight and check on it every so often to make sure the color is developing correctly. Pour 50 microliters of Stop Solution into each well. Suddenly, the blue became yellow. If you see that the color shift is not happening evenly, press the plate lightly to make sure everything is mixed. To ensure proper mixing, apply the Stop Solution to the wells at the same time as the TMB substrate solution. Quickly use a microplate reader set to 450 nm to determine the optical density (OD) value of every well.

Sonography of the vagina

Using a vaginal probe and a frequency of 8 MhZ, the Mindray DC-70 and E6 machines from GE were used to evaluate the pelvic organs, including the uterus, endometrium, and adnexa, as well as to measure ovarian volume and antral follicle counting (AFC). This procedure was done on all patients while they were in the lithotomy position. The 8 MHz ultrasound probe was used to measure the cervical length, which is defined as the distance between the internal and external os. The value was determined by taking the minimum of three measurements. The direction of the picture was given extra attention during the insertion of the transvaginal transducer. The probe was positioned either against the external cervical OS or in the distal vagina. By gliding the probe laterally from one adnexa to the other, sagittal imaging was acquired. By angling the probe at 90 degrees, a transverse or semi-coronal orientation might be obtained. Moving the probe from the front to the back allowed for further imaging. As a preliminary

assessment, we swept the probe from midline to lateral borders at the bilateral adnexa to conduct a broad inspection. After that, the probe was turned 90 degrees and swept from front to back. After the test was done, we rinsed the probe under running water and soap to get rid of any remaining gel. It was suggested to use a high-level disinfectant after drying the probe.

Sample size calculation:

The sample size calculation was performed using G.power 3.1.9.2 (Universitat Kiel, Germany). The sample size was calculated according to the mean AMH which was higher in PCOS women with oligo-/amenorrhea compared to PCOS women with normal menstrual patterns (8.27 ± 5.05 vs. 5.34 ± 3.19), according to a previous study [7]. Based on the following considerations: 0.05 α error and 90% power of the study, allocation ration 1:1. 10 cases were added to overcome dropout. Therefore, 100 patients were allocated (50 patients in each group).

Statistical analysis:

Statistical approaches were used on the tabulated data using the statistical program for the social sciences (SPSS). Numbers and percentages were used to represent categorical data. Standard deviation (SD) and mean (SD) were used to describe continuous data. Appropriate significance tests were used. With this study, a 0.05 level of significance was considered acceptable.

Results

Table 1 demonstrates that although there was a statistically significant difference between the groups in terms of body mass index (BMI), there was no such difference in terms of age. Regarding temperature, pulse, diastolic blood pressure (DBP), and systolic blood pressure (SBP), there was no statistically significant difference between the groups that were evaluated. Laboratory examination, Ferriman-Gallwey score (FG score), and ovarian volume (ml) were among the variables where the groups were found to vary significantly statistically.

Table 1: Distribution of demographic data, distribution of vital signs, distribution of laboratory investigation, and ovarian volume (ml) between the studied groups

		PCOS group (N=50)	Control group (N=50)	Test	P value
Age		32 ± 4.5	33 ± 5.3	1.0170	0.31
BMI		27.1 ± 7.2	24.1 ± 4.1	2.5603	**0.01***
vital signs	**SBP (mm/Hg)**	101 ± 12.9	99.5 ± 11.8	0.6067	0.54
	DBP (mm/Hg)	76.3 ± 8.9	74.8 ± 8.4	0.8667	0.38
	Pulse (beat/min)	80.1 ± 11.7	77.9 ± 11.4	0.9523	0.34
	Temperature (°C)	37.1 ± 0.3	37 ± 0.3	1.6667	0.09
laboratory investigation	**LH (IU/l)**	8.5±5.2	5.2±2.4	t = 4.07	**< 0.001***
	FSH (IU/l)	6.6±2.1	5.7±2	t = 2.19	**0.03***
	Testosterone (nmol/l)	2±0.6	1.6±0.6	t = 3.33	**0.001***
	Androstenedione (ng/ml)	2.5±1	1.7±0.8	t = 4.41	**< 0.001***
FG score		11.43±7.05	6.31±3.28	t =	**< 0.001***
Ovarian volume (ml)		8.7±4.4	15±5.6	t = 6.25	**< 0.001***

Data are presented as mean ± SD or frequency (%). SD; Standard Deviation, BMI; body math index., SD: standard deviation, t: T test, FG Score: Ferriman-Gallwey score. PCOS: Polycystic Ovary Syndrome, SBP: systolic blood pressure, DBP: diastolic blood pressure, * significant as P-value ≤ 0.05.

Table 2 shows the distribution of past history in the PCOS group.

Table 2: Distribution of past history in PCOS group

	PCOS group (N=50)
Diabetes Mellitus	12 (24%)
Hypertension	10 (20%)
Thyroid Abnormalities	28 (56%)
Family History of PCOS	7 (14%)

Data are presented as frequency (%).PCOS: Polycystic Ovary Syndrome,

Table 3 shows that there was a highly statistically significant decrease in the control group than PCOS Phenotypes regarding AMH Levels..

Table 3: Distribution of AMH (ng/mL) Levels in different PCOS Phenotypes and Controls.

	AMH (ng/mL) Levels	Test	P value
Controls	5.55±3.3	_---	----
PCOS	8.61±4.72	t = 3.75	**< 0.001***
HA	8.56±4.72	t = 3.69	**< 0.001***

ANOV	8.58±4.73	t = 3.71	**< 0.001***

t: T test, PCOS; polycystic ovary syndrome, HA; hyperandrogenism, and ANOV; oligo or anovulation. AMH: Anti-Mullerian Hormone, * significant as P-value ≤ 0.05.

s**Table 4**

Table 4: ROC curve for AMH as a predictor for PCOS

AUC	S.E.	Sig.	sensitivity	Specificity
0.80	0.046	**<0.001***	88%	72%

AUC: Area under curve, S.E.: Standard errors, Sig.: significant * significant as P-value ≤ 0.05.

Discussion

Serum AMH levels are higher in women with polycystic ovary syndrome compared to people without the condition. Blood AMH levels are higher in polycystic ovarian syndrome for two reasons: first, there are more antral follicles overall, and second, each follicle produces more AMH than the last [8].

There was a statistically significant difference in BMI across the groups, but no difference in age, according to the study. Inconsistent menstrual cycles were reported by 36 (or 72%) of the patients polled, oligomenorrhea by 33 (or 66%), amenorrhea by 13 (or 26%), hirsutism by 35 (or 70%), and facial acne by 24 (or 48%). A total of 28 patients (56%) had thyroid problems; 7 patients (14%), 12 patients (24%), 10 patients (20%), and 7 patients (14%), all with a family history of polycystic ovarian syndrome, were impacted by hypertension. No statistically significant differences were found between the groups when measuring systolic and diastolic blood pressure as well as pulse and temperature.

Furthermore, there was no significant difference between the age groups as shown by Ran & Li [9].

Gorsic et al. [8] also discovered a statistically significant difference in BMI across the groups under examination, and our data support their findings. All categories were statistically significant, with the exception of age.

Our results are at odds with those of Köninger et al. [10], who discovered that the age distributions of the PCOS study's patients and controls were substantially different.

There was a very significant difference between the groups assessed in terms of laboratory examination, FG score, and ovarian volume (ml). The PCOS phenotypic group had significantly higher AMH levels than the control group.

We found similar results to those of Bhide et al. [11], who demonstrated that the groups differed considerably in AMH levels.

While Piouka et al. [12] showed that AMH levels did not differ significantly between severe and mild PCOS, there was a significant difference between the two.

From what we can tell, AMH had an 88% success rate and a 72% specificity rate in predicting PCOS.

Polycystic ovarian syndrome (PCOS) may be diagnosed and its effects predicted using AMH level, according to research by Singh et al. [4]. Since an increase in AMH value indicates the existence of hyperandrogenism, serum AMH levels provide insight into the phenotype and severity of polycystic ovary syndrome.

In line with the results of Köninger et al. [10], who demonstrated a reduced sensitivity for the identification of moderate PCOS (64% vs. 82%), we discovered that the ideal AMH threshold for mild PCOS was 3.76 ng/ml, whereas for severe PCOS it was 4.23 ng/ml.

According to Köninger et al. [10], AMH and AFC may be utilized as diagnostic tools since they are excellent indications of the severity of PCOS. Still, ovarian volume isn't a great diagnostic tool since it wasn't as helpful as AFC in terms of AUCs.

The results of the traditional AMH Gen II test were shown to be unreliable after storage, according to the current study [13].

Caveats: The sample size may be too small to draw any firm conclusions, hence there may be some meaningless results. Being a center-center investigation, there was some heterogeneity among participants.

Conclusions:

PCOS women had higher hormonal levels of LH, FSH, testosterone and androstenedione, and higher FG score, while had lower ovarian volume.The PCOS women by its different phenotypes (hyperandrogenism or ANOV) had higher AMH levels. Additionally, the current study concluded that AMH is a significant predictor of PCOS with a sensitivity of 88% and a specificity of 72%.

Findings support the hypothesis that AMH is pivotal to polycystic ovary syndrome anovulation. We hypothesize that serum AMH measurement could aid in PCOS diagnosis and prognosis prediction based on our study.

We need further studies with larger samples and longer follow-up times to confirm these results and determine whether blood AMH measurement may be used to diagnose PCOS and predict its prognosis. Future studies should employ large-scale comparative observational studies or randomized controlled trials with sufficient sample sizes. It is crucial to include patients who are representative of the population in terms of age, gender, and disease severity, and to have a sample

size large enough to make meaningful conclusions while also adjusting for confounding factors. We further recommend that future studies use multicenter designs to corroborate our findings.

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Conflict of Interest: Nil

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