

Investigating the Relationship Between Serum Anti-Müllerian Hormone Levels and In Vitro Fertilization Outcomes in Women with Polycystic Ovary Syndrome

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ABSTRACT

Background: Emerging research indicates that increased Anti-Müllerian Hormone (AMH) levels may hinder follicular responsiveness to Follicle-Stimulating Hormone (FSH), with subsequent reduction in estradiol synthesis and impaired follicle selection.

Objective: This study aimed to evaluate the association between serum AMH concentrations and outcomes of in vitro fertilization (IVF) in infertile women diagnosed with polycystic ovary syndrome (PCOS).

Patients and methods: A retrospective analysis was conducted at the Assisted Reproductive Technology Unit (ARTU) of Ain Shams University Maternity Hospital. The study included 104 anovulatory infertile females with PCOS who underwent IVF or intracytoplasmic sperm injection (ICSI) procedures.

Results: AMH levels were inversely associated by both patient age and duration of infertility ($p < 0.001$ and $p = 0.030$, correspondingly). A significant positive relationship was identified between AMH levels and antral follicle count (AFC) ($p < 0.001$). Additionally, higher AMH levels had positive correlations with the anticipated number of oocytes, actual oocyte retrieval, mature (MII) oocytes, and fertilized oocytes ($p = 0.003, 0.004, 0.018$, and 0.048 , respectively). AMH levels also demonstrated significant positive correlations with rate of biochemical pregnancy, clinical pregnancy rate (CPR), and live births rate (LBR) ($p = 0.025, 0.036$, and 0.047 , correspondingly).

Conclusion: It could be concluded that serum AMH shows moderate predictive capability for ovarian hyperstimulation syndrome (OHSS) and key reproductive outcomes, including biochemical pregnancy rate, clinical pregnancy rate (CPR), and live birth rate (LBR). No significant relationship is observed between AMH levels and either the duration or total dosage of ovarian stimulation.

Keywords: AMH, In vitro fertilization, PCOS, Reproductive outcomes, Ovarian reserve.

INTRODUCTION

Anti-Müllerian Hormone (AMH), a dimeric glycoprotein, is a member of the transforming growth factor-beta (TGF- β) family, primarily released by pre-antral and early antral follicles. It has emerged as a valid biomarker for evaluating the ovarian follicular pool ⁽¹⁾. Even though AMH is broadly utilized to evaluate ovarian reserve, its most common application is in predicting ovarian response during controlled stimulation in ART, where it informs expectations regarding oocyte yield and aids in customizing stimulation protocols ⁽²⁻³⁾.

In terms of PCOS, a condition characterized by an increase in follicular pool and frequent oligo- or anovulation, AMH has gained attention as both a diagnostic and pathophysiological marker ⁽⁴⁻⁵⁾. Since 2003, PCOS diagnostic criteria have included follicular pool assessment using transvaginal ultrasonography, and AMH has been proposed as a surrogate marker for antral follicle count (AFC) ⁽⁶⁾.

Increased AMH values in PCOS are not merely reflective of follicular excess but are believed to contribute to the follicular arrest characteristic of the syndrome ⁽⁷⁾. Studies suggest that AMH inhibits aromatase activity in granulosa cells, thus reducing estradiol production and weakening the follicular response to FSH ⁽⁸⁻⁹⁾.

This disrupts the essential FSH/estradiol feedback mechanism responsible for upregulating FSH receptors, thereby impairing follicle development ⁽¹⁰⁾. As a result, excessive AMH could have

a main role in the pathogenesis of anovulation and dysfunctional folliculogenesis in PCOS.

The aim of the current work was to evaluate the association between serum AMH concentrations and outcomes of in vitro fertilization (IVF) in infertile women diagnosed with polycystic ovary syndrome (PCOS).

PATIENTS AND METHODS

This retrospective study included a total of 94 infertile women with PCOS, attending the ARTU of Ain Shams University Maternity Hospital, Cairo, Egypt. Data collection spanned from August 2021 to February 2022.

The study included infertile women with PCOS who underwent IVF or ICSI at the hospital during the study period. Although the projected sample size was 104 participants, only 94 patient records met the strict eligibility criteria by the end of the six-month period.

Inclusion criteria: Infertile women aged 18–39 years, with a confirmed diagnosis of PCOS who had failed to conceive after completing at least three cycles of ovulation induction with oral agents followed by six cycles using gonadotropins. Additionally, their male partners were required to have normal semen parameters in agreement with the latest World Health Organization (WHO) strategies.

Exclusion criteria: Women had any of the following: systemic or endocrine diseases (poorly controlled

diabetes, liver or renal impairment, significant anemia, thromboembolic history, or uncontrolled hypertension), uterine abnormalities (such as Asherman's syndrome, uterine septum, fibroids, or adenomyosis), abnormal uterine cavity confirmed by sonohysterography, hysterosalpingography, or hysteroscopy, or contraindications to pregnancy. Other exclusions included hypersensitivity to ovulation induction agents, ovarian or abdominal conditions impeding adequate transvaginal ultrasound (TVS) assessment, tubal disease (e.g., hydrosalpinx or pyosalpinx), ovarian cysts, or history of oophorectomy.

Sampling method:

A convenience sampling technique was applied, including all eligible women undergoing IVF/ICSI during the study period.

Study procedures:

Women meeting the Rotterdam criteria for PCOS and fulfilling the study's eligibility criteria were enrolled. Retrospective collection of patients' data was conducted based on electronic medical records. Additional missing information, including pregnancy outcomes, was obtained via phone interviews.

Collected data included demographic and clinical characteristics which comprise age, body mass index (BMI), parity, and relevant lifestyle factors. Baseline hormonal profiles (FSH, LH, prolactin, estradiol, AMH, and TSH), imaging results (TVS, HSG, and hysteroscopy), details of ovulation induction protocols (types, dosages, and duration of medications), number of oocytes retrieved and percentage of mature (MII) oocytes, fertilization rates, embryo transfer (ET) details (day of transfer, number of embryos transferred), and IVF/ICSI outcomes (biochemical pregnancy rate, CPR, and LBR) were recorded.

In addition, obstetric and fetal outcomes were assessed, including maternal complications (e.g., miscarriage, preterm labor, antepartum hemorrhage, ectopic pregnancy, gestational diabetes (GDM), preeclampsia, and anemia) and neonatal outcomes (e.g., congenital anomalies, intrauterine fetal death, and NICU admission). Adverse outcomes related to ART, such as multiple gestations, heterotopic pregnancy, and prematurity, were also documented.

Ethical approval:

This study was approved by Ain Shams University's Faculty of Medicine Ethics Committee. Each participant received a full summary of the study's aims prior to completing an informed consent form. The study protocol conformed to the Helsinki Declaration, the ethical norm of the World Medical Association for human testing.

Statistical analysis

Data analysis was conducted using the SPSS, version 16.0 (Chicago, IL, USA). Continuous variables were

expressed as mean \pm SD. Comparisons between responders and non-responders were conducted using the t-test for normally distributed data, and the U test for non-parametric variables. Categorical variables were analyzed using the Chi-square (χ^2) test.

ROC curve was used to assess the predictive performance of serum AMH levels. Additionally, logistic regression analysis was applied to explore the relationship between serum AMH and other relevant variables in predicting ovarian response to human menopausal gonadotropin (HMG) stimulation. A p-value < 0.05 was considered statistically significant

RESULTS

Age demonstrated a strong, statistically significant negative correlation with AMH ($R = -0.746$, $p < 0.001$), consistent with established literature indicating that AMH levels decline with increasing age. Infertility duration also showed a mild but significant negative correlation ($p=0.03$), which would suggest a gradual decline in ovarian reserve over prolonged periods of infertility. BMI, however, showed a negligible correlation ($R = -0.042$, $p = 0.688$), indicating no meaningful association in this cohort (Table 1).

Table (1): Association between serum AMH levels and demographic characteristics of the patients

Variables	AMH	
	R	p-value
Demographic, (N=200)		
Age	-0.746	<0.001*
BMI	-0.042	0.688
Infertility duration	-0.224	0.030*

Stimulation duration and total induction dose both showed negligible and non-significant relationships with AMH ($R = -0.013$ and -0.020 , respectively; $p > 0.8$). This suggested that, within this cohort, AMH levels were not predictive of the amount or duration of gonadotropins required. AFC showed a moderately significant positive relationship with AMH ($R = 0.410$, $p < 0.001$), consistent with established evidence linking AMH with ovarian reserve markers (Table 2).

Table (2): Relationship between serum AMH levels and total induction dose, stimulation duration, and AFC

Variables	AMH	
	R	p-value
Induction, (N=200)		
Stimulation duration	-0.013	0.900
Total dose	-0.020	0.846
AFC	0.410	<0.001*

Both the number of retrieved and mature oocytes showed a significant positive correlation between serum AMH levels (Table 3).

Table (3): Relationship between serum AMH levels (ng/mL) and controlled ovarian stimulation (COS) outcomes

Outcomes	Findings				^p-value
	Positive		Negative		
Fertilization	89	4.5±2.2	5	6.5±3.7	0.057
Embryo transfer	84	4.5±2.2	10	5.2±3.2	0.382

Significant positive relationships were observed between baseline AMH and: number of expected ovum pickup, total number of oocytes retrieved, number of mature (M2) oocytes, and number of fertilized oocytes. Non-significant correlations are seen with day of ovum pickup, fertilization rate, day of ET, and ET number (Table 4).

Table (4): Association between serum AMH levels and controlled ovarian stimulation (COS) parameters

Variables	Baseline AMH	
	R	p-value
Day of ovum pickup	0.202	0.055
Number of expected ovum pickup	0.307	0.003*
Number of oocytes	0.302	0.004*
Number of M2 oocytes	0.247	0.018*
Fertilization, (N=89)		
Number of fertilized oocytes	0.210	0.048*
Fertilization rate	0.012	0.909
Embryo transfer, (N=84)		
Day of embryo transfer	0.013	0.906
Number of transferred embryos	-0.038	0.730

Table (5) shows diminished pregnancy outcomes at elevated AMH levels.

Table (5): Association between serum AMH levels and pregnancy outcomes

Outcomes	Findings				^p-value
	Positive		Negative		
Biochemical pregnancy	69	4.9±2.1	25	3.7±2.7	0.025*
Clinical pregnancy	59	5.0±2.1	35	3.9±2.6	0.036*
Live birth	57	5.0±2.1	37	4.0±2.6	0.047*

Table (6) shows significant positive correlation between serum AMH levels and pregnancy complications.

Table (6): Relationship between serum AMH levels (ng/mL) and maternal, IVF-related, and fetal complications

Outcomes	Findings				^p-value
	Positive		Negative		
Abortion	5	4.7±3.2	89	4.6±2.3	0.893
Preterm labor	9	4.8±2.2	85	4.6±2.4	0.750
Pregnancy induced hypertension (PIH)	4	3.2±1.3	90	4.7±2.4	0.237
Gestational DM (GDM)	6	4.2±1.1	88	4.6±2.4	0.683
Antepartum hemorrhage	7	3.8±1.4	87	4.7±2.4	0.372
Ovarian hyper-stimulation syndrome	8	7.1±2.0	86	4.4±2.2	0.001*
Multiple pregnancy	7	3.7±1.7	87	4.7±2.4	0.303
NICU admission	6	4.1±1.6	88	4.6±2.4	0.617

Table (7) shows a significant positive relationship between serum AMH levels and pregnancy complications.

Table (7): Diagnostic accuracy of serum AMH levels in predicting various clinical outcomes

Factors	AUC	SE	P-value	95% CI	Cut point
Biochemical pregnancy	0.884	0.073	0.007*	0.641–1.000	≥2.4 & ≤8.1
Clinical pregnancy	0.850	0.065	0.015*	0.722–0.978	≥2.4 & ≤8.1
Live birth	0.845	0.063	0.016*	0.722–0.968	≥2.8 & ≤8.1
OHSS	0.821	0.062	0.003*	0.700–0.942	≥5.1

Table (8) shows AMH cut-off levels between 2.4–8.1 ng/mL offer moderate predictive value for key outcomes, including OHSS, pregnancy rates, and live births.

Table (8): Predictive value of AMH ≥ 2.4 ng/mL for pregnancy outcomes

Characteristics	Biochemical pregnancy AMH ≥ 2.4 & ≤ 8.1	Clinical pregnancy AMH ≥ 2.4 & ≤ 8.1	Live birth AMH ≥ 2.8 & ≤ 8.1
Sensitivity	85.4%	86.3%	88.4%
Specificity	52.0%	45.7%	51.4%
DA	69.1%	64.9%	61.7%
YI	27.4%	22.0%	19.8%
PPV	81.3%	70.3%	68.4%
NPV	83.3%	85.3%	81.4%
LR+	1.57	1.40	1.41
LR-	0.47	0.52	0.61
DOR	3.31	2.71	2.29

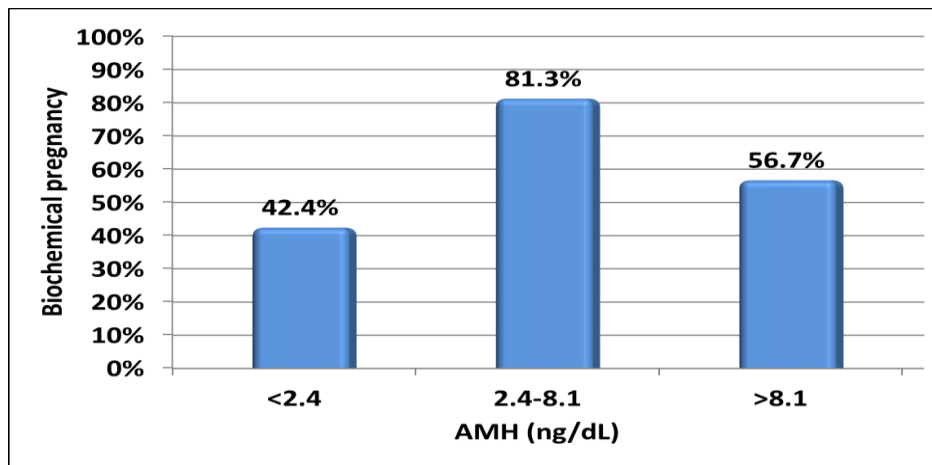


Figure (1): Correlation between extremes of AMH level and biochemical pregnancy.

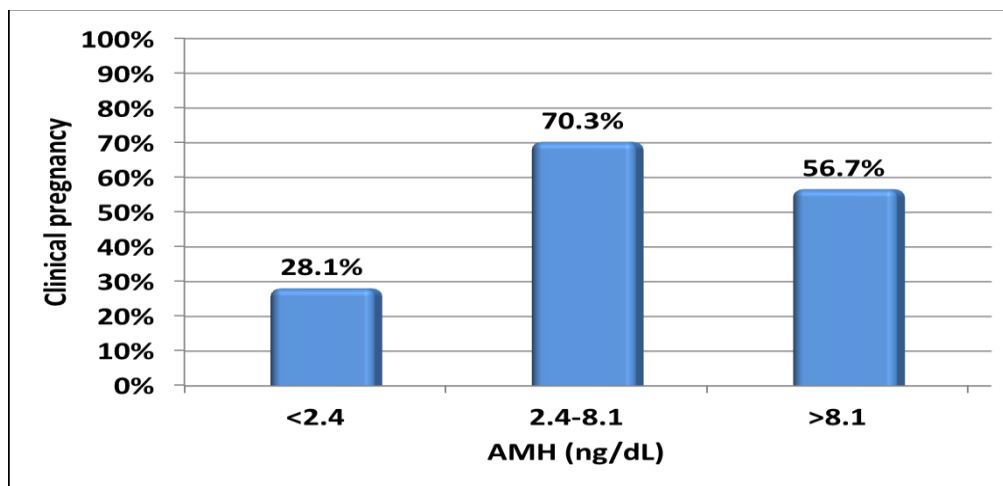


Figure (2): Correlation between extremes of AMH level and clinical pregnancy

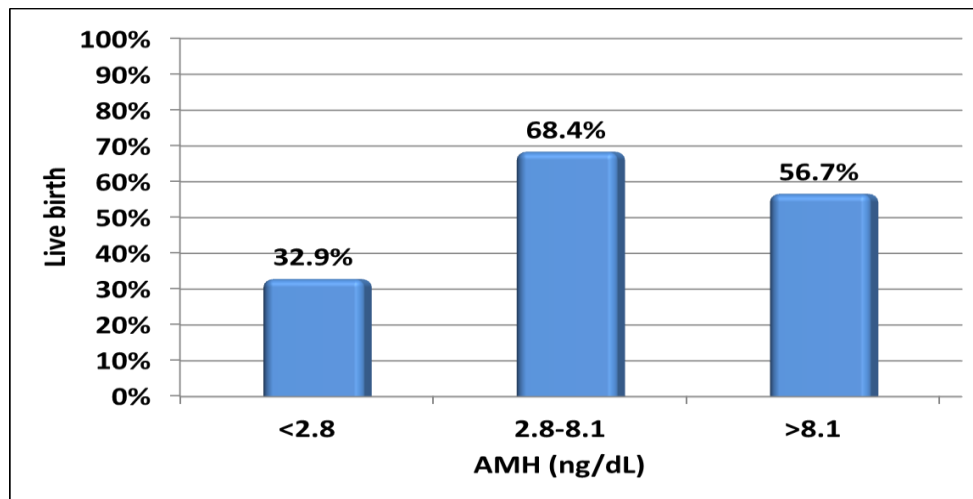


Figure (3): Correlation between extremes of AMH level and live birth rate.

DISCUSSION

The present prospective study explored the relationship between AMH levels and various clinical outcomes in females undergoing ICSI. Our findings corroborated those of **Umarsingh et al.**⁽¹¹⁾ who recorded an insignificant negative relationship between AMH levels and age, suggesting a trend toward reduced ovarian reserve with advancing maternal age.

Similarly, the findings of **Oke et al.**⁽¹²⁾ aligned with ours, indicating an inverse association between AMH levels and both age and duration of infertility. **Sun et al.**⁽¹³⁾ who analyzed the influence of AMH on oocyte yield during controlled ovarian hyperstimulation (COH), also observed that infertility duration increased with age, particularly in women aged 36 to 43 years.

Our results were consistent with **Hussein et al.**⁽¹⁴⁾ who identified a significant negative relationship between AMH and maternal age. Additionally, **Albu and Albu**⁽¹⁵⁾ found a positive relationship between AMH and BMI, particularly in younger females with normal weight and ovarian reserve. However, **Bernardi et al.**⁽¹⁶⁾ challenged these findings, suggesting that obesity might exert a cumulative negative effect on ovarian reserve, as evidenced by lower AMH concentrations among obese participants.

The current study is further reinforced by **Permadi et al.**⁽¹⁷⁾ who demonstrated a significant positive relationship between AMH, AFC, and the number of retrieved oocytes. **Zhang et al.**⁽¹⁸⁾ also highlighted the interrelationship between AMH and AFC, noting discrepancies between the two indicators clinically.

In cases with PCOS, AMH's predictive value appeared less clear. **Mumford et al.**⁽¹⁹⁾ and **Amer et al.**⁽²⁰⁾ both noted that elevated AMH levels were associated with reduced sensitivity to ovulation induction, possibly necessitating higher doses of gonadotropins. **Guo et al.**⁽²¹⁾ didn't detect a significant relationship between baseline AMH levels and fertilization or embryo availability rates, a finding partly inconsistent with our observations. Similarly, **Tal et al.**⁽²²⁾ found no consistent trend in fertilization rates across different AMH groups

but reported a higher number of oocytes retrieved with increasing AMH.

Umarsingh et al.⁽¹¹⁾ recorded a weak, insignificant relationship between AMH and fertilized oocytes, which contrasts with the current study's findings. On the other hand, **Arabzadeh et al.**⁽²³⁾ supported our data by identifying a significant positive relationship between serum AMH levels and both the number of retrieved and mature oocytes.

Discrepancies in pregnancy outcomes related to AMH levels were evident in the literature. **Guo et al.**⁽²¹⁾ and **Tal et al.**⁽²²⁾ both recorded lower LBR and CPR among females with higher AMH levels, echoing our finding of diminished pregnancy outcomes at AMH levels >8.1 ng/mL. Conversely, **Umarsingh et al.**⁽¹¹⁾ and **Arabzadeh et al.**⁽²³⁾ didn't find statistically significant associations between AMH and clinical pregnancy outcomes.

Regarding OHSS, our results agreed with those of **Stracquadanio et al.**⁽²⁴⁾ who noticed that OHSS occurred primarily in females with high serum AMH. Several studies, including those by **Lee et al.**⁽²⁵⁾, **Singh and Singh**⁽²⁶⁾, and **Aghssa et al.**⁽²⁷⁾ emphasized the predictive value of AMH in assessing OHSS risk. **Aghssa et al.**⁽²⁷⁾, for example, suggested a high risk of OHSS at AMH levels exceeding 6.95 ng/mL.

Pregnancy complications which included ectopic pregnancy, GDM, and hypertensive disorders were linked to AMH and PCOS status. **Wang et al.**⁽²⁸⁾, **Yan et al.**⁽²⁹⁾ and **Bagegni et al.**⁽³⁰⁾ recorded higher incidences of these complications among PCOS patients, in line with the risk elevation noted in our study.

Diagnostic performance of AMH in predicting pregnancy and stimulation outcomes has been variably supported. For instance, **Amer et al.**⁽²⁰⁾ identified AMH cutoffs that predicted poor ovarian response, while **Sahmay et al.**⁽³¹⁾ highlighted AMH as a significant independent predictor of CPR. **Seckin et al.**⁽³²⁾, however, demonstrated that AMH wasn't predictive of pregnancy in females undergoing intrauterine

insemination (IUI), likely due to differences in treatment modalities.

Sahmay *et al.* ⁽³¹⁾ and **Negjyp** ⁽³³⁾ further endorsed the AMH role as a prognostic marker, particularly in women of advanced reproductive age. These findings reinforced the notion that moderate AMH levels are more conducive to favorable outcomes, whereas extremely high or low levels may be detrimental.

CONCLUSION

It could be concluded that serum AMH levels have an inverse relationship with maternal age and infertility duration. While AMH does not significantly impact the duration or total dose of ovulation induction, it is positively accompanied by biochemical pregnancy rate, CPR, and LBR. High AMH levels, particularly those exceeding 5.1 ng/mL, are significantly correlated with increased risk of OHSS. Diagnostic performance analysis indicates that AMH cut-off levels between 2.4 and 8.1 ng/mL offer moderate predictive value for key outcomes, including OHSS, pregnancy rates, and live births. However, extremely elevated AMH levels (>8.1 ng/mL) are associated with poorer reproductive outcomes and higher OHSS risk. Thus, maintaining AMH levels within an optimal range may be critical in achieving successful IVF outcomes and minimizing complications.

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