Original paper

# Anti-müllerian Hormone Associated with Antral Follicular Count in Cases of Polycystic Ovary Syndrome

**Raghad Numan Talib[1], Maysaloon Adnan Abdul Razzak[2]**

[1]Karbala health directorate/ Karbala/ Iraq
[2]College of Medicine/ University of Kerbala/ Karbala/ Iraq

**Article information:**
**Received:** 2022-10-13
**Accepted**: 2022-12-22
**Vol. 15, No. 1, DEC, 2022.**

**Correspondence:** Maysaloon Adnan Abdul Razzak
**Email:** maysaloon07707@gmail.com

## Abstract

**Background:** Anti-Müllerian hormone (AMH) synthesized in the "granulosa cells" of the ovarian graffian follicles and can suppress the growth of primordial follicles.
**Objective**: To assess the AMH association with antral follicular count on the 2nd menstrual day to evaluate its diagnostic potency in the definition of polycystic ovary syndrome (PCOS).
**Patients and methods:** |A case control study, involving two groups of total hundred infertile women. The first group included fifty PCOS cases, while the controls included 50 non-PCOS infertile cases. Detailed history was obtained from candidates, and on the 2-3 cycle day underwent the following hematological tests: testosterone, follicular stimulating hormone, AMH, luteinizing hormone, and estradiol. As well, transvaginal ultrasonography was performed to assess hormonal/follicular association evaluated by the count of early antral follicles.
**Results:** The AMH levels in the PCOS were significantly higher compared to the controls (13.8±5.9 vs**.** 6.0±2.6ng/ml), correspondingly. A significant increase in the follicles' count among PCOS women compared to control (17.6±6.8 vs. 8.0±3.8), respectively. AMH correlated significantly high with follicular count in the PCOS group. The levels of AMH and testosterone were significantly correlated in the PCOS group.
**Conclusion:** During the initial follicular phase, AMH in PCOS is strongly correlated with antral follicle status. Such a link is stronger when compared with other hormones affecting follicular function. This could highlight the importance of AMH concentration as superior predictor of the early follicular counts than traditional hormonal assays. Hence, AMH levels offer a good diagnostic potency in the definition of PCOS.
**Keywords:** PCOS, antral follicle count, (AFC), AMH, and infertility.

## Introduction

Infertility is distressing nearly 15%- 20% of couples attempting gestation as a common medical problem [(1)]. Polycystic ovarian syndrome (PCOS) represents a major reason for female infertility and a common gynecological illness distressing around 15% of females during reproductive years [(2)]. PCOS is a multifaceted, heterogeneous syndrome of undefined etiology, nevertheless, environmental, lifestyle factors, and hereditary, remain controversial [(3)]. PCOS is characterized by the incidence of the following as defined by "Rotterdam criteria, National institute of health (NIH), and Androgen Excess PCOS Society": oligo/anovulation, androgen excess and micropolycystic syndrome (ovarian size more than 10ml and/or >12ovarian follicles), exclusion of other causes of polycystic ovaries [(4, 5)].Women with PCOS usually will consult for various extents of androgen excess (acne, hirsutism, seborrhea), menstrual disorders, overweight, and infertility [(2)].

Anti-müllerian hormone (AMH) is a valuable laboratory investigation for infertility assessment. AMH is synthesized by granulosa cells (of early ovarian follicles) and used to assess the reserve of the ovaries. AMH is one of the "transforming growth factor-β (TGF-β) family" that also includes activin and inhibin [(6)]. AMH serum concentrations remain steady through menstruation since its synthesis is independent of gonadotropin levels [(7)]. Patients with PCOS reveal AMH values 2-3 times more than non-PCOS females owing to increased antral follicles. The raised AMH levels revealed an association with the severity of PCOS in other studies [(8)].Transvaginal ultrasonography (TVUS) is a useful tool to assess the endometrial thickness,

antral follicle count (AFC), and analyze uterine anomalies [9].

Reduced AFC in infertile women shows that factors influencing the quantity of surviving follicles in younger women also affect the quality of oocytes and the likelihood of conception [10]. A number of non-invasive indicators, such as FSH, E2, inhibin B, and AMH and AFC, have been developed to evaluate "ovarian reserve" [11]. However, some researchers contend that the quantity rather than the quality of the surviving oocytes is what the ovarian reserve predominantly indicates[12]. According to a number of studies, ovarian reserve testing does not accurately predict whether women who use assisted reproductive technologies will become pregnant or experience a miscarriage [13]. Others, however, contend that high FSH, a sign of inadequate ovarian reserve, is connected to a delay in spontaneous conception regardless of age, an increase in miscarriages, and an earlier age of menopause [14]. AMH is a promising diagnostic tool for PCOS as an addition to the current Rotterdam criteria, despite its low sensitivity and specificity and the fact that no single AMH cut-off is diagnostic for the condition [15].

Given this ongoing debate and the scant information currently available, the objective of this study is to assess the relationship between serum AMH levels and AFCs in cases of PCOS.

## Patients and Methods

### Sampling and setting

A case-control within cross-sectional approach study. It was conducted on the patients attending Babylon Maternity and Pediatric Teaching Hospital, throughout the period from (September 2020 to December 2021). The patient group included 50 infertile women (primary and secondary) with PCOs cases diagnosed by the gynecologists at the hospital based on the Rotterdam criteria [5] irrespective to other clinical or medical backgrounds of the females. In females with menstrual abnormalities, the previous cycle whether spontaneous or induced by progesterone (10mg/day for one week) was considered. Those with any of the following were omitted from this study: unexplained infertility, endometriosis, hypothalamic amenorrhea, and any women with a minimum of one follicle more than 9mm diameter at sonography or plasma estradiol ($E_2$) concentration exceeding 80pg/ml. The control group comprised 50 infertile women with normal menstruation and without any prior hormonal therapy.

### History and clinical examination

All candidates underwent detailed obstetric and gynecological history. They were comforted regarding the privacy and secrecy of their data and written consent was taken from them. Women were assessed during days 2-3 of the follicular phase. A thorough clinical and physical examination was done. Body mass index (BMI) was measured by calculating the body weight and the patient height.

*Biochemical assays*

Hematogenous early-morning sampling was drained during the days 2-3 of the last menstrual cycle (early follicular phase) for the measurement of AMH, Testosterone, Estradiol, LH, and FSH according to the provider's directions. In the PCOS group, the latest period was either natural or induced by the progesterone administration (10mg/day for one week). Any Women with estradiol levels exceeding 80pg/ml were not enrolled. Blood AMH levels were measured through the enzyme immunoassay AMH provided by "Immunotech®, Beckman Coulter company, France". LH, FSH, and E2 were evaluated using the VIDAS technique using enzyme-linked fluorescent assay by Biomerieux®, France.

### Ultrasonographic study and antral follicular count (AFC)

Transvaginal ultrasonography (TVUS) was done on the day of blood sampling, using a "5-MHz vaginal probe, (Philips HD11XE®, Japan)". Real-time measures were made using the best possible ovarian view. A transvaginal scan of the early follicular phase measures the total number of follicles measuring 2-10 mm in diameter. According to the size of the residual follicular pool and the number of oocytes extracted after stimulation, the number of antral follicles (AFC) is correlated with both of these factors. The follicular counts were counted by scanning the two ovaries individually from the inner to the outer borders longitudinally. All ovarian follicles measuring 2.0-9.0mm were totaled. If the dominant follicle was >9mm, the TVUS exam was repeated in the next cycle.

*Statistical method*

Statistical evaluations were done using the SPSS Statistics V-20. The descriptive examination was applied to display the means and SD for BMI, ages, and biochemical assays. Contrasts of any two independent sets were prepared through the Student T-test.

The association between AMH and other study variables were evaluated by the Pearson correlation (correlation coefficient). A P≤0.01 reflected highly significant and p ≤ 0.05 significant values.

## Results

### Demographic characteristics

The study comprised a total of 100 women aged 20-44 year; the mean age and SD (31.3±4.3year). They were divided into two groups as shown in table-1. The control set comprised 50 patients (21.4-43.0 years old) and a mean of 34.2±5.4 year presented with infertility due to non-PCOS causes. 28 (56%) women were complaining of primary infertility and 22 (44%) women with secondary infertility of 2-9.4 year duration. Their BMI ranged between 22.7 and 32.4 Kg/m$^2$, and in 76% of them the BMI was > 25 kg/m$^2$.

The fifty PCOS women had ages ranging from 20.5 to 41.8 year, and a mean of 32.4± 6.2 year. There BMI ranged from 20 to 38.4 kg/m$^2$ and in 84% of them the BMI was > 25kg/m$^2$. Primary infertility was the main complaint of 60% of them for a mean duration of 5.4±2.8 year. There were no statistically significant variations in the mean of the two study groups for the age, BMI, parity, and duration of parity. Sixteen of the PCOS women had amenorrhea (30%), 26 of them had oligomenorrhea (62%) and eight (8%) of them had a regular cycle (table-1).

Table (2) shows the menstrual status of the PCOS group as long as the entire control group had a regular cycle.

Table-3 displays significant differences in testosterone levels between the group of PCOS and the control group (P-0.02) with highly significant differences in AMH level and AFC between the two study groups, (0.001 and 0.001, respectively). Distribution of Anti-Mullerian hormone with age and BMI between the study groups

There was a highly significant differences (P-0.001) in the levels of AMH among PCOS women aged 25-29 year. As well, there were highly significant differences in the levels of AMH among PCOS women within the BMI subgroups (25-35 Kg/m$^2$), (table-4).

Pearson correlation revealed that the increase in LH level (P-0.07) and the decrease in FSH and estradiol levels were not significant (P-0.08 and P-0.11), correspondingly. However, a significant correlation between AMH and testosterone hormone (P-0.05) in the cases of PCOS was observed (table 5). There was a significant correlation between the levels of testosterone hormone and the AFCs in the control group and the PCOS group where both of them already increased in the PCOS group, table 6.

**Table 1.** The Demographic features of the PCOS and control groups

| **Characters** | | **PCOS women N=50** | | **Control women N=50** | | **P-value** |
|---|---|---|---|---|---|---|
| | | Number | Percentage | Number | Percentage | |
| **Age groups (year)** | 20-24<br>25-29<br>30-34<br>≥35 | 10<br>15<br>12<br>13 | 20<br>30<br>24<br>26 | 10<br>8<br>12<br>20 | 20<br>16<br>24<br>40 | 0.111 |
| **BMI groups Kg/m$^2$** | 20-24<br>25-29<br>30-34<br>≥35 | 8<br>19<br>12<br>11 | 16<br>38<br>24<br>22 | 12<br>17<br>21<br>0 | 24<br>34<br>42<br>0 | 0.130 |
| **Parity** | $P_0$<br>$P_1$<br>$P_2$ | 32<br>13<br>5 | 64<br>26<br>10 | 28<br>13<br>9 | 56<br>26<br>18 | 0.116 |
| **Type of infertility** | **Primary**<br>**Secondary** | 30<br>20 | 60<br>40 | 28<br>22 | 56<br>44 | 0.223 |
| **Duration of infertility (year)** | **1-5**<br>**6-10**<br>**≥11** | 23<br>20<br>7 | 46<br>40<br>14 | 25<br>15<br>10 | 50<br>30<br>20 | 0.343 |

*The hormonal and ultrasound criteria in PCOS and control groups*

**Table 2.** The menstrual status in the PCOS group

| PCOS Group (N-50) | | |
|---|---|---|
| Menstrual status | N | Percentage |
| Amenorrhea | 16 | 30 |
| Oligomenorrhea | 26 | 62 |
| Regular cycle | 8 | 8 |

**Table 3.** The differences in hormonal level (mean± SD) and AFC between study groups

| Variables | Patient N=50 | Control N=50 | P-Value |
|---|---|---|---|
| **FSH (mIU/l)** | 6.9±1.1 | 7.5±1.9 | 0.1 |
| **LH (mIU/l)** | 9.4±3.2 | 8.5±2.4 | 0.1 |
| **$E_2$ (pg/ml)** | 12.3±9.6 | 14.9±9.6 | 0.2 |
| **Testosterone (ng/ml)** | 0.9±0.7 | 0.7±0.2 | 0.02 |
| **AMH (ng/ml)** | 13.8±5.9 | 6.1±2.6 | 0.001 |
| **Antral follicles Counts** | 17.7±6.7 | 8.1±3.8 | < 0.001 |

**Table 4.** The distribution of AMH, age groups, and BMI between the study groups, (values are shown as mean ± SD)

| Characteristics | AMH (ng/ml) | | *P*-value |
|---|---|---|---|
| | Patient N=50 | Control N=50 | |
| **Age Group (year)** | 3.78 5.70) Total (± | (6.02 ± 2.59) Total | < 0.001 |
| **20-24** | 9.60 ± 3.81 | 6.13 ± 2.70 | 0.210 |
| **25-29** | 16.25 ± 4.80 | 5.96 ± 2.96 | 0.001 |
| **30-34** | 14.66 ± 6.66 | 5.2 ± 2.15 | 0.001 |
| **≥35** | 13.17 ± 5.80 | 6.35 ± 2.71 | 0.001 |
| **BMI Groups ($kg/m^2$)** | Total (29.93±5.90) | Total (28.54±2.43) | |
| **20-24** | 9.15 ± 3.92 | 4.57± 2.14 | 0.081 |
| **25-29** | 15.13 ± 6.59 | 4.74 ± 1.62 | 0.001 |
| **30-34** | ± 4.87 TZ14.95 | 8.56 ± 2.23 | 0.001 |
| **≥35** | 15.65 ± 5.17 | N=0 | - |

*The correlation between AMH and other hormones among study groups*

**Table 5.** The correlation between levels of AMH and other hormones among the study groups

| Hormones | Pearson correlation | Patient N=50 | Control N=50 |
|---|---|---|---|
| **FSH (mIU/l)** | *r* | -0.45 | -0.18 |
| | *p* | 0.080 | 0.211 |
| **LH (mIU/l)** | *r* | 0.74 | -0.166 |
| | *p* | 0.061 | 0.250 |
| **Estradiol (pg/ml)** | *r* | -0.39 | 0.1 |
| | *p* | 0.091 | 0.970 |
| **Testosterones (ng/ml)** | *r* | 0.25 | 0.63 |
| | *p* | 0.050 | 0.060 |

*The correlation between the AFC and hormonal level*

**Table 6**. Correlation between the hormonal levels and AFCs in PCOS and control group

| Hormones | Pearson correlation | Patient N=50 | Control N=50 |
|---|---|---|---|
| **FSH (mIU/l)** | *r* | 0.22 | 0.03 |
| | *p* | 0.112 | 0.801 |
| **LH (mIU/l)** | *r* | 0.24 | 0.04 |
| | *p* | 0.112 | 0.813 |
| **E2 (pg/ml)** | *r* | 0.32 | 0.04 |
| | *p* | 0.083 | 0.857 |
| **Testosterone (ng/ml)** | *r* | 0.39 | 0.04 |
| | *p* | **0.006** | 0.824 |

*Correlation between AFCs & AMH among study groups*

Antral follicular counts were significantly correlated with AMH in healthy infertile women (P-0.01). However, the correlation was highly significant (P-0.001) in cases of PCOS, (table-7). Correlation tables and data within are non-clear and inappropriate and

Table 5, 6, 7, better to be removed as currently tables are not correct or revised (yes, we apologies for this inappropriateness, it was revised accordingly)

**Table 7.** The correlation between the levels of AMH and the antral follicles count

| | Pearson correlation | Patient | Control |
|---|---|---|---|
| **AFCs** | ***r*** | 0.85 | 0.36 |
| | ***p*** | **0.001** | **00.01** |

## Discussion

This case-control study intended to assess the relationship between serum concentrations of AMH with the AFCs on days 2-3 of the cycle among infertile women with PCOS. The main findings were significantly higher values of AMH levels and AFC, and significantly higher testosterone levels among the PCOS patients compared with the control women. As well, a significant correlation between the levels of AMH and testosterone hormone among PCOS women was reported. Moreover, the correlation between AFC and plasma AMH in the group of PCOS was highly significant and significant in healthy women.

PCOS women have a higher BMI than that of the control which might owe to the pathophysiology of PCOS due to increasing insulin-resistance and hyperandrogenemia [(16)]. The study revealed a highly significant association between AMH with women aged 25 years and more with PCOS.

There were non-significant variations between the two groups regarding the type of infertility (primary or secondary) because the choice of the participants was according to the cause not on the type of infertility. This was the explanation for no variations in the parity of the two groups.

Several scholars have suggested that AMH is a probable pointer for the follicular pool. AFCs were closely linked with the AMH plasma levels on day 3 of the menstrual cycle in infertile patients, in comparison with other hormones like estradiol, and FSH [17]. Later, several revisions found a significant increase in AMH values in PCOS women was related to the AFCs [18, 19].

Results of the control patients in the current study approve the previous reports that displayed a correlation between AMH and the small AFCs found in normal women [20]. Van Rooij and his colleagues reported that levels of serum AMH were a good predictor of the count of recovered oocytes [21].

Hence, all the aforementioned data led to the assumption that AMH is a biological indicator of the early follicular phase AFC in fertile and PCOS infertile women. Besides, it was revealed that AMH level was greatly reproducible between cycles, a point that emphasizes its strength as a biomarker of the AFC (22).

A closer look at the current study revealed that AMH was associated significantly with early AFCs than did the other hormones (FSH, LH, and $E_2$) that modulate their stimulation by FSH [23, 24]. This denotes that serum $E_2$ values not just depend on the masses of the existing active follicular granulosa cells, (as expressed by follicular count and size, but on their hormonal stimulation by FSH as well.

Consequently, serum AMH levels may signify a more reliable and independent biomarker of early antral follicle physiology than FSH and $E_2$ on cycle days 2-3. The higher basal AMH values in the PCOS women as compared with normal control of this study confirmed the results of previous researchers who attributed these results to the great AFCs of that observed in PCOS characteristically [25]. Nevertheless, it is worth recalling that neither a raised AMH level nor a raised AFCs is by itself adequate to identify PCOS. Hence, both of these criteria should be integrated into the Rotterdam description.

The current study put forward the claim that the AMH levels were closely associated with hyperandrogenemia (high testosterone levels). This is consistent with a recent study that also concluded a positive link between the blood androgens level with AMH in PCOS [26]. Testosterone can stimulate the growth of ovarian follicles by enhancing the biological actions of FSH and insulin-like growth factor-1 [27, 28]. The excessive ovarian production of AMH by ovarian granulosa cells may explain the hyperandrogenism of PCOS.

Overall, the link between AMH with sex body hormones is complex and varying, hence, additional studies are required to specify the correlation. Concordant to our outcomes, Lv PP and his colleagues as well, revealed a positive close association between serum AMH and testosterone levels in PCOS women but not in controls [28]. This could signify a positive impact of AMH on "theca interna cells" causing testosterone over secretion and a subsequently increased sum of early growing ovarian follicles, and hence ovarian hyperandrogenism and the biological process of terminal follicular maturation are the most affected.

Alternatively, the available piece of data shows that AMH suppresses the androgen conversion to $E_2$ via down-regulating the gene expression of the aromatase enzyme [28]. This supports the current study finding of a negative link between plasma levels of AMH and $E_2$ among PCOS women.

Along similar lines, higher AMH values presented by Cook et al. and in the present study were linked with lower levels of FSH and $E_2$ [25]. It is therefore attractive to postulate that a high serum AMH is enrolled in the deficiency of FSH-induced aromatase action that describes follicular arresting of the PCOS.

Of note, and since AMH is an initial product (compared to $E_2$) from the ovarian follicles, these differences could reflect alterations in the sampling day. In some studies, blood was sampled randomly from oligomenorrheic patients, while our women were tested on days 2-3 after either spontaneous or progestin-induced cycles. Therefore, the means of $E_2$ levels were higher in the other revisions [29].

Ovarian follicles obtained from experimental AMH-knockout-mice have been displayed to be more FSH-sensitive than those obtained from the wild-

type mice; signifying that the AMH suppressing effect on body aromatase enzyme acts via a decline in sensitivity of granulosa cells to FSH. According to the findings, the infantile increase in FSH levels decreases AMH expression in preantral/early antral follicles, favoring Cyp19a1 aromatase expression and E2 synthesis. This, together with recent findings that AMH can act on both the hypothalamus and the pituitary to boost gonadotropin levels, implies that AMH is a major regulator of the gonadotropic axis throughout the infantile period, leading to adult reproductive function programming [30].A prior published study described the highest expression of AMH in small follicles as slowly disappearing from large-sized FSH-dependent follicles.

Based on the evidence currently available, it seems fair to suggest that AMH serum levels and AFCs were higher in PCOS women and associated with each other positively. This approves that a large count of small antral follicles in general, associated with the polycystic histological appearance of the ovary, is behind the raised AMH synthesis [31, 32], a finding that agreed with the current study.

The idea that the smaller antral follicles may not always be seen by TVUS scanning and do contribute to the AMH values may help to explain some of our findings and raise questions about the role of serum AMH in PCOS diagnosis. However, additional future well-designed large cohorts could further validate the association of AMH with AFCs in PCOS.

## Conclusion

The levels of serum AMH in PCOS is intensely correlated with the status of AFCs throughout the initial follicular phase. The association is stronger than that detected with other body hormones of follicular function. Serum AMH measure in PCOS on days 2-3 of the cycle is a superior predictor of the early AFCs than traditional hormonal assays. Hence, AMH serum levels offer a good diagnostic potency in the definition of PCOS.

## References


1. Al-Bdairi A, Makki HA, Al-Shalah MA. Preoperative Measures of Serum Inhibin B, and FSH Levels Predict Sperms Retrieval Outcome in Non-Obstructive Azoospermic Males. Clin Schizophr Relat Psychoses. 2021;15:1-5.
2. Collée J, Mawet M, Tebache L, Nisolle M, Brichant G. Polycystic ovarian syndrome and infertility: overview and insights of the putative treatments. Gynecological Endocrinology. 2021;37(10):869-74.
3. Al-Bdairi AA, Al-kadhim HK, Al-Shaikh SF, Al-Hindy HA. ABO Blood grouping and Rhesus factor: Association with ovarian reserve and the outcomes after in-vitro fertilization. History of Medicine. 2022;8(1):18-28.
4. March WA, Moore VM, Willson KJ, Phillips DI, Norman RJ, Davies MJ. The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. Human reproduction (Oxford, England). 2010;25(2):544-51.
5. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Human reproduction (Oxford, England). 2004;19(1):41-7.
6. Howard JA, Hart, K. N., & Thompson, T. B. Molecular Mechanisms of AMH Signaling. Frontiers in Endocrinology. 2022;13.
7. Practice Committee otASfRM. Definitions of infertility and recurrent pregnancy loss: a committee opinion. Fertility and sterility. 2013;99(1):63.
8. Sahmay S, Aydogan Mathyk B, Sofiyeva N, Atakul N, Azemi A, Erel T. Serum AMH levels and insulin resistance in women with PCOS. European journal of obstetrics, gynecology, and reproductive biology. 2018;224:159-64.
9. Moorthy RS. TRANSVAGINAL SONOGRAPHY. Med J Armed Forces India. 2000;56(3):181-3.
10. Rosen MP, Johnstone E, Addauan-Andersen C, Cedars MI. A lower antral follicle count is associated with infertility. Fertility and sterility. 2011;95(6):1950-4, 4.e1.
11. Lambalk CB, van Disseldorp J, de Koning CH, Broekmans FJ. Testing ovarian reserve to predict age at menopause. Maturitas. 2009;63(4):280-91.
12. Broekmans FJ, Kwee J, Hendriks DJ, Mol BW, Lambalk CB. A systematic review of tests predicting ovarian reserve and IVF outcome. Hum Reprod Update. 2006;12(6):685-718.
13. Haadsma ML, Groen H, Fidler V, Seinen LH, Broekmans FJ, Heineman MJ, et al. The predictive value of ovarian reserve tests for miscarriage in a population of subfertile ovulatory women. Human reproduction (Oxford, England). 2009;24(3):546-52.
14. Kok HS, van Asselt KM, van der Schouw YT, Grobbee DE, te Velde ER, Pearson PL, et al. Subfertility reflects accelerated ovarian ageing. Human reproduction (Oxford, England). 2003;18(3):644-8.
15. Saxena U, Ramani M, Singh P. Role of AMH as Diagnostic Tool for Polycystic Ovarian Syndrome. Journal of obstetrics and gynaecology of India. 2018;68(2):117-22.
16. Zeng X, Xie YJ, Liu YT, Long SL, Mo ZC. Polycystic ovarian syndrome: Correlation between hyperandrogenism, insulin resistance and obesity. Clinica chimica acta; international journal of clinical chemistry. 2020;502:214-21.
17. Fanchin R, Schonäuer LM, Righini C, Guibourdenche J, Frydman R, Taieb J. Serum anti-Müllerian hormone is

more strongly related to ovarian follicular status than serum inhibin B, estradiol, FSH and LH on day 3. Human Reproduction. 2003;18(2):323-7.
18. Zhang Y, Xu Y, Xue Q, Shang J, Yang X, Shan X, et al. Discordance between antral follicle counts and anti-Müllerian hormone levels in women undergoing in vitro fertilization. Reproductive Biology and Endocrinology. 2019;17(1):51.
19. Abbara A, Eng PC, Phylactou M, Clarke SA, Hunjan T, Roberts R, et al. Anti-Müllerian hormone (AMH) in the Diagnosis of Menstrual Disturbance Due to Polycystic Ovarian Syndrome. 2019;10.
20. Kalaiselvi VS, P S, K P, Krishna GP. The anti mullerian hormone- a novel marker for assessing the ovarian reserve in women with regular menstrual cycles. Journal of clinical and diagnostic research : JCDR. 2012;6(10):1636-9.
21. Seifer DB, Baker VL, Leader B. Age-specific serum anti-Müllerian hormone values for 17,120 women presenting to fertility centers within the United States. Fertility and sterility. 2011;95(2):747-50.
22. La Marca A, Grisendi V, Griesinger G. How Much Does AMH Really Vary in Normal Women? International journal of endocrinology. 2013;2013:959487.
23. Shahrokh Tehraninezhad E, Mehrabi F, Taati R, Kalantar V, Aziminekoo E, Tarafdari A. Analysis of ovarian reserve markers (AMH, FSH, AFC) in different age strata in IVF/ICSI patients. International journal of reproductive biomedicine. 2016;14(8):501-6.
24. Qiu X, Gao Y, Guo T. Decomposition of Clinical Significance of FSH, LH, E2, AMH, and AFC Standards in Females at Lofty Elevation Based on HIF1α. Contrast media & molecular imaging. 2022;2022:6112659.
25. Köninger A, Koch L, Enekwe A, Birdir C, Kasimir-Bauer S, Kimmig R, et al. Change of anti-Mullerian-hormone levels during follicular phase in PCOS patients. Gynecological endocrinology : the official journal of the International Society of Gynecological Endocrinology. 2015;31(1):26-30.
26. Kollmann M, Obermayer-Pietsch B, Lerchbaum E, Lang U, Herzog SA, Trummer C, et al. Androgen and Anti-Mullerian Hormone Concentrations at Term in Newborns and Their Mothers with and without Polycystic Ovary Syndrome. 2019;8(11):1817.
27. Dewailly D, Barbotin A-L, Dumont A, Catteau-Jonard S, Robin G. Role of Anti-Müllerian Hormone in the Pathogenesis of Polycystic Ovary Syndrome. 2020;11.
28. Lv P-P, Jin M, Rao J-P, Chen J, Wang L-Q, Huang C-C, et al. Role of anti-Müllerian hormone and testosterone in follicular growth: a cross-sectional study. BMC Endocr Disord. 2020;20(1):101-.
29. Steffensen LL, Ernst EH, Amoushahi M, Ernst E, Lykke-Hartmann K. Transcripts Encoding the Androgen Receptor and IGF-Related Molecules Are Differently Expressed in Human Granulosa Cells From Primordial and Primary Follicles. 2018;6.
30. Devillers MM, Petit F, Cluzet V, François CM, Giton F, Garrel G, et al. FSH inhibits AMH to support ovarian estradiol synthesis in infantile mice. The Journal of endocrinology. 2019;240(2):215-28.
31. Rudnicka E, Kunicki M, Calik-Ksepka A, Suchta K, Duszewska A, Smolarczyk K, et al. Anti-Müllerian Hormone in Pathogenesis, Diagnostic and Treatment of PCOS. International journal of molecular sciences. 2021;22(22).
32. Bhide P, Dilgil M, Gudi A, Shah A, Akwaa C, Homburg R. Each small antral follicle in ovaries of women with polycystic ovary syndrome produces more antimüllerian hormone than its counterpart in a normal ovary: an observational cross-sectional study. Fertility and sterility. 2015;103(2):537-41.