Serum anti-Mullerian hormone levels in Turkish girls aged 18 and younger for ovarian reserve determination

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Abstract

Objective: Our aim was to show that anti-Mullerian hormone (AMH) may be used as a quantitative marker of ovarian reserve in Turkish girls aged 18 years and younger and establish the reference values for AMH in Turkish girls.

Material and Methods: This retrospective study included girls between 8-18 years old, without premature ovarian failure or without genetic factors resulting in ovarian dysgenesis. Blood specimens were collected after overnight fasting early in the morning during the early follicular phase. Measurement of serum levels of gonadotropins and AMH was done. Mean serum AMH levels of different age groups and best fitting curve representing AMH percentiles (10^{th}, 25^{th}, 50^{th}, 75^{th}, 90^{th}) were calculated.

Results: In total 785 girls with a mean age of 16.16±1.90 years were included, divided into seven age groups. The mean serum AMH level for the total cohort was 5.20±4.19 ng/mL. There was a significant difference between the mean values of AMH in age groups as follows: ≤12 and 17-≤18 (p=0.011). The best fitting curves for AMH percentiles were 4^{th} order polynomial functions. There was a significant correlation between AMH and age and follicle stimulating hormone levels (r=0.148, p<0.001 and r=-0.092, p=0.010).

Conclusion: Our results reflect the real-life data for serum AMH values in Turkish girls. Our nomogram may be useful for counseling adolescents about their ovarian reserve and diagnosing other gynecological diseases. A longitudinal study is necessary for improving the predictive value of AMH values in girls aged 18 and younger. (J Turk Ger Gynecol Assoc. 2024; 25: 138-43)

Keywords: Anti-Mullerian hormone, ovarian reserve, adolescent, nomograms

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Introduction

Anti-Mullerian hormone, (AMH), is a dimeric glycoprotein and a member of transforming growth factor-beta family, produced by the granulosa cells of primary, preantral and early antral follicles (1). It affects ovarian folliculogenesis by inhibiting the recruitment of primordial follicles. Moreover, it reduces the sensitivity of antral follicles to follicle-stimulating hormone (FSH) and inhibits FSH-stimulated estradiol production and aromatase expression (2). It has been assumed that after the 36^{th} week of gestation, the number of fetal primordial follicles decline with age until a critical level. However, the increased rate of primordial follicle recruitment overcomes the decline in total primordial follicle and serum AMH levels increase until the age of 14 years (3). This confirms that AMH levels are not regulated by gonadotropins but also that AMH level is not totally independent from gonadotropins (4).

The rate of follicle decline, in other words ovarian reserve, is influenced by age, genetic and environmental factors (1). Currently, basal FSH, antral follicle count (AFC) and AMH are frequently used in clinical practice for assessing ovarian reserve. FSH, due to the unknown variation pattern and AMH, due to its interindividual differences in random measurements may not give accurate results for patients during childhood and adolescence (5,6). AMH may serve better than AFC as an ovarian reserve marker in children and adolescents due to the difficulty in assessing AFC by transabdominal ultrasound in non-sexually active females. Although reference levels of AMH, are used for the evaluation of ovarian reserve in infertility

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patients; the physiological role and the clinical use of AMH in adolescence and childhood is less often discussed (7-9). Low or undetectable AMH levels are already used for the diagnosis of premature ovarian failure in pediatric patient for example for Turner syndrome and in girls on chemotherapy. However, low AMH levels due to partial gonadotropin dependency in hypogonadotropic hypogonadism patients are not reliable for the diagnosis of ovarian failure (10). In addition to that, in contrast to adult patients, the expected high levels of AMH in polycystic ovary syndrome (PCOS) are not necessarily observed in adolescents with PCOS (11-13). Thus, the aim of this study was to show that AMH may be used for assessing ovarian reserve and to establish reference values for AMH in Turkish girls.

Material and Methods

Study design and patient selection

This retrospective study was performed in the gynecology outpatient clinic of a faculty, from 2010 to 2020. AMH levels were obtained from all girls aged between 8-18 years who attended our clinic with a complaint unrelated to ovarian function, such as abdominal pain, suprapubic pain, or heavy menstruation. The data was extracted from the electronic records. Patients diagnosed with premature ovarian failure or with other genetic factors resulting in ovarian dysgenesis were excluded. Patients who had surgery that may result in a decrease in ovarian function, such as ovarian cystectomy and who were using oral contraception during the examination were excluded.

All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Approval was granted by the İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine Clinical Research Ethics Committee (approval number: 52825153-604.01.01-134000, date: 12.10.2020).

Hormone assays

Blood specimens were collected after overnight fasting early in the morning from the antecubital vein during the early follicular phase (3^{rd}-5^{th} day of the cycle), if after menarche. Measurement of serum levels of gonadotropins, lutenizing hormone (LH) and FSH, was done at the biochemistry laboratory of our faculty.

Serum levels of AMH were measured using the DSL-10-14400 Active Mullerian Inhibiting Substance/AMH ELISA kit (Diagnostic Systems Laboratories, Webster, TX, USA) before 2015 and the Elecsys AMH Plus test on the Cobas-E electrochemiluminescence immunoassay platform (Roche Diagnostics GmbH, Mannheim, Germany) after 2015. Serum AMH values are reported as ng/mL and the assay range was 0.01-23 ng/mL. The intra- and inter-assay variation coefficients were <8% and <12%, respectively. Basal fasting FSH, LH and E_2 were measured by electrochemiluminescence method (Cobas 8000 systems, Roche Diagnostics GmbH, Mannheim, Germany).

Statistical analysis

Statistical analysis was performed by SPSS version 21 for Mac (IBM Inc., Armonk, NY, USA). Demographic characteristics are reported with descriptive statistics. Continuous variables are expressed as mean ± standard deviation. Statistical comparisons of the means of more than two groups were carried out according to ANOVA, where appropriate. A $p<0.05$ was accepted to be statistically significant. The 10^{th}, 25^{th}, 50^{th}, 75^{th} and 90^{th} percentiles of AMH by age were calculated and graphed according to the best fitting curve in Microsoft Excel, version 16.53 for Mac 21. A fourth order polynomial function was chosen since it was the best fitting curve for AMH percentiles with highest R^2 values. The correlation between age, FSH and AMH was calculated by Pearson's correlation coefficient.

Data availability statement

The data associated with the paper are not publicly available but are available from the corresponding author on reasonable request.

Results

In total 785 Turkish girls with mean age of 16.16±1.90 years were included. The girls were divided into seven groups by age: 8-12; 12.01-13; 13.01-14; 14.01-15; 15.01-16; 16.01-17; and 17.01-18. The hormonal characteristics of our cohort are presented in Table 1 for all participants. The mean serum AMH level for the whole cohort was 5.20±4.19 ng/mL. The mean serum FSH and LH levels for the whole cohort were 5.30±1.94 IU/mL and 6.81±4.82 IU/mL, respectively. The percentiles of serum AMH levels are presented at Table 2. There was a significant difference between the mean values of AMH in age groups when comparing the ≤12 group with the 17-≤18 group ($p=0.011$). There was no significant difference between the other age groups.

A fourth order polynomial function was the best fitting curve for AMH percentiles in our cohort. R^2 values of the curves were listed below.

10^{th} percentile: $y=0.0114x^4-0.6827x^3+15.166x^2-147.91x+534.8$ ($R^2=0.9566$)

25^{th} percentile: $y=0.0106x^4-0.6483x^3+14.784x^2-148.25x+552.6$ ($R^2=0.9408$)

50^{th} percentile: $y=0.0119x^4-0.7197x^3+16.186x^2-159.91x+587.7$ ($R^2=0.9357$)

75^{th} percentile: $y=0.0568x^4-3.4249x^3+76.835x^2-759.08x+2789.5$ ($R^2=0.9383$)

90^{th} percentile: $y=0.067x^4-3.9546x^3+86.668x^2-834.81x+2987.1$ ($R^2=0.9139$)

The nomogram of serum AMH levels is shown in Figure 1. There was a statistically significant but weak correlation between AMH and age and AMH and FSH level (r=0.148, p<0.001 and r=-0.092, p=0.010) (Table 3).

Discussion

This study suggested that AMH may be a reliable ovarian reserve marker for children and adolescents. We also established reference values for serum AMH levels for a population of Turkish girls.

Table 1. Hormonal characteristics of the cohort

Age, years (n)	FSH (IU/mL) (mean ± SD)	LH (IU/mL) (mean ± SD)	AMH (ng/mL) (mean ± SD)	AMH (ng/mL) (median)
8-12 (36)	4.50±2.40	4.56±4.74	3.22±3.74	2.42
12.01-13 (27)	5.60±1.95	6.00±4.42	3.58±4.34	2.61
13.01-14 (69)	5.35±1.78	6.28±5.32	4.15±3.11	3.20
14.01-15 (117)	5.45±1.94	7.37±5.21	5.57±4.62	4.07
15.01-16 (126)	5.26±1.98	6.78±4.79	4.87±3.81	3.69
16.01-17 (169)	5.31±2.04	6.75±4.38	5.47±3.99	4.35
17.01-18 (241)	5.32±1.78	7.19±4.77	5.78±4.43	4.43
Total (785)	5.30±1.94	6.81±4.82	5.20±4.19	4.04

FSH: Follicle-stimulating hormone, LH: Luteinizing hormone, AMH: Anti-Mullerian hormone, SD: Standard deviation

Table 2. Percentile values of serum AMH in Turkish girls

Age, years (n)	5th percentile	10th percentile	25th percentile	50th percentile	75th percentile	90th percentile	95th percentile
8-12 (36)	0.36	0.52	1.47	2.42	4.19	5.44	8.89
12.01-13 (27)	0.37	0.66	1.27	2.61	3.78	7.03	17.16
13.01-14 (69)	1.01	1.44	2.13	3.20	5.74	8.84	10.98
14.01-15 (117)	0.98	1.49	2.31	4.07	7.28	11.68	17.15
15.01-16 (126)	0.86	1.59	2.44	3.69	6.39	9.33	11.43
16.01-17 (169)	1.14	1.52	2.79	4.35	7.04	10.37	14.18
17.01-18 (241)	0.93	1.58	2.79	4.43	7.45	11.64	16.92

AMH: Anti-Mullerian hormone

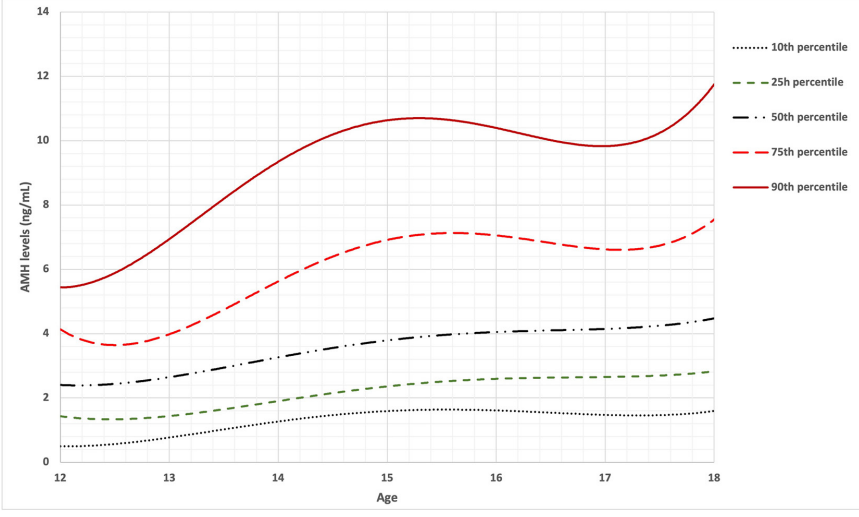

Figure 1. AMH nomogram in Turkish girls based on 4th degree polynomial function. Reference lines of serum AMH for the 10th, 25th, 50th, 75th, 90th percentiles vs. age were demonstrated
AMH: Anti-Mullerian hormone

Table 3. Correlation of AMH with age and FSH

	AMH	
	r	p
Age	0.148	<0.001
FSH	-0.092	0.010

FSH: Follicle-stimulating hormone, AMH: Anti-Mullerian hormone

AMH levels in adolescence have an increasing pattern and peak before pubertal onset. Even though the mean age of menarche in Turkish girls was reported as between 12-13 years (14), we didn't observe a peak around the age groups of ≤12 and 12-≤13. Unlike other studies, we have found a significant but weak correlation of AMH with age and FSH (6,8). Thus, we believe that our nomogram will be a guide for interpreting random AMH measurements during consultation of girls in pediatrics and gynecology clinics.

In the postnatal period, FSH surge increases AMH levels by stimulating ovarian folliculogenesis and follicular growth (6,15,16). Due to ongoing recruitment and follicle growth initiation in a very large pool, a steady increase is observed in serum AMH during childhood, independent of gonadotropins (17). Hagen et al. (3) showed that AMH levels increase significantly by 17% before pubertal onset due to preceding follicular recruitment (18). After pubertal onset due to the growth of follicles beyond the stage of AMH production (redistribution of follicle pool), AMH levels decrease by 30% (3,19). Central inhibition of gonadotropin secretion in childhood and FSH surge before puberty complicate the diagnosis of ovarian failure. Therefore, AMH may serve better than FSH as an ovarian reserve marker in adolescence. Our results confirm the earlier studies that demonstrated minor fluctuations in AMH during childhood and adolescence. Stable AMH levels are achieved by the increased recruitment versus extensive follicle loss (19). Our results partially show the prepubertal increase in AMH. Yet, the increasing rate of recruitment in follicles up to 14-15 years of age was shown clearly by the increase in AMH levels, followed by a plateau, as has been described previously (8,17,19-21). AMH levels >3 ng/mL show more variability across the menstrual cycle compared to low levels (22), so the variability observed in higher percentiles in our results may be attributed to this observation. These interindividual differences don't affect the clinical management since lower AMH levels are more useful for the diagnosis of premature ovarian failure (9,10). Furthermore, wide variation of AMH levels in healthy adolescents may be due to the different rates of follicle loss between individuals (6). Therefore, the diagnostic power of high AMH levels for pubertal girls is yet to be determined.

The clinical use of AMH levels in the pediatric age group includes during the follow-up of patients with central precocious puberty and patients with cancer under gonadotoxic chemotherapy or galactosemia, the determination of the presence of testicular tissue and the diagnosis of persistent Mullerian Duct Syndrome, primary ovarian failure and hypogonadotropic hypogonadism, Klinefelter Syndrome and granulosa cell tumors (10,23,24). There are various studies investigating AMH levels in adolescent patients with PCOS but reporting conflicting results. Therefore, AMH was assumed to have poor diagnostic potency for adolescents with PCOS according to the current literature (12,13,25-27). It should be kept in mind that multifollicular ovarian morphology seen on ultrasound during mid-late puberty may not necessarily represent PCOS. That is indeed a physiological change of puberty.

Study limitations

In this study, we lacked information about the patient's menstrual cycle history, age of menarche, body mass index (BMI), clinical hyperandrogenism and ovarian volume. Thus, we cannot comment on the prevalence of PCOS in our cohort. There are conflicting results about the impact of BMI on AMH values in the literature. Some authors suggested that BMI does not affect AMH values in women of reproductive age, with or without PCOS (28) while others have reported a positive correlation between AMH and BMI (29). Moreover, another study stated that the cut off value of AMH for diagnosing PCOS decreased gradually with increasing BMI (30,31). The lack of BMI data for our subjects prevented us from commenting on the impact of BMI on AMH values in Turkish girls. We also found a higher mean for serum AMH levels in adolescence compared to the other studies (17). This may be due to the high prevalence of PCOS in Turkish population (26%, as reported in Global Burden of Disease Study in 2016) between ages of 15-49 years (32). In an earlier study, it was shown that a quadratic equation was the best model to describe declining serum AMH levels with age (33). The quadratic model didn't fit our data since the age of our cohort was different. Our data is unique in the way that it includes the largest number of girls aged 18 and younger reported until now.

Conclusion

Predicting the menopausal age by AMH levels during childhood and adolescence is not reliable because of the different rate of follicle loss between individuals. However, our results provide real-life data for understanding and interpreting AMH values during childhood and adolescence in Turkish girls. Our nomogram may be useful for counselling girls about their ovarian reserve and diagnosing other gynecological diseases. A longitudinal study is necessary for improving the predictive value of AMH values in girls aged 18 and younger.

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Informed Consent: *Retrospective study.*

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