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Anti-Müllerian Hormone and Its Clinical Utility in the Pediatric and Adolescent Population

Rubén Tlapanco Vargas[1*], Guisela María Recinos Tobar[2], Jonathan RT Lakey[3,4] and Pedro Gutierrez Castrellon[4]

[1]Specialist in Human Reproductive Biology, Instituto Nacional de Perinatología, Mexico City, Mexico
[2]Specialist in Human Reproductive Biology, Guatemala City, Guatemala
[3]Departments of Surgery and Biomedical Engineering, University of California, Irvine, CA, USA
[4]Elemental Translation Research, SAPI, Mexico City, Mexico

***Corresponding author:** Rubén Tlapanco Vargas, Instituto Nacional de Perinatología, C. Montes Urales 800, Lomas-Virreyes, Lomas de Chapultepec IV Secc, Miguel Hidalgo, 11000 Ciudad de México.

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Abstract

Anti-Müllerian hormone (AMH) is a dimeric glycoprotein of the transforming growth factor-β family, secreted by the granulosa cells of preantral and small antral follicles, whose serum concentration reflects the growing follicle pool and, indirectly, the resting primordial follicle pool. This narrative review summarizes the clinical utility of AMH in the pediatric and adolescent population. In polycystic ovary syndrome (PCOS)—the most common endocrinopathy of reproductive-age women—serum AMH is two to four times higher than in healthy women and correlates with disease severity, hyperandrogenism, insulin resistance, and impaired folliculogenesis. Because the diagnosis of PCOS in adolescents is hampered by the physiological overlap of menstrual irregularity, the limited applicability of the modified Ferriman–Gallwey scale, the absence of validated biochemical cut-offs, and the risk of overdiagnosis when polycystic ovarian morphology is assessed by ultrasound, AMH has emerged as a more objective marker. Its stability across the menstrual cycle and its minimal fluctuation during adolescence facilitate the establishment of cut-off values, and elevated adolescent AMH has been associated with an increased risk of PCOS in adulthood. Although AMH cannot be used in isolation, it is a valuable component of the diagnostic algorithm and may potentially replace ultrasound in this age group. Beyond PCOS, AMH is a useful marker of gonadal function in disorders of sex development, of ovarian reserve in Turner syndrome, and of gonadotoxic damage in childhood and adolescent cancer survivors, in whom serial measurement can identify candidates for fertility preservation.

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Incorporating AMH measurement into the clinical evaluation of adolescents with endocrine dysfunction, an oncological history, or reproductive risk factors represents a step toward more preventive, precise, and personalized medicine.

Keywords

Anti-Müllerian hormone; Polycystic ovary syndrome; Adolescents; Pediatrics; Ovarian reserve; Hyperandrogenism; Turner syndrome; Fertility preservation; Biomarker.

Abbreviations

AMH: Anti-Müllerian Hormone; PCOS: Polycystic Ovary Syndrome; TGF-β: Transforming Growth Factor-β; FSH: Follicle-Stimulating Hormone; AFC: Antral Follicle Count; FAI: Free Androgen Index; SHBG: Sex Hormone-Binding Globulin; DHEA-S: Dehydroepiandrosterone Sulfate; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; IR: Insulin Resistance; T2DM: Type 2 Diabetes Mellitus; DSD: Disorders of Sex Development; MRI: Magnetic Resonance Imaging; AACE: American Association of Clinical Endocrinology.

Introduction

Anti-Müllerian hormone (AMH) is a dimeric glycoprotein belonging to the transforming growth factor-β (TGF-β) family of proteins; it is secreted predominantly by the granulosa cells of preantral and small antral follicles. In adult women, serum AMH concentrations reflect the size of the pool of growing ovarian follicles and, indirectly, the pool of resting primordial follicles. Its physiological functions include inhibiting the recruitment of primordial follicles, exerting an inhibitory effect on gonadotropin-dependent follicular development, and suppressing aromatase activity. These actions may explain why elevated AMH levels could be implicated in the ovulatory dysfunction and hyperandrogenism that characterize women with polycystic ovary syndrome (PCOS) [1-4].

Utility in Polycystic Ovary Syndrome

Diagnosis and the rotterdam criteria

PCOS is the most common endocrinopathy among women of reproductive age, affecting 5–15% of them. It is a heterogeneous condition that may present with reproductive, endocrine, and metabolic alterations, which can vary throughout life [5]. The Rotterdam criteria, introduced in 2003 and subsequently updated in 2023, require the presence of at least two of three criteria to establish a diagnosis of PCOS: clinical or biochemical hyperandrogenism, oligo-anovulation, and polycystic ovarian morphology, after exclusion of other etiologies [1].

Clinical and biochemical hyperandrogenism

Clinical hyperandrogenism may present as hirsutism, which is assessed using the modified Ferriman–Gallwey visual scale, with the diagnostic threshold depending on ethnicity. Other possible manifestations include female-pattern alopecia and acne; however, these are poor predictors of biochemical hyperandrogenism in the absence of hirsutism. In adolescents with acne, hyperandrogenism should be considered only in cases of severe acne [6].

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Assessment of biochemical hyperandrogenism should include measurement of total and free testosterone; when these are normal, measurement of androstenedione and dehydroepiandrosterone sulfate (DHEA-S) may be considered. The free androgen index (FAI), calculated by dividing total testosterone (nmol/L) by sex hormone–binding globulin (SHBG, nmol/L), also allows the diagnosis of biochemical hyperandrogenism, with values >4.5% reported in the Mexican population [7,8]. With regard to laboratory technique, tandem mass spectrometry is recommended for measuring total testosterone, androstenedione, and DHEA-S, whereas free testosterone can be assessed using a calculated formula, equilibrium dialysis, or ammonium sulfate precipitation [9]. When androgen levels are markedly above the laboratory reference ranges, causes of hyperandrogenemia other than PCOS must be ruled out [6].

Ovulatory dysfunction

Irregular cycles are defined as those lasting >90 days during the first-year post-menarche; <21 or >45 days during the second-year post-menarche; or <21 days, >35 days, or fewer than eight cycles per year from the third-year post-menarche onward. Primary amenorrhea by age 15 years, or more than three years after thelarche, is also considered a diagnostic criterion. Ovulatory dysfunction may occur even in patients with regular cycles, so measurement of serum progesterone may be necessary to confirm ovulation [6].

Polycystic ovarian morphology

For the diagnosis of polycystic ovarian morphology, a follicle number per ovary ≥20 in at least one ovary is considered the most effective ultrasonographic marker. An ovarian volume ≥10 mL, or a follicle number per cross-section ≥10 in at least one ovary, may be used as a diagnostic criterion when older technology or insufficient image quality is used. Importantly, there are no definitive diagnostic criteria for polycystic ovarian morphology in adolescents, and it is therefore recommended that this criterion not be included in the diagnosis of PCOS in this population [6]. Adolescents with features of PCOS who do not meet the full diagnostic criteria should be considered at increased risk, and reassessment is suggested upon reaching reproductive maturity, at eight years post-menarche [6].

AMH as a marker of severity and metabolic risk

AMH levels are two to four times higher in women with PCOS than in healthy women, and this elevation has been described across all PCOS populations. The increase is secondary both to the greater number of preantral and small antral follicles and to the increased AMH secretion by each follicle. Because of the close relationship between circulating AMH levels and the antral follicle count (AFC) on ultrasound, AMH has been proposed as an alternative marker of ovulatory dysfunction in PCOS, and even as a stand-alone diagnostic test for PCOS [10].

Serum AMH concentrations correlate directly with the severity of PCOS symptoms; levels are significantly higher in the presence of hyperandrogenism, leading to the proposal that elevated AMH could serve as a marker of hyperandrogenism [10]. Insulin resistance (IR) is another condition associated with PCOS; some studies have described a significant positive correlation between AMH and the homeostatic model assessment of insulin resistance (HOMA-IR) and propose that AMH levels could be used as a prognostic factor for metformin therapy, reporting a decrease in AMH values after treatment [11-13].

AMH plays a role in folliculogenesis by regulating the recruitment of primordial follicles and gonadotropin-dependent follicular development; therefore, elevated AMH concentrations may be related to the impaired folliculogenesis observed in patients with PCOS [10]. In 2010, Hart et al. demonstrated a positive

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correlation between AMH values and elevated levels of advanced glycation end products, which are linked to the impaired folliculogenesis present in PCOS [14]. They further confirmed that AMH levels are a better predictor of women with anovulation and hyperandrogenemia without polycystic ovarian morphology, which would allow identification of adolescents with a more adverse metabolic profile and a higher risk of cardiovascular disease and type 2 diabetes mellitus (T2DM) than other phenotypes [14].

Intra- and inter-cycle Variability

AMH concentrations remain essentially constant throughout the menstrual cycle and are reproducible from one cycle to another, a property that reinforces its usefulness as an objective marker independent of cycle timing [15]. Some investigators have studied the relationship among AMH concentrations, body weight, and menstrual cycles in patients with overweight and PCOS, describing a significant decrease in serum AMH levels after lifestyle modification through a balanced diet and regular physical activity [10].

Diagnostic challenges in adolescents and the value of AMH

Early follow-up allows the early detection of metabolic alterations and endometrial atypia, which in turn enables early interventions that reduce the future risks of metabolic syndrome and endometrial carcinoma; more objective diagnostic criteria for adolescents are therefore needed [16]. The advantages of having a biochemical marker for PCOS are especially relevant in the adolescent population, in whom transvaginal ultrasound may not be appropriate [14]. A key limitation of including the ultrasonographic criterion is the risk of overdiagnosis of PCOS in adolescents, given the high incidence of multifollicular ovaries during the first eight years post-menarche and the fact that this is a largely sexually inactive population [9].

In such cases, transabdominal ultrasonography or magnetic resonance imaging (MRI) could be considered. However, visualization of the ovaries and antral follicle counting may be difficult by the transabdominal route. MRI may display follicles more clearly; nevertheless, the reported follicle number depends on slice thickness and may be inaccurate because of artifacts. One of the greatest disadvantages of MRI is its high cost compared with ultrasound [16].

For these reasons, it has been recommended that the diagnosis in adolescents be based solely on the criteria of hyperandrogenism and oligo-anovulation; however, this can pose a considerable challenge for the clinician. The Ferriman–Gallwey scale used to assess hirsutism was standardized in White women, most of them older than 24 years, and may therefore not be a useful tool for the adolescent population; moreover, some adolescents may experience transient hirsutism, which could lead to a misdiagnosis of PCOS [17]. In addition, cut-off values to define biochemical hyperandrogenism have not been established in this population [14]. Furthermore, menstrual irregularity and ovulatory dysfunction are a normal, physiological component of the first years after menarche [9].

For all these reasons, measurement of AMH levels could be considered a more objective and highly valuable diagnostic method for the diagnosis of PCOS in the adolescent population [16]. AMH levels decline gradually from the age of 25 years, which makes it difficult to establish cut-off values; however, in adolescent women the fluctuation is minimal, allowing cut-off values to be established more readily [16,18,19].

Adolescent AMH as a predictor of future PCOS

In 2023, Hagen et al. published the results of a longitudinal cohort of 695 healthy Danish girls born between 1997 and 2010, who were assessed at different time points from infancy to adolescence (infancy, mean age 0.3 years; mid-childhood, 7.2 years; puberty, 11.3 years; and adolescence, 15.9 years) to determine whether serum AMH concentrations in infancy and mid-childhood correlate with AMH concentrations and ovarian morphology during puberty and adolescence. They reported that serum AMH concentrations during infancy, mid-childhood, and puberty were strongly correlated with AMH concentrations during adolescence, as well as with the ovarian follicle count; high AMH levels in infancy remained elevated into adolescence, and, likewise, low AMH levels in infancy remained low into adolescence. These findings suggest highly stable ovarian activity in healthy women and support the theory that AMH levels in early life are a useful clinical tool for predicting future ovarian activity [20].

In 2021, Caanen et al. reported that elevated AMH levels during adolescence are a risk factor for the development of PCOS in adulthood; they compared adolescent AMH concentrations in adult women with and without PCOS and found that those with PCOS in adulthood had had higher AMH concentrations during adolescence [21]. Moreover, when the adolescent AMH cut-off was set at 6 ng/mL, the sensitivity and specificity for PCOS in adulthood were 50% and 87%, respectively [21]. In this regard, measurement of AMH in adolescence is highly useful not only for the diagnosis of PCOS but also for determining future risk.

Given the difficulties associated with diagnosing PCOS in the adolescent population, AMH measurement represents a valuable alternative that could potentially replace the use of ultrasound in this age group, as recommended by the American Association of Clinical Endocrinology (AACE); and although AMH values cannot be used independently, they may be highly useful as part of the diagnostic algorithm together with the other established diagnostic criteria [10,22].

Other Uses of AMH in the Pediatric Population

Sexual differentiation and gonadal function

AMH is secreted in both females and males, by the ovaries and testes, respectively, and plays a crucial role in prenatal sexual differentiation by inhibiting the development of the Müllerian (paramesonephric) ducts in males [10]. In males, AMH is secreted by immature Sertoli cells from the eighth week of gestation and persists until approximately two years of postnatal life, at which point it begins to decline gradually until puberty, becoming undetectable in adults. In females, AMH secretion begins at week 36 of gestation, reaches a neonatal peak, and then remains low until puberty, after which levels begin to rise until reaching a plateau in adolescence; the decline begins in the middle of the second decade of life, and AMH becomes undetectable years before menopause [10].

Serum AMH levels reflect the presence and function of testicular tissue in prepubertal boys without the need for a stimulation test, whereas measurement of gonadotropin and testosterone concentrations—which remain very low until the onset of puberty—lacks clinical utility as a marker of hypothalamic–pituitary–testicular axis function. AMH determination is therefore extremely useful, since Sertoli cells remain active throughout childhood [23].

Disorders of sex development

The simultaneous assessment of AMH and testosterone—markers of Sertoli and Leydig cell function, respectively—provides a simple and useful tool for the pediatric endocrinologist. In patients with

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disorders of sex development, when both AMH and testosterone levels are below the normal male reference range, gonadal dysgenesis should be suspected; cases in which AMH is within the normal reference range but testosterone is low are indicative of specific Leydig cell disorders; and when both hormones are within or above the normal reference ranges, the picture probably corresponds to a defect in androgen sensitivity. Low AMH values are indicative of primary or central hypogonadism, whereas undetectable values suggest anorchia [23].

Turner syndrome

In patients with Turner syndrome, AMH assessment plays a significant role in estimating ovarian function, since this chromosomal alteration causes accelerated depletion of the ovarian reserve. Girls with concentrations above the detection level are more likely to experience spontaneous thelarche and menarche and to retain some ovarian function during their reproductive life. Assessing ovarian reserve through AMH in these patients allows clinicians to discuss with the patients and their guardians the likelihood of spontaneous pregnancy, its risks, and the currently available options for fertility preservation, such as vitrification of oocytes or ovarian tissue in patients with residual ovarian function [20,24].

Fertility preservation in childhood cancer survivors

Measurement of AMH concentrations can also be a useful tool in the pediatric and adolescent population of cancer survivors. As pediatric cancer survival rates increase, so does the risk of ovarian dysfunction associated with heavy-metal chemotherapy, which may manifest either as a low ovarian reserve with preserved ovarian function or, conversely, as more extensive damage with premature ovarian insufficiency, early menopause, and infertility. In 2023, Pruett et al. reported that up to one-fifth of female cancer survivors—diagnosed between 11 and 21 years of age and treated with cisplatin—had AMH values indicative of low ovarian reserve on measurements taken at least one year after completing therapy. They also showed that some patients with initially normal AMH values had levels fall below the normal range on subsequent measurements, underscoring the need for serial AMH measurement. Adolescents and young adult women with low AMH concentrations but preserved ovarian function may have a window of opportunity to undergo ovarian stimulation for oocyte cryopreservation, if this was not performed prior to cancer treatment [25].

Conclusion

Measurement of anti-Müllerian hormone (AMH) in the adolescent population represents a diagnostic and prognostic tool of high clinical value that should be considered and implemented routinely in pediatric and gynecological practice. The complexity and ambiguity of the current diagnostic criteria for PCOS in adolescents, together with the limitations of ultrasound and the variability of the clinical manifestations characteristic of this stage, make objective and reliable markers indispensable.

AMH levels have shown a significant correlation with ovulatory dysfunction, hyperandrogenism, and PCOS severity, and also act as an early indicator of future metabolic and reproductive risk. Their measurement at early ages would allow not only a more accurate and timelier diagnosis of PCOS but also the identification of adolescents at increased risk of developing long-term metabolic and reproductive complications, enabling early and personalized interventions.

Moreover, AMH has applications beyond PCOS, serving as a useful marker for evaluating gonadal function

in disorders of sex development, for monitoring oncology patients who survive gonadotoxic treatments, and for estimating ovarian reserve without the need for invasive procedures. Its stability during adolescence and its independence from the menstrual cycle make AMH an ideal biomarker for this population.

In this context, promoting the incorporation of AMH measurement into the clinical approach to adolescents with signs of endocrine dysfunction, an oncological history, or reproductive risk factors represents a crucial step toward more preventive, precise, and personalized medicine. Its implementation would not only enrich the diagnostic algorithm for PCOS in adolescents but would also open new opportunities to preserve reproductive health from the early stages of life.

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None.

Conflict of interest

The authors declare that they have no conflict of interest.

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