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Polycystic Ovary Syndrome in Light of Hormonal Panel Interpretations Literature Review

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Abstract
Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder defined by a triad of ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology (PCOM).
The "hormonal panel" for PCOS includes ovarian and adrenal androgens, gonadotropins, sex hormone–binding globulin (SHBG), anti–Müllerian hormone (AMH), and tests that rule out other conditions (like TSH, prolactin, and 17-hydroxyprogesterone).
In the last ten years, evidence-based guidelines have improved diagnostic and management pathways and have thought about how to interpret certain analytes, especially AMH.
This review consolidates contemporary evidence regarding the diagnostic, phenotyping, and prognostic functions of the hormonal panel in PCOS, emphasizing the advantages and drawbacks of essential biomarkers and presenting practical testing algorithms derived from the 2023 International Evidence-based PCOS Guideline and recent systematic reviews.

Introduction

PCOS impacts approximately 8–13% of women of reproductive age, contingent upon diagnostic criteria and the studied population, imposing reproductive, metabolic, and psychological challenges throughout their lifespan.

The pathophysiology integrates hyperandrogenism, insulin resistance (IR), and modified hypothalamic-pituitary-ovarian signaling, alongside genetic and epigenetic modifiers. Hormonal testing is essential for diagnosis, phenotyping, the exclusion of mimics, and ongoing care.

The Rotterdam consensus (2003) expanded the definition of PCOS to include any two of [ovulatory dysfunction, hyperandrogenism, PCOM]. Subsequent revisions have focused on ultrasound thresholds, adolescent diagnosis, and the potential for AMH to replace PCOM.

This Review

1. Delineates contemporary diagnostic frameworks;
2. Assesses the efficacy and limitations of principal analytes—AMH, androgen metrics, LH/FSH ratio, SHBG, prolactin, TSH, and 17-hydroxyprogesterone; and
3. Offers a pragmatic interpretative strategy for various clinical scenarios (adult versus adolescent, fertility versus metabolic emphasis).

Ways to Find Studies and Choose Them:

We conducted a narrative search of PubMed and Scopus until August 12, 2025, utilizing combinations of: polycystic ovary syndrome OR PCOS AND (androgen OR testosterone OR DHEAS OR androstenedione OR anti-Müllerian hormone OR AMH OR LH OR FSH OR LH/FSH ratio OR SHBG OR insulin resistance OR prolactin OR TSH OR 17-hydroxyprogesterone), applying filters for English language and human studies.

We gave priority to guidelines, systematic reviews, meta-analyses, and large cohort studies.

We looked through the reference lists of important articles by hand. We left out material that hadn't been peer-reviewed, populations that didn't fit the standard definitions of PCOS, and abstracts that didn't have full text.

Diagnostic Frameworks and the Role of the Hormonal Panel, from the NIH (1990) to Rotterdam (2003) to the 2023 Guideline

The NIH 1990 definition focused on hyperandrogenism and ovulatory dysfunction, having ruled out other related conditions. Rotterdam (2003) broadened the classification to incorporate PCOM as one of three criteria, necessitating the presence of any two for diagnosis. This inclusion created phenotypes characterized by PCOM without hyperandrogenism, or by hyperandrogenism alongside PCOM, despite regular ovulatory patterns. The 2023 International Guideline made ultrasound thresholds clearer and said that high AMH could be used instead of PCOM in adults (with caution in teens).

These changes have a direct effect on which hormone tests are given priority and how they are read.

Teenagers

The guideline advises that, in adolescents, PCOS should be diagnosed solely in the presence of both persistent ovulatory dysfunction and clinical or biochemical hyperandrogenism, eschewing dependence on PCOM or AMH due to developmental variability. This has immediate implications for the hormonal panel: in teenagers, the focus is on reliable markers of hyperandrogenism and on exclusionary tests; AMH is not yet validated as a stand-alone diagnostic criterion.

What to Order and How to Read Core Androgen Measures

Total and Free Testosterone, A4, and DHEAS:

Biochemical hyperandrogenism can be diagnosed through elevated levels of total testosterone (TT), calculated free testosterone (cFT), free androgen index (FAI), androstenedione (A4), or, less consistently, dehydroepiandrosterone sulfate (DHEAS). High-quality evidence indicates that TT and cFT/FAI are the most valuable diagnostic analytes, while A4 is frequently informative and DHEAS is elevated only in a specific subset, indicating adrenal contribution. The quality of the assay (for example, LC-MS/MS for TT) and the time of day or phase are important.

- **Diagnostic accuracy:** A 2024 systematic review aimed at guiding the International Guideline determined that TT, cFT, FAI, and, in certain studies, A4 exhibit superior diagnostic efficacy for biochemical hyperandrogenism, whereas DHEAS and DHT demonstrate less reliability.
- **Clinical nuance:** A significant increase in DHEAS levels necessitates investigation for adrenal tumors or non-classic congenital adrenal hyperplasia (CAH), highlighting the function of 17-hydroxyprogesterone as a diagnostic exclusion test.

SHBG and FAI

Low SHBG, which is common in people with IR, obesity, and hyperinsulinemia, raises free androgen activity and FAI. SHBG is not a standalone diagnostic test; rather, it contextualizes androgen bioavailability and cardiometabolic risk. Recent data indicate that SHBG fluctuates with insulin sensitivity phenotypes; therefore, integrating SHBG with TT (to compute FAI) facilitates interpretation.

Gonadotropins: LH, FSH, and the LH/FSH Ratio

The long-taught LH/FSH ratio >2:1 does not have the sensitivity and specificity that was once thought because pulsatile secretion and differences between

tests make it hard to understand. Some populations exhibit elevated LH levels, resulting in a higher ratio; however, this ratio should not be utilized as the sole diagnostic criterion. It can, however, enhance a pattern of hyperandrogenism and anovulation.

AMH: From a marker of ovarian reserve to a signal for PCOS

Biology and Increase in PCOS

Granulosa cells in pre-antral and small antral follicles release AMH. On average, women with PCOS have serum AMH levels that are about two to three times higher than normal. This is because they have more small follicles and their granulosa cells are not working properly.

Mechanistic studies associate AMH with modified follicle selection and potentially with GnRH neuronal activity, corroborating its involvement in the anovulatory phenotype.

Diagnostic Applications and Thresholds

There is increasing evidence—consolidated for the 2023 Guideline—that AMH can aid in diagnosis in adults and may replace PCOM where high-quality assays and validated thresholds are available. Meta-analytic estimates have suggested thresholds around ~4.7 ng/mL for adult diagnosis; however, these values are assay- and age-dependent, and cut-offs differ among populations. Adolescents should not be diagnosed solely based on AMH levels.

Implications for Prognosis and Treatment

AMH is linked to ovulatory status and could be used to track how well certain treatments work. Initial evidence indicates that metabolic therapies (e.g., metformin in IR-predominant phenotypes) may influence AMH levels; however, this is not yet a standard monitoring objective, as clinical outcomes remain the primary focus.

The Panel, Metabolic Hormones, and Insulin Resistance

IR is prevalent among PCOS phenotypes and engages in bidirectional interactions with hyperandrogenism. Insulin resistance lowers SHBG in the liver, which raises free androgens. High levels of insulin also increase theca cell steroidogenesis and can change the way gonadotropins work.

Fasting insulin/HOMA-IR are not diagnostic for PCOS; however, they stratify metabolic risk and contextualize low SHBG and elevated FAI. The clinician should include the OGTT or HbA1c, fasting lipid profile, and weight/waist measurement in the initial care.

Tests that leave people out and common mimics:

The hormonal panel in PCOS must exclude alternative etiologies of anovulation and hyperandrogenism.

- TSH: Primary thyroid dysfunction may resemble menstrual irregularity.
- Prolactin: Hyperprolactinemia may lead to oligoamenorrhea; significant elevations indicate pituitary pathology.
- 17-hydroxyprogesterone: High morning follicular-phase values (>200 ng/dL; assay-specific) could be a sign of non-classic CAH.
- Cushing's/androgen-secreting tumors: Clinical indicators of concern or significantly elevated androgen levels necessitate focused testing and imaging.

Modern guideline algorithms stress these to keep PCOS from being overdiagnosed.

Phenotyping and Life Stage Considerations:

Adult Phenotypes (Rotterdam A–D):

Biochemical profiles differ among phenotypes. Classic hyperandrogenic, anovulatory phenotypes (A/B) usually have higher TT/FAI and sometimes higher LH than "non-hyperandrogenic" phenotypes (C/D). AMH is typically elevated across phenotypes, exhibiting the strongest correlation with anovulation severity.

Teenagers

Because physiologic anovulation and acne/hirsutism can happen at the same time as normal puberty, it is important to rule out other causes before diagnosing an adolescent with both persistent ovulatory dysfunction and hyperandrogenism. PCOM and AMH are not recommended as first-line tests at this age.

Effects of Ethnicity and BMI

Androgen and SHBG levels differ based on BMI and ethnicity; obesity decreases SHBG and increases FAI regardless of absolute TT, potentially revealing

hyperandrogenism. This emphasizes the necessity of integrating biochemical tests with clinical evaluation and metabolic profiling.

Putting It All Together: A Useful Hormonal Testing Algorithm

1. Verify the clinical context (adult versus adolescent; fertility versus metabolic focus).
2. For adults, the first-line labs are TT (preferably LC-MS/MS), SHBG (to figure out FAI/cFT), ± A4; prolactin; TSH; and morning 17-OHP (if androgens are high or the risk is high). If you think the source is the adrenal gland, think about DHEAS.
3. Think about AMH in adults when there are validated tests and thresholds, especially when ultrasound isn't available or isn't clear. Don't use AMH in teens.
4. The LH/FSH ratio is not a diagnostic tool; it is only optional context.5. A metabolic work-up (OGTT or HbA1c, lipids, blood pressure, anthropometrics) to figure out the level of cardiometabolic risk and what low SHBG/FAI means.

New trails and therapeutic interventions:

AMH as a Target for Treatment

Preclinical and translational research indicates that AMH signaling is involved in follicular arrest and GnRH activity; an AMH-blocking antibody has demonstrated encouraging pathophysiologic reversal in preliminary studies, indicating potential novel disease-modifying therapies in the future. Although not yet a clinical standard, it highlights the potential for a "diagnostic" hormone to evolve into a therapeutic target.

Fertility Prognosis:

A very high AMH level in PCOS means there are too many follicles, but it doesn't mean that assisted reproduction will lead to a live birth. In fact, a very high AMH level can mean a lower chance of live birth and a higher chance of miscarriage after fresh embryo transfer, which suggests problems with the quality of folliculogenesis.

Changes to lifestyle and metabolism:

Because IR interacts with too much androgen, weight management and insulin-sensitizing strategies are still very important. Dietary patterns, such as ketogenic regimens, are being investigated for metabolic and reproductive outcomes. Although results are inconsistent and reliant on adherence, they underscore the metabolic foundations of the hormonal panel.

Limitations of Hormonal Testing in PCOS: Assay variability:

Immunoassays can overestimate or underestimate TT at low female ranges; LC-MS/MS is preferred when available.

- Biologic variability: Pulsatility (e.g., LH), diurnal fluctuations, and cycle phase influence interpretation
- Phenotypic heterogeneity: Not all cases of PCOS exhibit overt biochemical hyperandrogenism; clinical manifestations are essential.
- Age and life stage: AMH and ultrasound thresholds must be interpreted in relation to age; AMH is not a reliable diagnostic marker in adolescents.

Conclusion

The contemporary hormonal panel for PCOS relies on precise androgen evaluation (TT with SHBG-based cFT/FAI; ± A4), contextual indicators (SHBG, LH/FSH as corroborative data), AMH (in adults with established thresholds and assays), and exclusionary tests (TSH, prolactin, 17-OHP).

Using IR, BMI, and phenotype to help you understand the results makes it easier to make a diagnosis and plan care. The 2023 International Evidence-based Guideline connects the steps in diagnosis with the realities of testing and stresses the need for special care for teens and the need to assess metabolic risk. Future research will probably improve AMH's clinical cut-offs, make androgen tests the same all over the world, and look into hormonal pathway modulation (like AMH blockade) as a targeted treatment.

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