

FEATURES OF ANTI-MÜLLERIAN HORMONE LEVELS IN OVARIAN HYPERANDROGENISM DEPENDING ON OBESITY TYPE AND FAT DISTRIBUTION

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Background

Ovarian hyperandrogenism (OHA) is associated with multi-level disturbances of folliculogenesis arising from the interplay of hormonal, metabolic, and immune-inflammatory pathways. The combined effects of body mass index (BMI), body fat distribution, and inflammatory cytokine milieu appear to exert an integrated influence on ovarian reserve, highlighting the need for a comprehensive assessment of these factors when predicting reproductive potential and tailoring individualized management strategies in women with OHA. Anti-Müllerian hormone (AMH) is widely regarded as a key biomarker of the functional follicular pool and a potential indicator of phenotypic heterogeneity in polycystic ovary syndrome (PCOS); however, available evidence on AMH variability across metabolic phenotypes and fat distribution patterns in hyperandrogenic patients remains limited (Ma Y.C. et al., 2025).

Objective

To evaluate serum AMH levels in women with ovarian hyperandrogenism according to obesity phenotype (visceral vs gynoid) and to compare the results with those of clinically healthy controls.

Materials and Methods

Women aged 22–45 years were enrolled and allocated into three groups based on clinical/endocrine and anthropometric characteristics. The main cohort included patients with ovarian hyperandrogenism (OHA) and obesity (BMI ≥ 30 kg/m²). Within this cohort, participants were stratified by fat distribution into a visceral obesity group (waist-to-hip ratio [WHR] ≥ 0.85 ; n=48) and a gynoid obesity group (WHR < 0.85 ; n=17). OHA was confirmed using a composite of clinical, laboratory, and ultrasound criteria. The control group comprised clinically healthy women with normal BMI, regular ovulatory menstrual cycles, and no clinical or biochemical evidence of hyperandrogenism (n=20). Serum anti-Müllerian hormone (AMH)

concentrations were measured by solid-phase enzyme-linked immunosorbent assay (ELISA) using a commercial kit (ELISA Kit for Anti-Müllerian Hormone [AMH], Cloud-Clone Corp., China) according to the manufacturer's instructions. Statistical analyses included assessment of central tendency and between-group comparisons.

Results

In women with ovarian hyperandrogenism (OHA) and visceral obesity (WHR ≥ 0.85), serum AMH levels were markedly reduced compared with healthy controls (2.34 ± 0.14 vs 4.51 ± 0.27 pg/mL; $p < 0.001$). In women with OHA and gynoid obesity (WHR < 0.85), AMH was also lower than in controls (3.52 ± 0.32 pg/mL; $p < 0.01$) but remained significantly higher than in the visceral obesity subgroup.

Discussion

The observed AMH differences are likely driven by the distinct effects of fat distribution on ovarian function. Visceral adiposity, through its stronger association with insulin resistance and chronic low-grade inflammation, may suppress granulosa cell secretory activity and reduce AMH production. In contrast, gynoid fat distribution is typically linked to a less adverse metabolic and inflammatory profile, which may contribute to relative preservation of folliculogenesis and higher AMH levels compared with visceral obesity.