

## 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  $\geq 30$  kg/m<sup>2</sup>). Within this cohort, participants were stratified by fat distribution into a visceral obesity group (waist-to-hip ratio [WHR]  $\geq 0.85$ ; 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 ( $\text{WHR} \geq 0.85$ ), serum AMH levels were markedly reduced compared with healthy controls ( $2.34 \pm 0.14$  vs  $4.51 \pm 0.27$  pg/mL;  $p < 0.001$ ). In women with OHA and gynoid obesity ( $\text{WHR} < 0.85$ ), AMH was also lower than in controls ( $3.52 \pm 0.32$  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.