

17th World Congress of the Academy of Human Reproduction

15–18 March 2017
Rome, Italy

TITLE

AMH AND OVARIAN MORPHOLOGY IN WOMEN WITH ISOLATED HYPOGONADOTROPIC HYPOGONADISM. EFFECTS OF RECOMBINANT HUMAN FSH.

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ABSTRACT

Context: isolated hypogonadotropic hypogonadism (IHH)/Kallmann syndrome (KS), characterized by absent pubertal development secondary to FSH and LH deficiencies, is very rare in women and prevents pubertal ovarian stimulation. Ovarian AMH secretion, and antral follicle count (AFC), have not been studied. Objective: Evaluate AMH, ovarian volume (OV) and AFC in women with IHH/KS, and their responses to recombinant human FSH (rhFSH). Patients: We compared 68 IHH/KS women and 51 healthy women. Interventions: Serum LH, FSH, sex steroids, inhibin B (InhB) and AMH were measured in untreated IHH/KS women and controls. OV and AFC were also evaluated by ovarian sonography. Changes in ovarian morphology and hormone levels during rhFSH administration were assessed in 12 IHH/KS women with low AMH and AFC. Main Outcome Measure(s): AMH, inhB, OV and AFC in women with IHH/KS compared to the controls, and their responses to rhFSH Results: Serum InhB was lower in the IHH/KS women than in the controls ($12,9 \pm 9,6$ vs 74 ± 24 ; $p < 0,0001$). AMH levels were also significantly lower ($26,5$ pmol/l vs $44,4$; $p < 0,001$), but two-thirds had normal values. Mean OV, total AFC, larger AFC (>6 mm) and smaller AFC (≤ 6 mm) were lower in the IHH/KS patients than in the controls: OV (mL) ($1,6 \pm 0,6$ vs $6,2 \pm 1,9$), total AFC ($3,6 \pm 1,9$ vs $12,3 \pm 5$). Ovarian stimulation by rhFSH led to a marked, significant and sustained increase in both serum E2 and InhB levels and in the number of larger AF. Serum AMH and the number of smaller AF increased early during rhFSH stimulation but then declined despite continued stimulation. Conclusions: We show that women with IHH/KS have both low AMH levels and low AFC. However, the decrease of both parameters can be reversed by FSH administration. Serum AMH concentration and AFC should not serve as prognostic markers of fertility in this specific population.

INSTITUTE

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