INTRODUCTION

Gonadotrophin deficiency occurs in a spectrum of conditions with severe life-long absence of LH and FSH in hypogonadotrophic hypogonadism to acquired suppression of gonadotropin releasing hormone (WHO Group I anovulatory infertility disorders).

Studies have previously demonstrated a subgroup of hypogonadotrophic patients with so-called multicystic ovaries. Serum anti-Mullerian hormone (AMH) concentrations correlate with the number of pre-antral and small antral follicles in the ovaries.

AMH is thought to play an important role in both ovarian primordial follicle recruitment and selection of the dominant follicle. Studies have shown a positive correlation between AMH and LH concentrations with a negative correlation between AMH and FSH serum levels in a population of PCO patients. Whether AMH is driving the gonadotropins or visa-versa has not been established.

HH provides a model to explore the relationships between gonadotrophins and AMH.

This study aims to add further discussion regarding the role of AMH, by investigating levels of AMH levels in a group of patients with hypogonadotrophic-hypogonadism with very low levels of serum gonadotropin levels.

RESULTS

12 patients were recruited with age, BMI, amenorrhoea status, AFC, serum gonadotrophin and AMH concentrations measured.

There was a positive correlation between the two variables, Spearman rho=0.79, n=12, p=0.004, with high AFC associated with high levels of AMH.

All cases by definition had very low FSH and LH levels and there was no relationship between AMH and gonadotrophin levels. Neither was there any relationship between those with primary and secondary amenorrhoea. We distinguished two distinct groups of patients, 6 patients with low AFC count and with correspondingly low AMH levels; and a second 6 patients with high AFC and relatively high AMH levels (we subsequently labelled these Group 1 and 2 respectively).

The means (+/-SD) of the above parameters for these two groups are demonstrated in Table 2.

While mean age, FSH, LH and BMI were the same for the two groups, both AMH concentrations and AFC were significantly higher, by design, in Group 2. FSH requirement in Group 2 patients was significantly lower and number of eggs retrieved significantly higher compared with Group 1.

<table>
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<th>Case Number</th>
<th>Age (years)</th>
<th>BMI (kg/m²)</th>
<th>Amenorrhoea</th>
<th>FSH (u/L)</th>
<th>LH (u/L)</th>
<th>AFC</th>
<th>AMH (pmol/L)</th>
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DISCUSSION

We delineated two distinct subgroups of patients with hypogonadotropic-hypogonadism, distinguished by the AFC.

There were significant differences between these subgroups with regard to serum AMH levels and ovarian response to controlled ovarian hyperstimulation during IVF treatment.

Previous studies have also discussed this distinction and suggested that those with hypogonadotrophic-hypogonadism and ultrasound findings similar to polycystic ovaries and a high ovarian response to ovulation induction have indeed co-existent hypogonadotrophic-hypogonadism and PCOS.

While this may be true, further investigation is needed to determine the difference between these two groups of patients with the same ‘umbrella’ diagnosis.

As was expected the AMH levels showed a positive correlation with the number of ovarian antral follicles, which was in keeping with previously published data.

As all patients in this present study had very low levels of gonadotrophins, it gave an opportunity to further delineate the relationship between AMH and endogenous gonadotrophins.

Previous studies have shown a significant correlation between AMH and LH (positive) and FSH (negative). All patients had low gonadotrophin levels but with wide ranging AMH levels, specifically in the second subgroup of patients with the higher AFC count in whom the conclusion must be that the small antral follicles are intrinsic producers of AMH and independent of gonadotrophin influence.

REFERENCES


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