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Circulating anti-Müllerian hormone as predictor of ovarian response to gonadotrophins in women with polycystic ovary syndrome

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Objective *To investigate the impact of high circulating anti-Müllerian hormone (AMH) on the outcome of ovulation induction using human menopausal gonadotropin (hMG) in women with polycystic ovary syndrome (PCOS).*
Methods *This prospective study included 63 anovulatory women with PCOS who underwent hMG ovarian stimulation. Serum AMH concentrations were compared between responders and non-responders. The receiver-operating characteristic (ROC) curve was used to evaluate the prognostic value of circulating AMH.*
Results *hMG responders had a significantly lower serum AMH concentration compared with non-responders (8.43 ± 2.18 μg/L vs 11.05 ± 2.85 μg/L, P<0.001). In multivariate Logistic regression analysis, AMH was an independent predictor of ovulation induction by hMG in PCOS patients. ROC curve analysis showed AMH was a useful predictor of ovulation induction by hMG in PCOS patients, having 91.7% specificity and 66.7% sensitivity when the threshold AMH concentration was 10.12 μg/L.*
Conclusion *Serum AMH can be used as an effective parameter to predict ovarian response to hMG treatment in PCOS patients.*

Key words: polycystic ovary syndrome (PCOS); anti-Müllerian hormone (AMH); human menopausal gonadotropin (hMG); ovulation induction (OI)

Anti-Müllerian hormone (AMH), also known as Müllerian inhibiting substance (MIS), is producted specifically by granulosa cells (GC) of early developing pre-antral and small antral follicles in the ovary, and declines with advancing age. AMH has therefore been proposed as a novel clinical marker of the ovarian reserve[1]. Inaddition, serum AMH levels

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are increased significantly in women with polycystic ovary syndrome (PCOS) when compared with normo-ovulatory women which attract the interest of many researchers[2,3].

PCOS is the most common endocrine disorder in women of reproductive age, with a prevalence of approximately 5%−10%. PCOS is the major cause of anovulatory infertility[4]. Women with this syndrome are characterized by an excessive number of small antral follicles (2−3 fold that of normal ovaries) and a fail in selection of one follicle from the increased pool of selectable follicles, which named follicular arrest (FA)[5]. As an exclusively GC product, it was proposed that the rise in serum AMH level in PCOS patients was a consequence of the increased small antral follicle number in these ovaries[6]. The increased AMH level, as a result, is considered as an important contributor to FA[7]. Recent study found that AMH can reduce the follicle sensitivity to FSH and oestradiol production, thus preventing follicle selection[8].

At present, the treatment of oligo- or anovulatory infertility is referred to as ovulation induction (OI). First-line treatment for OI is using clomiphene citrate (CC). However, 20%−25% of the women are resistant to CC and fail to ovulate. In such cases, gonadotropins (Gn) is recommended as second-line intervention[9].

Recently, several studies demonstrated that serum AMH concentrations accurately predicted ovarian response to Gn during *in vitro* fertilization (IVF)[10-12]. However, no studies have focused on women with PCOS in OI in China. Since the increased AMH would impair the action of FSH and contribute to the FA of PCOS, it appears that those women with the highest AMH concentrations may have the worst outcome. So we designed a study to investigate whether serum AMH has a role in predicting ovary response to human menopausal gonadotrophin (hMG) treatment in a large cohort of infertile women with PCOS.

Materials & Methods

Patients

Subjects included 63 CC-resistant women with PCOS who received OI with hMG (Lizhu Pharmacy) with timed intercourse (TI) between June 2012 and August 2015. The diagnosis of PCOS was based on the Rotterdam criteria, in which at least two of the following three criteria were met: 1) oligomenorrhea or amenorrhea; 2) hyperandrogenaemia; and 3) sonographic appearance of polycystic ovaries. No PCOS patient had evidence of hyperprolactinemia, Cushing's syndrome, congenital adrenal hyperplasia or androgen-secreting tumors. The inclusion criteria included: 1) patients were ⩽ 35 years old; 2) fail to conceive despite having unprotected sex for at least two years; 3) all women had at least one of patent fallopian tubes proved by hysterosalpingography; 4) partners with normal