Introduction

Polycystic ovary syndrome (PCOS) is a highly prevalent (5–10%) endocrine-metabolic dysfunction in premenopausal women and is the most frequent cause of anovulatory infertility and hyperandrogenism in women **(Jonard et al., 2003)**.

Ovarian morphology, assessed by ultrasound and by histological examination, has shown that polycystic ovaries are characterized by an excessive number of growing follicles compared with normal ovaries **(Webber et al., 2003)**, suggesting an altered folliculogenesis in this syndrome.

For many years, different combinations of clinical (irregular menstrual cycles, hirsutism, and acne), biological (elevated serum testosterone or androstenedione levels or increased LH/FSH ratio), and ultrasound (U/S) criteria have been proposed, with very little international consensus. Indeed, the conservative definition for PCOS that was issued from a conference held in the National Institutes of Health in 1992 **(Zawadski et al., 1992)** did not satisfy many authors because it omitted the U/S criteria.

More recently, during another consensus conference held in Rotterdam in 2003 (The **Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group, 2004)** it has been proposed to include in the definition of PCOS the U/S criteria that are considered at the present time as the most specific, namely an increased ovarian volume (>10 ml) and/or the presence of 12 or more follicles in each ovary measuring 2–9 m **(Balen et al., 2003)**.

Anti-Müllerian hormone (AMH), a dimeric glycoprotein member of the TGFß superfamily **(Josso and di Clemente, 1999)**, may constitute a marker of follicular development. AMH is produced exclusively in the gonads **(Lee and Donahoe, 1993)**, and its dimorphic expression in ovary and testis is crucial for the normal differentiation of reproductive structures **(Josso et al., 1993 and Lamarre et al., 1993)**.

During early fetal development, Sertoli cell expression of AMH results in Müllerian duct regression in the male **(Lee and Donahoe, 1993)**. In contrast, in the female fetus, granulosa cell expression of AMH occurs during late gestation, after the Müllerian ducts have lost sensitivity to AMH **(Rajpert-De Meyts et al., 1999)**.

Serum AMH levels seem to be correlated with the development of preantral and small antral follicles, from puberty to the end of reproductive life **(Cook et al., 2000)**. During this period, AMH expression in the human ovary is similar to that observed in the mouse and rat **(Weenen et al., 2004)**, suggesting an important role for AMH in the regulation of early follicle growth.

Recent studies have validated the use of serum AMH levels as a marker for the quantitative aspect of ovarian reserve. Because AMH levels are strongly correlated with the size of the follicle pool, and because of the lack of cycle variations, serum levels of AMH are a good candidate for inclusion in standard diagnostic procedures to assess other ovarian dysfunctions, such as polycystic ovary syndrome (PCOS), in which the antral follicle pool is enlarged. . Knowledge of the serum AMH levels in such conditions might provide more insight into the possible cause or effect of altered AMH levels **(van Rooij et al., 2005)**.