Premature Ovarian Insufficiency

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Introduction
Premature Ovarian Insufficiency (POI) is a condition of hypergonadotropin hypergonadism characterized by loss of ovarian function that occurs before 40 years of age, the cut-off age of 40 years is used as it represents two standard deviations below the middle age of natural menopause [1,2]. It develops in approximately 1% of women, an incidence of 1 in 100 women before age 40 and 1 in 1000 women before age 30 [3]. The frequency tends to vary according to ethnicity, is more frequent in women of Hispanic and African American origin (1.4%), and less frequent in Asian women (0.5%) [1]. The main etiological groups that can develop a POI are genetic causes (30%) and those that stand out mainly X-linked and autosomal mutations; autoimmune causes (4-30%) such as thyroiditis and type I diabetes mellitus; metabolic causes such as galactosemia and infectious causes, and even after procedures such as chemotherapy, radiation or surgeries; however, in most cases, the cause remains unknown (Idiopathic POI) [4].

In Premature Ovarian Insufficiency, the ovaries show a deterioration of their germinative and hormonal functions due to the depletion of the number or dysfunction of the ovarian follicles, in the latter situation the follicle does not respond to gonadotropins, which is preferably associated with enzyme deficiency (17α-hydroxylase, 17,20-desmolase, aromatase) and receptor mutation (FSH, luteinizing hormone [LH], G protein); Unlike menopause, in premature ovarian failure there is no definitive cessation of follicular function and intermittent ovulation may still occur, and natural pregnancies are present in approximately 5% to 10% of cases [1,3,5].

Women with POI should be asked about personal, menstrual, medical, and family history in detail. Positive family history is present in up to 30% of POI cases; therefore, a detailed family history of POI should be sought [6].

The clinical presentation of POI is usually secondary amenorrhea or oligomenorrhea (for at least 4 months), subfertility, and symptoms of estrogen deficiency such as excessive sweating, altered mood, decreased libido, weakness, dry skin and mucosa [2,3,6]. It is the cause in 10% to 28% of women with primary amenorrhea and in 4% to 18% of women with secondary amenorrhea [5]. It is also characterized by low levels of estradiol (<20 pg / ml), due to the result of ovarian disorders in cases in which the feedback mechanism stimulates the pituitary gland to secrete gonadotrophic hormone (high levels of FSH), these levels Insufficient estrogens are associated with metabolic disorders, leading to cardiovascular diseases such as atherosclerosis [3]. However, it can be extremely variable, with few or no symptoms occurring in some women due to intermittent production of ovarian hormones [2].

The diagnosis should be confirmed by two elevated FSH tests, 4 to 6 weeks apart. The most commonly used diagnostic limit is >40 IU/l, although the National Institute for Health and Care Excellence guidelines suggest >30 IU/l and the European Society for Human Reproduction and Embryology guidelines suggest an even lower cut-off value (>25 IU/l). If there is still menstruation, these tests should be done on day 2-3 of the menstrual cycle. However, it is important that POI is not overdiagnosed in patients with irregular menstrual cycles [2].

Additionally, different evaluations should be carried out to investigate the cause of the POI or to evaluate its scope, mainly a hormonal and genetic evaluation, in some cases some imaging tests are indicated to see complications. The hormonal evaluation evaluates FSH, prolactin, thyroid stimulating hormone (TSH) and anti-Mullerian hormone (AMH); and in genetics, the karyotype should be investigated, this should be requested for all women with non-iatrogenic POI, especially before the age of 30, but the routine investigation of autosomal genetic abnormalities is not recommended, except when a specific mutation is suspected [1]. Patients with POI should have their levels of vitamin 25OHD3 measured, since it could diagnose a possible deficiency and prevent loss of bone mass due to lack of estrogen, since bone remodeling is increased, but bone resorption exceeds that of bone formation [3,6].

Regarding its treatment, its objective is the relief of symptoms and the reduction of the consequences of hypoestrogenism, combined with psychosocial support, with special care in reproductive aspects [1]. Hormone replacement therapy remains the first-line treatment for the relief of vasomotor symptoms, the prevention of long-term morbidity and early mortality related to prolonged estrogen deficiency. At present, there is very little evidence regarding the optimal method of hormone replacement [4]. Studies have shown that estrogen can be administered orally or transdermally, the transdermal route is preferred since the first-pass effect through the liver is eliminated, which reduces the risk of venous thromboembolism, in addition to the fact...
that hormonal levels have been shown serum levels closer to age-related physiological levels, this to mimic ovarian hormone production [1,6]. In women with an intact uterus, progestin should be added continuously or cyclically to protect the endometrium. The recommended doses to be administered in hormone replacement is 1 to 2 mg of 17-β-estradiol orally per day, 100-200 mcg / day of transdermal estradiol or 0.625 to 1,250 mg / day orally of conjugated equine estrogen; all this combined with a healthy lifestyle [6].

In conclusion, Premature Ovarian Insufficiency represents an intermittent loss of ovarian function in women under 40 years of age. In most cases the cause is unknown, but it has been related to genetic factors involving the X chromosome, as well as autoimmune factors, among others. An early diagnosis could prevent complications and prompt symptom management for a better quality of life. Treatment should be guided by gynecology, reproductive health, and psychology; estrogen therapy is the mainstay of treatment and with this, satisfactory results have been seen.

References