

Prediction of the Syndrome Premature Ovarian Insufficiency

Tangirova Yulduz Alimovna, Yusupov Shokhrub

Samarkand State Medical Institute, Republic of Uzbekistan, Samarkand, Uzbekistan

ABSTRACT

Premature ovarian failure syndrome is a symptom complex characterized by hypergonadotropic amenorrhea in women under 40. Known causes include: 1. Genetic aberrations that can affect the X chromosome or autosomes. 2. Autoimmune damage to the ovaries, as evidenced by the observed association of POF with other autoimmune disorders. 3. Iatrogenic after surgery, radiotherapy or chemotherapy, as in malignant neoplasms. 4. Environmental factors such as viral infections and toxins, the mechanism of action of which is not known.

KEYWORDS: *Premature ovarian failure syndrome, follicle-stimulating hormone, family history*

How to cite this paper: Tangirova Yulduz Alimovna | Yusupov Shokhrub "Prediction of the Syndrome Premature Ovarian Insufficiency" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-6 | Issue-3, April 2022, pp.1548-1550, URL: www.ijtsrd.com/papers/ijtsrd49766.pdf



Copyright © 2022 by author (s) and International Journal of Trend in Scientific Research and Development Journal. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0) (<http://creativecommons.org/licenses/by/4.0>)



Relevance: POI or premature menopause is a pathological condition characterized by the cessation of the functional activity of the ovaries in women under the age of 40 and manifested by amenorrhea by high levels of gonadotropins in the blood, infertility and symptoms of estrogen deficiency [1]. Today, the frequency of POI, according to various sources, ranges from 1-3% up to 10% of the female population[3]. In December 2015, the European Society for Human Reproduction and Embryology (ESHRE) published guidelines for the management of women with POI, according to which the diagnosis of the disease is established based on the presence of oligo/amenorrhea for at least 4 months. in women under 40 years of age and FSH levels over 25 IU/L in two studies performed 4 weeks apart. The recommended minimum screening plan for suspected POF is: karyotyping, pelvic ultrasound, FMR1 premutation testing, autoimmune antibody testing, densitometry, thyroid antibody testing(4). The causes of POI are heterogeneous and can be represented by genetic, enzymatic, autoimmune, infectious-toxic, iatrogenic and psychological factors, defects in the structures of gonadotropins, as well as their combination [5]. However, despite modern diagnostic capabilities, in most cases it is extremely difficult to

accurately identify the etiology of this disease; therefore, the idiopathic form in the structure of POI continues to account for more than 50% [9]. With idiopathic autoimmune and genetic origin, POI is distinguished by familial and sporadic forms. The frequency of familial forms of POI is 4-31%, the frequency of the idiopathic form is 81-87.3% [6, 8].

There are 3 types of genetic factors in which POI is observed:

- Chromosomal diseases: Shereshevsky-Turner syndrome, violation of the structure of the X chromosome (fragile X syndrome);
- Monogenic diseases caused by a single gene mutation;
- Polygenic diseases caused by the interaction of several genes and external factors [4,7, 11].

Of interest are studies of markers of ovarian reserve, such as anti-Müllerian hormone (AMH). Serum AMH levels follow a decrease in the number of follicles over time in healthy women and fall to very low levels before menopause. It was found that when measuring the level of AMH in different groups of patients with secondary amenorrhea compared with healthy controls, low AMH levels are more common in patients with POI [8]. However, low AMH levels

can also be found in women with regular cycles and low ovarian reserve. The analysis used in most studies to date is not sensitive enough in this context, as AMH levels become undetectable around 5 years before menopause. This may change with the development of technology. It should be emphasized that women attending fertility clinics with low AMH levels but regular menstrual cycles should not be diagnosed with POI.

There is no evidence to support the inclusion of ultrasound in investigations to establish a diagnosis—there are no ultrasound criteria for POI. Since ovarian function can fluctuate in women with POI, follicular activity may be observed, which does not distinguish POI from other diagnoses. There is also no evidence to include laparoscopy as a diagnostic modality for POF, with or without ovarian biopsy.

Premature ovarian failure has received increasing attention from clinicians and researchers over the past two decades as it has become apparent that this is not a very rare disease. The frequency of this syndrome is 3.7%, with an increase in this indicator in developing countries. The prevalence of POI is approximately 1% in women under 40 years of age and 0.1% in women under 30 years of age. Spontaneous POF syndrome affects ~1% of women under the age of 40 years and ~0.1% of women under the age of 30 years. An estimated 5% of women experience early menopause before the age of 45 [9].

The health hazard of women with POI is a deficiency of sex hormones with metabolic disorders, manifested in diseases of the cardiovascular system.

The aim of the study was to determine the prognostic factors for the development of premature ovarian failure syndrome.

Material and research methods

92 women with a diagnosis of premature ovarian failure were examined. The diagnosis was established on the basis of the cessation of menstruation at the age of 40 years. The diagnosis was confirmed by double determination of follicle-stimulating hormone (FSH) in the blood with an interval of 2-4 weeks, the values of which corresponded to the menopausal range (40 IU/l and more). The exclusion criteria for participation in the study were:

- Shershersky Turner syndrome
- iatrogenic etiological factors (history of chemoradiotherapy, hysterectomy and ovarian surgery)
- Tumors of the reproductive system.
- history of cancer

Thus, the study was conducted among patients with idiopathic POI. The study was conducted on the basis

of a carefully collected family history. The mean age of patients with POI was 34.27 ± 0.58 years and ranged from 24 to 39 years. The study of the features of the formation of menstrual function in this group of patients revealed that the average age of menarche was 14.27 ± 0.51 (13-17) years. 67 (73.1%) women complained of infertility and 59 (64.2%) women reported hot flashes. Vaginal dryness was observed in 19 (20.9%) patients. All patients with POI had a normal menstrual cycle in the past before the development of the disease. The onset of menstrual disorders in women with POI averaged at the age of 31.5 ± 0.48 years. The mean age of onset of amenorrhea was 32.5 ± 0.7 years. According to our research, 51 (55.6%) women with POI were not pregnant before the development of the disease. Thus, the study was conducted among patients with idiopathic POI. The study was conducted on the basis of a carefully collected family history.

Results and its discussion

Examination of patients with POI in reproductive age revealed a high incidence of autoimmune diseases, including autoimmune hypothyroidism in 61 (66.7%) patients, diffuse toxic goiter in 17 (19%), rheumatoid arthritis in 6.5 (7.1%), systemic lupus erythematosus in 6.6 (7.2%), as well as the presence of chronic inflammatory diseases. The results of the study showed that in the development of idiopathic POI in 29 (31.5%) family history matters. In the study of menstrual function in 60 (65.2%) women, menarche was at the age of 13-15 years, in 15 (16.3%) early, in 17 (18.4%) later. It was found that half of the examined patients had BMI within the normal range (19.5-24.9). At the same time, the POI syndrome in the mothers of the patients occurred in 11 (12%) cases; in older sisters - in 3 (3.3%) cases; maternal grandmothers - in 9 (9.8%) cases; paternal grandmothers - in 6 (6.5%) cases. Attention is drawn to the fact that the family history of POI is more widespread in grandmothers, both on the mother's side and on the father's side, which in our study totaled 16.3% in relation to all examined and 51.7% in relation to patients with idiopathic POI.

Conclusion:

Early diagnosis of family predisposition can predict impending menopause and these women can be directed to achieve their reproductive goals through timely pregnancy planning. An adequate family history can distinguish between familial or sporadic causes of POI syndrome. The risk of developing POI in female relatives may be higher in familial POI compared with sporadic cases. Another risk factor for the development of POI syndrome can be autoimmune diseases: hypothyroidism, systemic

lupus erythematosus, rheumatoid arthritis, hyperthyroidism. The diagnosis of premature ovarian failure is based on the presence of menstrual irregularities and biochemical confirmation. The diagnosis of POI is usually confirmed in women under 40 years of age by a combination of a 4–6 month period of amenorrhea or oligomenorrhea and two consecutive measurements of elevated FSH levels >25 IU/L more than 4 weeks apart. There are no ideal biochemical markers for the diagnosis of POI. Thus, very few patients receive adequate counseling for the syndrome of premature ovarian failure, which is due to the lack of knowledge and experience of doctors. To this end, the study of this pathology remains an urgent problem for women of childbearing age. All available options for reproductive function should be offered and carried out separately or in combination.

Bibliography

- [1] Belotserkovtseva L.D., Kovalenko L.V., Korneeva E.V. Premature ovarian failure: a modern view on the etiology, pathogenesis and diagnosis (literature review) // Pathogenesis. - 2008 - V.6, No. 4. - pp. 40-44.
- [2] Kumyikova Z.Kh., Batyrova Z.K., Uvarova E.V. Premature ovarian failure in early reproductive age: modern aspects of diagnosis and management // Reproduction. health of children and adolescents. 2019. V. 15, No. 4. S. 53–60.
- [3] Akbari Asbagh F., Ebrahimi M. A case report of spontaneous pregnancy during hormonal replacement therapy for premature ovarian failure. Iran J. Reprod. Med. 2011; 9(1): 47-9.
- [4] Conway GS Premature ovarian failure. British Medical Bulletin. 2014; V 56, P 643–649.
- [5] Fallahian M., Pouresmaelli F., Azizi F., Zali M.R., Samani E.M. Existence of inhibin α -subunit gene mutation in a population of Iranian women with premature ovarian failure. Intern. J. Endocrin. Metab. 2009; 2(7): 67-71.
- [6] Ficiocioglu C., Yildirim G., Attar R., Kumbak B., Yesildaglar N. The significance of the number of CGG repeats and autoantibodies in premature ovarian failure. reproduction. Biomed. online. 2010; 20(6): 776-82.
- [7] Jeong H.J., Cho S.W., Kim H.A., Lee S.H., Cho J.H., Choi D.H. et al. G769A variation of inhibitory alpha-gene in Korean women with premature ovarian failure. Yonsei Med. J. 2004; 45(3): 479-82.
- [8] La Marca A, Brozzetti A, Sighinolfi G, Marzotti S, Volpe A, Falorni A. Primary ovarian insufficiency: autoimmune causes. Curr Opin Obstet Gynecol 2010;22: 277-282; La Marca A, Pati M, Orvieto R, Stabile G, Carducci Artenisio A, Volpe A. Serum anti-mullerian hormone levels in women with secondary amenorrhea. Fertil Steril 2006;85: 1547-1549
- [9] Shannon D. Sullivan, Philip M. Sarrel, Lawrence M. Nelson Hormone replacement therapy in young women with primary ovarian insufficiency and early menopause//Fertil Steril. December 2016; 106(7): 1588–1599. doi:10.1016/j.fertnstert.2016.09.046
- [10] Vujovic S. Aetiology of premature ovarian failure. Menopause International. 2009;15(2): p 72-75.
- [11] Webber L, Davies M, Anderson R, Bartlett J et al: The ESHRE Guideline Group on POI. ESHRE Guideline: management of women with premature ovarian insufficiency. Hum reproduction. 2016; 31(5):p 926-937.
- [12] Woad K.J., Pearson S.M., Harris S.E., Gersak K., Shelling A.N. Investigating the association between inhibitory alpha gene promoter polymorphisms and premature ovarian failure. fertil. Steril. 2009; 91(1): 62-6.