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Reproductive Changes In Women With Premature Ovarian Failure

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Abstract: Failure to become pregnant after therapy forces the patient to use assisted reproductive technologies using donor embryos and oocytes, while the effectiveness of in vitro fertilization does not exceed 58%. To prevent this outcome, if a woman has risk factors for developing premature ovarian failure, it is necessary to timely assess the ovarian reserve with the preservation of her own oocytes for subsequent assisted reproductive technologies.

Keywords: infertility, hypogonadism, premature ovarian failure, hormone replacement therapy.

INTRODUCTION

In the modern world, the time of reproductive debut has shifted to the age of 40 years, and therefore cases of women turning to an obstetrician-gynecologist with complaints of infertility have become more frequent. One of the causes of this condition may be premature ovarian failure, as a result of which the patient loses her reproductive potential before the age of 40. Often, the severity of this pathological process and its consequences remain underestimated, so a detailed study of the disease is necessary, searching for the most optimal methods of drug and non-drug treatment, improving the quality of life and prevention.

MATERIALS AND METHODS

Premature ovarian failure (POF) is a pathological condition caused by primary hypogonadism that occurs in women of reproductive age (before 40 years) with a previous normal menstrual cycle. This clinical syndrome is manifested by menstrual irregularities such as amenorrhea or oligomenorrhea, endocrine changes characterized by a decrease in the secretion of estradiol and an increase, according to the principle of negative feedback, in the level of gonadotropic hormones - luteinizing and follicle-stimulating [1].

The low prevalence of this disease among young women - up to 20 years - 1: 10,000, up to 30 years - 1:1000, up to 40 years - 1:100, determines the doctor’s low alertness towards this pathology, which leads to late diagnosis and the woman’s inability to get pregnant without using assisted reproductive technologies, because in 5-20% of cases, POF causes secondary amenorrhea, and in 6% - endocrine infertility [2].

RESULTS AND DISCUSSION

The cause of premature ovarian failure currently remains controversial. Despite the development of modern research methods and active diagnosis of this pathological process, about 60% is the idiopathic form of POF. There is evidence confirming the autoimmune origin

of the disease. It can occur in isolation or in combination with other autoimmune processes. The most common are autoimmune chronic adrenal insufficiency, which can either precede or manifest itself some time after the onset of premature ovarian failure, autoimmune damage to the thyroid gland (manifested in the form of hypothyroidism) and pancreas (type I diabetes mellitus). Therefore, it is recommended to conduct an annual screening study of the state of the endocrine system of women with an established diagnosis of POF [1,3].

The genetic factor plays an important role in the development of this disease. For the normal functioning of the reproductive system, including the ovaries, the correct structure of both the short and long arms of the X chromosome is necessary. When deletions occur in the terminal part of the proximal part of the short and long arms of the X chromosome, premature ovarian failure or primary amenorrhea develops in 80% of cases. With Martin-Bell syndrome, thinning of the ends of the X chromosome is observed, caused by an increase in the number of repeating nucleotide sequences Cytosine-Guanine-Guanine, which normally should not exceed
45. When repeating nucleotide sequences 56-200 times, a premutation is observed, causing premature ovarian failure in 25% cases [4]. Bone morphogenetic protein 15 (BMP15), located on the short arm of the X chromosome, is involved in folliculogenesis, in which the transformation of primordial follicles into preovulatory follicles occurs. When a mutation occurs in this gene, ovarian failure occurs. With Shereshevsky-Turner syndrome, characterized by the absence of one X chromosome, normal gamete formation occurs in the first trimester of pregnancy, which is subsequently replaced by involutive processes, and by the time of birth the number of follicles in the ovary decreases sharply. However, 6% of girls develop secondary sexual characteristics and experience a normal menstrual cycle [3].

An objective examination determines the correct body type, with the exception of women with Turner syndrome, satisfactory nutrition, the development of subcutaneous fat, and the absence of deviations in sexual development. When examining the mammary glands, glandular-cystic mastopathy may be detected, and during vaginoscopy - a negative “pupil” symptom. The karyopyknotic index is 0-10%. When conducting a bimanual gynecological examination, a decrease in the size of the uterus is determined [4].

Because The main purpose of married couples turning to an obstetrician-gynecologist is to restore reproductive function; it is necessary for young women at risk of developing POF, as well as with a latent form of the disease, to make a timely assessment of the ovarian reserve before its debut in order to use their own oocytes when using assisted reproductive technologies. With the development of the full form of premature ovarian failure, there is no effective method of achieving pregnancy with one’s own gametes, because ovarian stimulation brings positive results only in 5% of cases and is associated with the risk of severe complications. Therefore, to achieve pregnancy in such women, the use of donor embryos and oocytes is used, while the effectiveness of in vitro fertilization is 58%. If a premutation of the FMR1 gene is detected in a woman, ART programs with preimplantation genetic testing for monogenic diseases are necessary [2].

CONCLUSION

Thus, with timely diagnosis before the manifestation of the disease and adequate replacement therapy, it is possible to achieve a favorable clinical condition, prevent complications associated with estrogen deficiency and preserve a woman’s own genetic material for subsequent assisted reproductive technologies. It is necessary to search for new methods of therapy aimed at restoring the reproductive function of women.

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