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Modern Concepts about the Pathogenesis of Polycystic Ovary Syndrome

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Annotation: Polycystic ovary syndrome (PCOS), also known as Stein-Leventhal syndrome, is one of the most common gynecological disorders, affecting 5-20% of young women worldwide across different ethnic groups and races and is often the cause of infertility, miscarriage, and complications during pregnancy. pregnancy and childbirth. PCOS is a polysymptomatic disease characterized by hyperandrogenism, menstrual dysfunction, impaired production of female sex hormones and cystic changes in the ovaries on ultrasound. At the same time, PCOS is a metabolic disorder. The role of insulin resistance in the development of this condition has been proven. As a result, pathogenesis, diagnosis and treatment are of interest not only for gynecologists, but also for endocrinologists, cardiologists and other doctors, since PCOS is a serious problem associated with obesity, increased risks of endometrial adenocarcinoma, hypertension and cardiovascular complications, diabetes mellitus Type 2 and other serious consequences. Today, many researchers from different countries are actively studying the mechanisms of the formation of PCOS. Our article provides an overview of scientific sources devoted to the problem of polycystic ovary syndrome, highlights modern ideas about the etiology and pathogenesis of this disease, characteristic clinical manifestations and modern approaches to the diagnosis of PCOS.

Keywords: polycystic ovary syndrome, hormones, hyperandrogenism, insulin resistance.

Since the time of I. Stein and M. Leventhal, who in 1935 first gave a clear description of polycystic ovary syndrome (PCOS), questions regarding the etiology and pathogenesis of ovarian hyperandrogenism syndrome of non-tumor origin have not lost their relevance. PCOS is one of the most common forms of endocrinopathies in women, mainly of reproductive age. PCOS is a condition that is characterized by pathological changes in the structure and function of the ovaries and can lead to metabolic dysfunction, ovulatory infertility, endometrial cancer, preterm birth, perinatal mortality and other serious consequences. The main signs of PCOS are: hyperandrogenism, menstrual and/or ovulatory dysfunction and polycystic ovarian morphology. The disease is caused by both hereditary and environmental factors [1,2]. In general, PCOS is considered to occur at a similar frequency throughout the world, but prevalence rates depend on the diagnostic criteria chosen and the characteristics of the population sample. Based on the diagnostic criteria of the US National Institutes of Health (NIH) and the European Society of Human Reproduction and Embryology, as well as the American Society for Reproductive Medicine (ESHRE/ASRM), the prevalence of PCOS among women of reproductive age in the United States, Europe, Asia and Australia ranges from 6–9% up to 19.9%. The frequency of detection of the syndrome in women with menstrual irregularities ranges from 17.4% to 46.4%. PCOS occupies a leading place in the population of women with clinical manifestations of hyperandrogenism and is detected in 72.1–82% of cases, while among women with anovulatory infertility – in 55–91% of cases [3,4].

Also, diseases and conditions whose manifestations coincide with the symptoms of PCOS include: pregnancy, hypothalamic amenorrhea, premature ovarian failure, androgen-producing tumors, Cushing's disease, acromegaly [1].

Among the manifestations of PCOS are the following: menstrual irregularities, hirsutism, alopecia, hyperinsulinemia, disorders of carbohydrate and lipid metabolism, obesity, insulin resistance, mental disorders, infertility [6]. The mechanisms of development of PCOS at the level of the hypothalamic-pituitary complex, ovaries, adrenal glands, and adipose tissue are being actively studied. It has been shown that the development of polycystic ovary syndrome can be influenced by various environmental

factors, namely low socio-economic level and unhealthy lifestyle (smoking, overeating, lack of physical activity). However, genetic predisposition is of particular importance in the etiopathogenesis of PCOS. The risk of developing the disease increases by 30–50% in patients with a family history of PCOS. The patterns of inheritance of this disease have not been fully studied, however, taking into account the clinical and laboratory heterogeneity, the syndrome most likely has a polygenic or multifactorial type of inheritance. Candidate genes can be grouped into pathogenetic groups: 1) genes involved in the synthesis and action of steroid hormones; 2) genes responsible for the synthesis and regulation of pituitary hormones; 3) genes responsible for the synthesis and effects of insulin; 4) genes that regulate body weight; 5) genes encoding inflammatory mediators; 6) main genes Discoordination and disturbances in the interaction of LH and FSH, IGF-1, anti-Mullerian hormone (AMH), enzymes involved in the conversion of androgens and, possibly, other factors lead to irregular ovulation in PCOS or its absence [2]. For example, due to insufficient secretion of FSH and local inhibition of the action of FSH in PCOS, the formation of a dominant follicle occurs irregularly [2]. Follicular resistance to FSH can also be caused by other regulators of FSH action in the ovaries. One of these factors is the 2-3 times increased level of AMH in PCOS, which can reduce the sensitivity of individual ovarian follicles to FSH and block the conversion of androgens to estrogens by inhibiting aromatase activity, thereby further promoting the formation of GA. Genetic changes in the FSH molecule itself and its receptor may be partly responsible for some of the differences in sensitivity to FSH in patients with PCOS compared to healthy people. An increase in the level of circulating AMH occurs not only due to an increase in the number of small antral (maturing) follicles that produce AMH, but also due to increased secretion of AMH by the granulosa of these follicles. Although decreased levels of AMH in small primary and transitional follicles in women with anovulatory PCOS may promote the recruitment of additional growing follicles, hypersecretion of AMH in granulosa cells of more mature small antral follicles may subsequently impede further follicular growth through inhibition of FSH and the action of aromatase [15]. Consequently, in patients with the anovulatory phenotype of PCOS, FSH concentrations are usually insufficient to overcome the inhibition of aromatase activity by AMH in the antral follicle.

Thus, PCOS is a multifactorial, genetically determined pathological condition, in the pathogenesis of which disturbances of gonadotropic regulation, hyperandrogenism, insulin resistance, dysfunction of adipose tissue and others play an important role. At the same time, many questions remain open and are the subject of further research. One of the promising directions, in our opinion, is to establish the role of carbohydrate metabolism disorders, and especially the influence of gastrointestinal hormones in women of reproductive age, which will contribute to the pathogenetic substantiation of the early diagnosis and prognosis of PCOS.

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