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MODERN UNDERSTANDING OF POLYCYSTIC OVARIAN SYNDROME

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✓ ***Resume,***

Polycystic ovarian syndrome (TPS) is one of the urgent problems of obstetrics and gynecology, and this disease occurs in 80% of infertility cases. This disease is a complex multifactorial disease, degenerates with multigenicity and is formed under the influence of environmental factors. More than a thousand genes have been found in all tissues and cells of the ovary, which play an important role in the development of this disease.

To study the etiology and pathogenesis of polycystic ovarian syndrome based on genetic tests, which leads to improved diagnosis of the disease and individual assessment of risk factors in each patient, preventive and therapeutic methods are predicted.

Key words: ovaries, infertility, polycystic, multigenital, expression.

СОВРЕМЕННОЕ ПОНИМАНИЕ СИНДРОМА ПОЛИКИСТОЗА ЯИЧНИКОВ

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✓ ***Резюме***

Синдром поликистоза яичников (ТПС) является одной из актуальных проблем акушерства и гинекологии, и это заболевание встречается в 80% случаев бесплодия. Это заболевание представляет собой сложное многофакторное заболевание, дегенерирует с мультигенностью и формируется под влиянием факторов окружающей среды. Во всех тканях и клетках яичника обнаружено более тысячи генов, которые играют важную роль в развитии этого заболевания.

Изучение этиологии и патогенеза синдрома поликистоза яичников на основе генетических тестов, что приводит к улучшению диагностики заболевания и индивидуальной оценке факторов риска у каждой пациентки, прогнозируются профилактические и терапевтические методы.

Ключевые слова: яичники, бесплодие, поликистоз, мультигениталь-ность, экспрессия.

ТУХУМДОН ПОЛИКИСТОЗ СИНДРОМИ ХАҚИДА ЗАМОНАВИЙ ТУШУНЧА

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✓ ***Резюме***

Тухумдон поликистоз синдроми (ТПС) акушерство ва гинекологияда долзарб муаммолардан бири ҳисобланади ва бу касаллик бепуштликнинг 80 %-да учрайди. Бу касаллик кўп омилли мураккаб хасталик бўлиб, мультигенник билан насл суради ва экологик омиллар таъсирида шаклланади. Тухумдон таркибидаги барча тўқима ва ҳужайраларда жойлашган бу касаллик ривожланишида аҳамиятли бўлган мингдан зиёд генлар аниқланган. Тухумдон поликистоз синдроми этиология ва патогенезини генетик текширувлар асосида ўрганиш, бу касаллик диагностикасини яхшилашга олиб келади ва ҳар бир пациентда хавфли омилларини индивидуал баҳолаб, профилактика ва даволаш усуллари башорат қилинади.

Калитли сўзлар: тухумдон, бепуштлик, поликистоз, мультигенлик, экспрессия

Relevance

Polycystic ovarian syndrome (TPN) is one of the urgent problems of obstetrics and gynecology, and it is based on the widespread prevalence of the disease, with an abundance of unresolved issues on its pathogenesis, diagnosis and treatment. TPS accounts for 80% of anovulatory infertility [1]. This disease is a complex multifactorial disease, degenerates with multigenicity and is formed under the influence of environmental factors.

Over the past 5 years, many scientific articles have been published on the circulation of TPS [2], many of which are devoted to the etiology and pathogenesis of the disease. This is the main role of the ethnic factor in the extent of the spread of the disease. In particular, TPS in the USA is 8% for Americans, 4% for Europeans and three percent for Americans. It accounts for 6.8% in Greece and 6.5% in Spain [3].

The most frequent occurrence of TPS was found in Mexicans, that is, it was shown that 13% of cases are associated with insulin resistance and diabetes. The average amount of androgen hormone is determined individually depending on ethnicity and geographical area. Low socio-economic development and unhealthy standard of living are of great importance in the development of TPS [4]. At the same time, the main etiological factor is a genetic disorder.

Currently, a number of candidate genes associated with the development of this disease are found in the connective tissue of the ovary, in the Charvi lubricant, in the follicle fluid, in T-lymphocytes. In 2009, Z.A. Mahamed-Hussein and H. Sara studied 1081 genes, 1066 known and 15 unknown proteins, which, as it turned out, are associated with the TPS sympathocomplex [5]. According to the authors, 468 genes were associated with 339 proteins in TPS, of which 34 were transcription factors, 37 were identification proteins, 35 were dimeric proteins, 50 were protein resins, 13 were cycles, 24 were growth factors and 3 were follistatins. The authors' studies have shown that in the case of polycystic ovaries, a large number of genes are expressed, and changes in gene information were detected in the outbreak of the disease [6]. It was found that aspects related to the TPS generation are associated not only with the mother and sister, but also with men, that is, rapid hair loss in men, a decrease in hormone levels and dependence on insulin resistance were revealed. [7].

In addition, metabolic and endocrine disorders were detected during the fetal period, including a violation of carbohydrate metabolism and a violation of adrenal secretion [8]. The birth of a girl with a low weight is also a dangerous factor for the development of TPS. Hormonal and metabolic disorders in TPS are well studied. Oxidative stress, chronic inflammatory process, disorders of immune response management, hypercoagulation, angiogenesis, apoptosis and iron metabolism disorders play an important role in the pathogenesis of TPS. Hemochromatosis, or multiple accumulation of iron, causes the development of insulin resistance and type 2 diabetes [9]. The participation of vitamin D in the reproduction of TPS is discussed. Why is it said that this vitamin is involved in gene transcription and hormonal regulation. Vitamin D deficiency is considered significant in obesity, insulin resistance, hirsutism, menstrual cycle disorders, pregnancy disorders [10]. In 2015, data were collected on a special set of genes, in which 241 genes, 114 polymorphic nucleotides, 500 biochemical processes were collected, and they were associated with TPS [11]. Currently, 13 years before this period, significant Rotterdam norms were established in the diagnosis of TPS, and they are shown in an illustration of 4 phenotypes: I - hyperandrogenism and chronic anovulation; II - hyperandrogenism and polycystic ovaries; III - chronic anovulation and polycystic ovaries without hyperandrogenism; IV - hyperandrogenism, chronic anovulation and polycystic ovaries. At the same time, it is now unclear whether these 4 phenotypes are related to each other, or one passes into the other.

Antimicrobial hormone (AMG) is widely discussed as a universal marker of TPS. Special studies in 2013 showed that AMH has a high level of specificity and sensitivity (82.8%) in the diagnosis of polycystic ovaries. Its porosity is 4.7 ng/ml [12].

In accordance with the recommendations of the Society of Endocrinologists of the USA, AMH plays an important role in the detection and diagnosis of the androgen hormone in adolescent girls. Currently, the three Rotterdam standards remain the basis for the diagnosis of TPS in adolescent girls [13]. The American Society of Endocrinologists is invited to use hormonal contraceptives for recommendation [14]. On the effect of hormonal contraceptives on the concentration of androgens in TPS in 2014, a systematic analysis was carried out, which leads to a decrease in the total amount of testosterone, an increase in globulin-binding steroids. p. The Uras study showed the effect of acetate on the exchange of chromadine contraceptives ug uglerod [15]. The effect of contraceptives on chronic inflammation and endometrial

dysfunction affecting the pathogenesis of TPS has been confirmed. Estradiol during ovulation reduces the synthesis of 6-interleukin in the follicular phase of kuchaitiradi, then the synthesis of inflammatory cytokines in the luteinized phase of kuchaitiradi [16]. Low progesterone levels in chronic anovulation and TPS lead to disorders of the immune system and autoimmune disorders [17].

Contraceptives used in combination with metformin have a neutral effect on C-reactive protein, carbohydrate and fat metabolism, as well as endocrine activity [18]. As you know, TPS increases the risk of endometrial cancer. Studies conducted in 2014 showed that TPS tripled the risk of developing endometrial cancer in women younger than 54 years [19]. Women are recommended to use antiandrogenic drugs, as the veins suffer from many complications of thromboembolism when using contraceptives. Based on a study by European scientists, the use of acetate chlormatine contraceptives reduced the risk of venous thromboembolism compared with hormonal contraceptives to a minimum [20]. If the patient stops giving contraceptives to get pregnant, the ovulatory cycle is evaluated. The results of the study showed that when stopping taking contraceptives, 42.4% of women have a menstrual cycle [20]. In the presence of TPS in obese women, the body mass index is taken into account, blood pressure and pregnancy tests are carried out.

It is known that women with TPS have a high risk of fetal loss, factors affecting it are androgenemia, insulin resistance, hypercoagulation and high resistance of the uterine arteries. These tumors cause infertility due to impaired folliculogenesis and implantation. The fact is that TPS begins in adolescence in girls and is clinically aggravated on the basis of hyperandrogenemia and immature development of the reproductive system. Uncontrolled onset of menstruation in adolescent girls often occurs with hyperandrogenemia. If hyperandrogenemia develops in a stagnant state, the formation of TPS begins. The lack of progesterone during puberty leads to multiple production of luteinizing hormone, resulting in the production of a lot of testosterone and androgen. It is known that these processes cause a violation of the vegetative control of the body and stress adaptation processes. The maintenance of the internal environment of the body in Meyer depends on the autonomic nervous system, especially it regulates the work of the genitals and the formation of direct reproductive health. If neurotransmitters of the central nervous system incorrectly affect the genitourinary organs, hormone production is disrupted, including hyperandrogenemia, which causes TPS.

Thus, the study of the etiology and pathogenesis of polycystic ovarian syndrome based on genetic studies, which leads to improved diagnosis of the disease and individual assessment of risk factors in each patient, preventive and therapeutic methods are predicted.

LIST OF REFERENCES:

1. Lebby I., Temime B.R., Fedlaoi A., Feki A. Ovarian drilling in PCOS: is it really useful? The Front Is Heaving. 2015;2:30.
2. Erin K. Barthelmess and Rajesh K. Polycystic ovary syndrome Naz: current state and prospects for the future Before Biosci (Eliteed). 2015;6:104-19.
3. Ozziz R., Woods K.S., Reina R., Ki T.J., Knochenhauer E.S., Star B.O. Prevalence and features of polycystic ovary syndrome in an unselected population. //J. Clinic. Endocrinol. Metab. 2004;89:2745-49.
4. Merkin S.S., Ozziz R., Siman T., Calderon-Margalit R., Daviglus M., Kif K., Mathews K., Sternfeld B., Siskovik D. Socio-economic status and polycystic ovary syndrome. //J. Women's health. 2011;20:413-19.
5. Mohamed-hUssein Z.A. Aaron S. Constructing the pathway of polycystic ovary syndrome (PCOS) based on the interaction of proteins associated with PCOS obtained from bibliomic data. Theor. //This is a BIAL. Med. model. 2009;6:18.
6. 6.Uvarova E.V., Khatsenko E.P. Polycystic ovary syndrome from the perspective of modern pathogenesis. Reproductive health of children and adolescents. 2013. 7c.
7. Dushkova M., Chermakova I., Hill M., Vankova M., Samalakova P., Starka L. What are the markers of the male equivalent of polycystic ovary syndrome? Physiol. Usage 2004;53:287-94.
8. Gur E.B., Karadeniz M., Turan G.A. Fetal programming of polycystic ovary syndrome. The world of S. Diabetes. 2015;6(7):936-42.
9. Escabar-Morreale iron metabolism and polycystic ovary syndrome. Trends of the endocrine system. Metab. 2012;23(10):509-1.
10. Irani M., Merhi Z. The role of vitamin D in ovarian physiology and its effect on reproductive function: a systematic look at infertility. 2014;102(2):460-68.
11. Joseph S., Barei R.S., Focrea R., Idikola-Thomas S. PCOSKB: knowledge base on

genes, diseases, ontology terms and biochemical pathways associated with polycystic ovary syndrome. Nucleic Acids, Res. 2016; 44(D1).
12. Iliadromiti S., Kelsey T.U., Anderson R.A., Nelson S.M. Can the anti-Muller hormone predict the diagnosis of polycystic ovary syndrome? Systematic review and meta-analysis of extracted data. //J. Klin. Endocrinol. Metab. 2013;98(8):3332 40.
13. Legro R.S., Arslanyan S.A., Ermann D.A., Hoger K.M., Murad M.H., Pasquali R., Welt K.K. Endocrine Society. Diagnosis and treatment of polycystic ovary syndrome: a guide to the clinical practice of the Endocrine Society. //J. Klin. Endocrinol. Metab. 2013;98(12):4565-92.
14. Caruso S. Sorry., Malandrino S., Flower., Lo Presti L., Cianci Hyperandrogenic women receiving oral contraceptives of continuous action. //Eur. J. Obstetrics. Gynecol. Reprod. This is BIAL. 2013;171(2):307-10.
15. Ores R., Arro M., Pani F., Maratta M.F., Pilloni M., Guerriero S., Etzi R., Zedda P., Sorge R., Lello S., Melis G.B., Pauletti A.M. Endocrinological, metabolic and clinical features of treatment with oral contraceptives that continue taking ethinyl estradiol plus chlormadinone acetate in non-obese women with polycystic ovary syndrome. //Contraception. 2010;82(2):131-38.
16. Angstwurm M.U., Gartner R., Ziegler-Heitbrok H.U. Levels of IL-6 in blood plasma during the normal menstrual cycle. Cytokine. 1997;9:370-74.
17. Petrikova J., Lazurowa I., Yehuda S. polycystic ovary Syndrome and autoimmunity. //Eur. J. Intern. Med. 2010;21:369-71.
18. Ilie R. I. Marian I., Masan T., Elijah R., Mahan, L., Dana I., S. E. Pepin Этинилэстрадиол30 Mcgrenere and Metformin: can this combination to improve endothelial dysfunction in polycystic ovary syndrome? BMC endocrinologist. Disord. 2012;12:9.
19. Barry S.A., Azizia M.M., Hardiman P.J. The risk of endometrial, ovarian and breast cancer in women with polycystic ovary syndrome: a systematic review and meta-analysis. This is a Drone. Reprod. Update. 2014;20(5):748-58.
20. Shindler A.E. Non-contraceptive advantages of oral hormonal contraceptives // Endocrinol. Metab. 2013;11(1):41-7.-7.

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