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Polycystic Ovary Syndrome (Pcos)

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Annotation: Polycystic ovary syndrome (PCOS) - fairly common disease which is one of the most relevant problems in gynecological endocrinology. IN review article presents modern data on the etiology, pathogenesis of PCOS. Given rationale for the frequent combination of hyperandrogen and insulin resistance in PCOS. Also characteristic clinical manifestations of PCOS and modern approaches to diagnostics and treatment, including drugs rats of neurotransmitter action (sibutramin, orlistat), drugs of the insulinsensitizers - metformin, progestogens or low-dose single-phase COCs with progesterone 3-generation tagens, synthetic anti-esstrogens - clostilbegit, and other drugs funds. Perspectives are being considered and methods of surgical intervention and tactics for infertility against the background of PCOS.

Key words: PCOS, infertility, sibutramine, orlistat, COC, hyperandrogenemia, insulin resistance, progestogens

Polycystic ovary syndrome (PCOS) - one of the most urgent problems of gynecological logical endocrinology. It is a heterogeneous, hereditary disease characterized by menstrual disorders trual cycle, chronic anovulation and infertility diarrhea, hyperandrogenism (HA), cystic change ovaries and an increase in their size. PCOS mostly occurs in women of reproductive active age, among which the frequency of the disease is 4–12%. Detection rates of this disease are quite variable due to heterogeneity of clinical and endocrinological
manifestations and the ambiguity of their assessment. IN various European distribution studies The prevalence of PCOS is 6.5–8%. Especially often the disease occurs in patients with anovulatory infertility, hyperandrogenism and associated her dermatopathies. It should be noted that in the last. For a decade, there has been an increase in the number of patients who do not have typical manifestations of PCOS.

Discussions about terminology in our country, as in the world, ceased - at the present time adopted a single term "syndrome of polycystic ovaries (PCOS).

The pathogenesis of PCOS appears to be extremely complex and, despite the large number of proposed other theories of the development of the disease, none of them revealed to the end the causes of occurrence and mechanisms of development of endocrinological and metabolicic disorders in this disease. A lot of supporters of the hypothesis of the formation of PCOS on against the background of the primary (from the pubertal period) disturbed of the circoral rhythm of gonadoliberin. During puberty, which is critical in the life of a girl, many factors surround environment, heredity, etc. can contribute to lead to the development of a number of endocrinopathies. Undoubtedly, stress plays an important role in increases the synthesis of opioids (β-endorphin) and the neuroendocrine control of the regulation secretion of GnRH and gonadotropins. At the same time, the basal the level of LH secretion is growing against the background of a relative a significant decrease in FSH production. Increased stimulation of LH disrupts the process of folliculogenesis: in cystic atresia of the follicle is formed in the ovaries fish with hyperplasia of theca-cells, stroma and increased androgen synthesis. Against the backdrop of a lack of FSH there is an accumulation of androgens and a deficiency of estradiol. The latter stimulates the synthesis of LH, increasing his baseline. The emerging GA is also promotes follicular atresia, hyperplasia stroma of the theca cells and albuginea.

In addition, it is known that the degree of GA is positively correlates with the level of inhibin B, which suppresses FSH secretion.

An increase in GnRH secretion can also be secondary nym, as a reaction to the hyperproduction of androgens and decrease in the synthesis of estradiol in the ovaries. The pathogenesis of gonadotropic dysfunction in patients with various clinical and morphological forms of PCOS are different: central and local

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(ovarian). Primary disruption of steroid biosynthesis can be the result of a violation of auto-paracrine regulation growth and maturation of primordial follicles catching, as well as dysregulation of cytochrome P450c17. In my
turn, a decrease in the level of estradiol stimulates secretion of GnRH. Ovarian GA may occur in patients with normal levels of gonadotropins and elevated
reaction of theca-cells of the PCOS to a normal level of LH. In the future, excessive secretion of androgens occurs through the same mechanisms. There is evidence to support that androgen-secreting tissues PCOS to LH higher, i.e. suppression of gonadotropic function reduces the synthesis of androgens in the theca cells of PCOS: there is an autonomous secretion of androgens in the PCOS under the influence of local factors.
Thus, there are endo- and exogenous factors (genetic, metabolic, etc.),
contributing to dysfunction of the hypothalamus pituitary system, leading to increased synthesis of LH. It has been found that sex hormones action is mediated by a number of growth factors (EGF, IGF-I, FGF, etc.), which affect steroidogenesis in ovaries auto-, para- and intracrine effects. Clinicians drew attention to the frequent combination research on GA and insulin resistance (IR) in PCOS. IR - decreased response of insulin-sensitive tissues on insulin despite its sufficient concentration in the body. In the future, IR leads to chronic compensatory hyperinsulinemia (GI) in turn, is an important link in the GA.
There is a hypothesis about the important role of obesity and GI in the pathogenesis of PCOS in IR patients. However, GI noted occurs in women without obesity, so excess body weight can only be a factor contributing to
development of IR in PCOS. The mechanisms of IR have not been finally established, however, there is evidence that they are due to responses at the receptor and post-receptor levels transmission of the insulin signal to the cell.
An important mechanism of IR may be a violation insulin metabolism in the liver, controlled C-peptide and β-endorphins. Latest play an important role in the neuro-endocrine control of secretion gonadoliberin, therefore, with PCOS may be involved in the pathogenesis of IR in some patients with PCOS.
A certain role in peripheral IR belongs to belongs to GA, since androgens change the structure muscle tissue in the direction of the prevalence of muscle
type II fibers that are less sensitive to insulin. Concomitant obesity, more often visceral, in about 50% of patients aggravates existing impaired insulin sensitivity synergistic effect. GA also contributes to an increase in the concentration "free" testosterone by reducing the formation sex steroid-binding globulin (PSSH) in the liver. It has been established that insulin regulates the production of PSSH: with GI, the synthesis of the latter it decreases, which leads to an increase in the concentration walkie-talkies of "free" testosterone and estradiol with relevant clinical manifestations. In the formation of the GA, an important role belongs to adipose tissue in which extragonadal synthesis of sex steroids, independent of stimulus gonadotropins. For patients with PCOS, thorno-android (visceral) obesity. Express-obesity rates are positively correlated with blood testosterone levels. The development of visceral obesity occurs on against the background of a violation of neuroendocrine control of the center moat in the hypothalamus, which are responsible for "food behavior". The same mechanisms increase the synthesis corticoliberin, ACTH, activating the production of steroids of the adrenal cortex. At the same time, it rises as production of adrenal androgens, and cortisol,which enhances metabolic disorders.

Adipocytes in android obesity have a high which density of β-adrenergic receptors, corticosteroid and androgen receptors and relatively low density of insulin receptors. This causes high sensitivity of visceral fat to action of catecholamines (lipolytic) and low to the action of insulin (anti-lipolytic).
Many PCOS patients with normal weight body and concomitant adrenal hyperandrogenius most logically overproduction of androgens explains genetically determined dysregulation cytochrome P450c17, a key enzyme in
androgen synthesis in both the ovaries and adrenal glands. The activity of this cytochrome is regulated mechanisms involved in the activation of insulin receptor. In this regard, the hypo-generality and genetic determinant thesis formation of ovarian, adrenal GA and IR in a group of patients with PCOS at normal weight body.
There is evidence that in patients with PCOS in the blood an increased concentration of the inhibitor is determined apoptosis, i.e. the process of physiological atresium and follicles, creating conditions for their

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persistence tendencies. Moreover, GI has been found to suppress mechanisms of apoptosis. The role of various growth factors in the pathophysiology of PCOS, in particular vascular endothelial growth factor - (VEGF), responsible for the processes of neoangiogenesis. Thus, the pathogenesis of PCOS is complex multicomponent and multifactorial with participation in the pathological process of the central regulatory systems, as well as ovaries, adrenal glands and other factors and has different mechanisms in patients with normal body weight, obesity, IR. Characteristic clinical manifestations of PCOS are oligomenorrhea, anovulation, infertility, obesity, excessive hair growth, acne, etc.

For PCOS in most obese patients burdened heredity took place: menstrual and reproductive disorders , as well as more common diabetes mellitus, parothyroid gland and hypertension. During puberty, these patients often endure forces of neuroinfection, ENT diseases, operations and injury. The age of menarche is timely, 12–13 years. For patients with PCOS are characterized by the presence of oligomenorrhea, and the intervals between menstruation gradually elongate until the development of secondary amenorrhea in the absence of timely adequate therapy. In the vast majority of patients (90-95%) infertility due to anovulation. At the same time, when pregnancy occurs, more often of all, its spontaneous interruption occurs in 1st trimester.

Approximately half of PCOS patients develop against the background of obesity, which is assessed using body mass index, BMI (normal = 19–25): ratio body weight (kg) to height (m2). About excess weight bodies are indicated by indicators of 26–30, and obesity is 30. PCOS is characterized by the distribution of fat tissues of android type (visceral obesity). The deposition of adipose tissue is observed mainly in anterior abdominal wall, internal mesentery organs and areas of the shoulder girdle. With this type obesity in most patients with PCOS (>70%) IR takes place. In addition to BMI, it is advisable to measure the ratio of the circumference waist (FROM) of the patient to the circumference of the hips (OB).

This index characterizes the type of obesity and the risk occurrence of metabolic disorders. While- If RT / R > 0.85, it should be assumed that there is visceral obesity in a patient. Among androgen-dependent dermatopathies, characterized by thorns for PCOS should be called overgrowth hair, the degree of expression of which is necessary evaluate in dynamics, against the background of therapy,

What are the rating scales used for? hirsutism (Ferriman-Gallway, Baron, etc.). Required we want to find out not only the time of occurrence hirsutism, but also try to clarify the source of hyperandrogeny, as well as to evaluate the dynamics of its development. Another symptom of HA is the appearance of "acne" on the skin of the face, back and chest. Significantly less often (5–8%) in PCOS, there are virilization symptoms: lowering the timbre of the voice, clitoral hypertrophy, alopecia.

In patients with PCOS and obesity, IR often manifests itself is "nigroid acanthosis": in places of increased skin friction (on the folds of the skin of the elbows and fingers, in the inguinal and axillary region, under the breasts, etc.) there are areas of hyperpigmentation.

In PCOS against the background of chronic anovulation and hyperestrogenia in most patients develop dyshormonal diseases of the dairy. Approximately 20% of women have colostrum secretion for background of functional hyperprolactinemia. On gynecological examination, approximately 65% of patients have external hair growth male genitalia, symmetrical enlarged ovaries with a dense consistency.

Collecting anamnesis, it is necessary to identify hereditary factors in the development of the syndrome. After inspection for the purpose diagnosis of overweight and obesity it is necessary to calculate the body mass index and the ratio waist circumference to hip circumference (normal <0.85).

Common clinical symptoms with PCOS are: menstrual irregularities (usually - oligomenorrhea, less often – disfunctional uteral bleedings, with lack of adequate treatment - amenorrhea II), anovulation, infertility, hirsutism.

The most important method for diagnosing PCOS is echography, including TsDK and ID. Currently The following ultrasound signs of PCOS are generally recognized: an increase in ovarian volume (V> 8–9 ml), the presence sets (more than 10–12 in one plane with scanning) follicles with a diameter of 3–8 mm, located under a thickened shell, thickened enlarged stroma of increased echogenicity, occupying more than 25% of the volume of the ovary. Thanks to the use training of modern ultrasound machines (including for 3 dimensional study) is currently possible make an accurate assessment of the number of follicles and stroma volume, as well as the ratio of these indicators. It has been found that the presence of a large number small follicles is combined with a higher LH and testosterone levels. It is also known that there positive correlation between the degree of IR, levels of LH, testosterone and ovarian volume, hyper-

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echogenicity of their stroma. Ultrasound allows for differential diagnosis with multifolliclar ovaries (MFN), which can be observed be given at early puberty, with hypogo-supratropic amenorrhea, chronic inflammatory processes and other states.Echo signs of MFN is the presence of diffuse located, against the background of a small number stroma of follicles with a diameter of 4–10 mm, with normal ovarian volume.

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