

Modern Views on Diagnosis and Treatment Methods Polycystic Ovary Syndrome

Olimova N. I.

Bukhara State Medical Institute

Annotation: The polycystic ovary syndrome is one of the most frequent endocrinopathies at women of reproductive age. Frequency polycystic ovary syndrome makes about 30% among patients of gynecologists-endocrinologists, and in structure endocrine baronesses reaches 75%. To 95% of cases of hirsutism at women it is connected with a polycystic ovary syndrome. Diagnostics methods are extensive and are not limited only to reproductive sphere. In article the basic methods of diagnostics and treatment strategy patients with polycystic ovary syndrome are presented.

Keywords: polycystic ovary syndrome, infertility, ovary, hyperandrogenism, hirsutism, ovulation.

Polycystic ovary syndrome (PCOS) is a pathology of the structure and function of the ovaries, the main criteria of which are chronic anovulation and hyperandrogenism. Polycystic ovary syndrome was first described by D. Stein and D. Leventhal in 1935. The incidence of PCOS is about 30% among patients of gynecologists and endocrinologists, and in the structure of endocrine infertility reaches 75%. Up to 95% of cases of hirsutism in women are associated with polycystic ovary syndrome [1, 2].

Diagnostics

A characteristic history, appearance and clinical symptoms facilitate the diagnosis of PCOS. In a modern clinic, the diagnosis of PCOS can be made without hormonal studies, although they also have characteristic features.

1. Taking an anamnesis (hereditary predisposition).
2. Objective examination: weight-height indicator, body type, uterine body is smaller than normal (30%), enlarged ovaries (41%).
3. Functional diagnostic tests: anovulation (88.5%); measurement of basal temperature; low estrogen saturation.
4. Ultrasound of the pelvic organs. Ultrasound should be performed in all patients with suspected PCOS. Patients with PCOS are characterized by a decrease in the anteroposterior size of the uterus and an increase in the volume of the ovaries compared to the norm. The diagnosis of PCOS can be established by transvaginal ultrasound, based on clear criteria for the echoscopic picture: the volume of the ovaries is more than 9 cm³, hyperplastic stroma makes up 25% of the volume, more than 10 atretic follicles with a diameter of up to 10 mm, located along the periphery under a thickened capsule [3].
5. The most informative modern methods are computed tomography and magnetic resonance imaging to exclude pituitary tumors. In 2/3 of patients no pathological changes are detected, and in 1/3 of patients an increase in the size of the sella turcica and osteoporosis. In the central form, osteoporosis or thickening of the bones of the cranial vault, reduction in size and narrowing of the entrance of the sella turcica [4, 5].
6. MRI (CT) of the adrenal glands – detection of hyperplasia of the adrenal cortex, exclusion of adrenal tumors.
7. Study of blood hormones: in patients with oligomenorrhea, hormone levels are determined on the 5th–8th day of the menstrual cycle, with amenorrhea - on any day. The basal level of LH exceeds

normal levels in 70% of patients, a decrease in the basal level of FSH is diagnosed in 30% of cases. The most characteristic sign of PCOS is the ratio of LH and FSH. The LH/FSH index is normal = 1–1.5, with PCOS – LH/FSH>2.5. Prolactin in PCOS is within normal limits or has a borderline value. Hyperprolactinemia (>700 mIU/l) occurs in ~7% (more often in central forms of the syndrome). Increased levels of total and free testosterone, 17-hydroxyprogesterone with normal DHEA-S levels. After a test with dexamethasone, the androgen content decreases slightly, by approximately 25% (due to the adrenal fraction) [6, 7, 8].

8. Metabolic disorders in PCOS are characterized by: increased levels of triglycerides, LDL, VLDL and decreased HDL. In clinical practice, a simple and accessible method for determining impaired glucose tolerance to insulin is the sugar curve. Insulin levels and androgen levels in the body are in balance. Hyperinsulinemia (HI) activates cytochrome P-450 and, as a result, the production of androgens increases, and the body, in turn, loses sensitivity to insulin [1, 7, 9, 10]. To determine insulin resistance (IR), the following indices are used: CARO index = fasting plasma glucose/fasting plasma immunoreactive insulin (IRI) level. Must be less than 0.33. HOMA-IR = (fasting IR level \times glycemic level) /22.5. Normal = 2.6–2.8. A value above 2.8 indicates the presence of insulin resistance.
9. Macroscopic signs of polycystic ovaries: increased size, smooth pearly white capsule of high density, through which small cysts are visible and the vascular network is visible. On section, the ovarian tissue is whitish-gray in color with occasional yellow splashes. On the periphery of the ovaries there are many cysts up to 0.5–1 cm in diameter. The tunica albuginea is unevenly thickened, sclerotic, with the presence of small groups of sclerotic blood vessels. The process of fibrosis is most pronounced in the superficial parts of the medulla [11].
10. Endometrial biopsy: indicated for women with acyclic bleeding due to the high frequency of endometrial hyperplastic processes.
11. According to indications - REG, consultation with a neurologist, ophthalmologist.

Thus, the diagnosis of PCOS is made based on the following data:

- timely age of menarche;
- disturbance of the menstrual cycle from the period of menarche in the vast majority of the oligomenorrhea type;
- hirsutism;
- more than half of women have obesity since menarche;
- primary infertility;
- chronic anovulation;
- increase in ovarian volume due to stroma and cystic follicles according to transvaginal echography;
- increasing testosterone levels;
- increase in LH and LH/FSH ratio [12-19].

Differential diagnosis of PCOS is carried out with hyperandrogenism caused by adrenogenital syndrome, as well as with virilizing tumors of the ovaries and/or adrenal glands.

As a rule, patients with PCOS consult a doctor with complaints of infertility. Therefore, the goal of treatment is restoration of ovulatory cycles.

For PCOS with obesity and normal weight

The sequence of therapeutic measures varies.

1. In the presence of obesity, the first stage of therapy is normalization of body weight. Reducing body weight against the background of a reduction diet leads to normalization of carbohydrate and

fat metabolism. In addition, against the background of a decrease in IR, automatically LH decreases, i.e. Folliculogenesis is restored on the diet. The diet involves reducing total calories food up to 1500–2000 kcal per day. Increased physical activity is an important component not only for normalization of body weight, but increased sensitivity muscle tissue to insulin. The most important thing is to convince the patient of the need to normalize body weight, as the first stage in the treatment of PCOS [3, 13]. If diet therapy is ineffective, drug treatment of obesity is used.

2. The second stage in treatment is medication therapy for metabolic disorders (IR and HI) in case of lack of effect from a reduction diet and physical loads. A drug that increases sensitivity peripheral tissues to insulin, is metformin from the biguanide class. Metformin leads to a decrease peripheral IR, improving glucose utilization in the liver, muscles and adipose tissue, normalizes lipid profile blood, reducing triglyceride and LDL levels. A drug prescribed for 3–6 months. under the control of a glucose tolerance test. Non-obese patients – 500 mg 2 times a day, with obesity – 850 mg 2 times a day [1, 2, 14, 15].

3. Antiandrogen therapy:

- oral contraceptives (COCs) (estrogen-progestin drugs). Their estrogen component suppresses androgen production in the ovary and leads to increased levels globulin that binds sex hormones, and progestogen the component suppresses LH production, normalizes LH/FSH index. The most effective, according to clinical studies, are COCs containing desogestrel, gestodene, and norgestimate.
- Spironolactone (veroshpiron) has weak antiandrogenic activity, blocks peripheral receptors and the synthesis of androgens in the adrenal glands and ovaries, reduces the activity of cytochrome P450c17; suppresses activity enzyme 5a-reductase, promotes weight loss bodies. Prescribed 200–300 mg in the 2nd phase of the menstrual cycle, since uterine bleeding occurs continuously for up to 1 year.

Contraindicated in the presence of mastopathy [16].

- Flutamide (flucinone), 250 mg – a non-steroidal antiandrogen used to treat prostate cancer. Possesses pronounced antiandrogenic activity. The mechanism of action is based mainly on inhibition of hair growth by blocking receptors and slight suppression testosterone synthesis. Prescribed for 6 months, 250–500 mg per day in 2 doses. For severe hirsutism treatment can be extended up to 12 months [1, 2, 6].
- Finasteride is a specific inhibitor of 5a-reductase, under the influence of which the formation of active dihydrotestosterone.
- Androcur is an antiandrogen, antigonadotropic, progestin drug. 1500 times stronger than progesterone. Fine suppresses LH synthesis. Prescribed at a dose of 50 mg/day from the 5th to the 14th day of the menstrual cycle.

4. Infertility treatment: stimulation of ovulation

- clomiphene citrate (clostilbegit) reduces the effect estrogen on the pituitary gland and helps increase the production of FSH, which stimulates follicle growth. Ovulation on during clomiphene therapy develops in 80% of patients, pregnancy - 65%. Reduces the level of insulin-like growth factor-1 in the blood, reduces testosterone synthesis, stimulates the development of the corpus luteum of the egg. In the absence of ovulation, the dose of clomiphene can be increased in each subsequent cycle by 50 mg, reaching 200 mg per day. In the absence of ovulation at the maximum dose for After 3 months, the patient can be considered resistant to clomiphene [3]. In the absence of pregnancy against the background of ovulatory periods cycles requires exclusion of peritoneal factors infertility during laparoscopy [3,17-24].
- Gonadotropin preparations are used when clomiphene is ineffective. Ovulation can be achieved in 94% of cases; pregnancy after several courses of drug administration occurs in 40% of cases.

Preparations:

- 1) with the action of FSH - pergonal, humegon, metrodin, puregon;
- 2) with the action of LH - choriogonin, prophasia, pregnyl.

In the presence of hormonal and ultrasound indicators of ovulation while taking clomiphene, it is recommended to administer an ovulatory dose of 7500–10000 human chorionic gonadotropin (hCG) - (prophase, pregnyl), after which ovulation is noted after 36–48 hours. It's important to remember that early administration of hCG may lead to premature luteinization of the immature follicle, and later administration hCG – to a luteolytic effect [3, 13, 17].

- In case of luteal phase deficiency, it is recommended prescribe gestagens in the second phase of the cycle from the 16th to the 25th day. In this case, progesterone preparations are preferable (duphaston), since norsteroid derivatives can have a luteolytic effect.
- Suprefact (buserilin, diferillin, zoladex).
- Lutrolef-cyclomat – use in circhoral mode, every 90 minutes, 20 mcg, for 10 days (from the 1st to the 10th day of the menstrual cycle). Allows achieve ovulation in more than 90%.
- Dopamine agonists – normalize the secretion of gonadotropins, LH decreases, LH/FSH index is normalized, the sensitivity of prolactotrophs to dopamine increases, Prolactin decreases, steroidogenesis is normalized.
- In case of I degree of hypoestrogenism (according to functional diagnostic tests and the LH/FSH ratio of 1.5-2.5), treatment with vitamin-mineral complexes with high content of antioxidant vitamins.
- Surgical treatment involves laparoscopic diathermy or laser drilling. Ovulation after surgery is restored in 90% of women, pregnancy in the next 8 months it occurs in 80%.

Methods of surgical treatment of PCOS:

- 1) Wedge resection of the ovaries. Pathophysiological mechanisms of wedge resection in stimulating ovulation based on reducing the volume of steroid-producing ovarian stroma. As a result, the sensitivity of the pituitary gland to GnRH is normalized and the hypothalamic-pituitary-ovarian connections are restored.
- 2) In addition to wedge resection, during laparoscopy It is possible to perform cauterization of the ovaries, which is based on the destruction of the stroma with a point electrode. Operation less traumatic and long-lasting compared to wedge resection. The frequency of ovulation induction after surgical laparoscopy is 84–89%, the onset pregnancy is observed in 72% on average [3, 7].

LITERATURE

1. Azziz R. Diagnosis of Polycystic Ovarian Syndrome: The Rotterdam Criteria Are Premature. *Journal of Clinical Endocrinology & Metabolism*, 2006. № 91 (3). P. 781-785.
2. Ehrmann DA. Polycystic ovary syndrome. *N Engl J Med*. 2005. № 352. P. 1223- 1236.
3. HIV, O. N. C. S. I. (2020). INFECTED WOMEN WITH INFLAMMATORY DISEASES OF THE GENITALS. *International Engineering Journal For Research & Development*, 5-5.
4. Homburg R. Diagnosis and management of polycystic ovary syndrome. Springer US. 2009. P. 238.
5. Ismatillayevna, O. N., Abulovna, M. F., & Ortiqovna, S. Z. (2022). RESULTS OF THE STUDY OF WOMEN'S IMMUNE SYSTEM IN INFECTIOUS DISEASES OF SMALL BELLY ORGANS. *World Bulletin of Public Health*, 16, 87-92.
6. Ismatilloevna, O. N. (2023). Hematological-Biochemical and Morphological Indicators of Laboratory Animals under the Influence of Gene-Modified Soybean. *INTERNATIONAL JOURNAL OF HEALTH SYSTEMS AND MEDICAL SCIENCES*, 2(4), 97-103.

7. Nafiye Y., Sevtap K., Muammer D. et al. The effect of serum and intrafollicular insulin resistance parameters and homocysteine levels of nonobese, nonhyperandrogenemic polycystic ovary syndrome patients on in vitro fertilization outcome. *Fertil. Steril.* 2010. № 93 (6). P. 1864–1869
8. Olimova, N. I. (2022). Analysis of the somatic and reproductive history of women with genital inflammatory diseases due to hiv infection. *Актуальные вопросы экспериментальной микробиологии: теория*, 1(2), 30.
9. Olimova, N. I. (2022). The Role Of Immunological Factors In The Pathogenesis Of Hiv Infection In Women Of Reproductive Age With Genital Inflammatory Diseases. *Journal of Pharmaceutical Negative Results*, 2695-2700.
10. Olimova, N. I., & Ikhtiyarova, G. A. (2021). Clinical and immunological features of Inflammatory diseases of the pelvis and genital Organs in hiv-infected women. *Psychology and education*, 58(2), 4996-5001.
11. Гинекология- национальное руководство под ред. В.И, Кулакова, Г.М. Савельевой, И.Б. Манухина. 2009. 532 с.
12. Манухин И.Б., Тумилович Л.Г., Геворкян М.А. Клинические лекции по гинекологической эндокринологии. М.: ГЭОТАР-Медиа, 2006. 320 с.
13. Назаренко Т.А. Синдром поликистозных яичников: современные подходы к диагностике и лечению бесплодия. М.: МЕД пресс-информ, 2005. 208 с.
14. Олимова, Н. И. (2023). СОВРЕМЕННЫЙ ВЗГЛЯД НА ЭТИОПАТОГЕНЕЗ ВИЧ-ИНФЕКЦИИ У ЖЕНЩИН РЕПРОДУКТИВНОГО ВОЗРАСТА С ВОСПАЛИТЕЛЬНЫМИ ЗАБОЛЕВАНИЯМИ ПОЛОВЫХ ПУТЕЙ. *BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIIY JURNALI*, 3(4), 276-279.
15. Пищулин А.А., Карпова Е.А. Овариальная гиперандрогения и метаболический синдром. *РМЖ*, 2001. № 9. С. 5-10.
16. Приложение к № 10. С. 16-19. 5.Carmina E. Diagnosis of polycystic ovary syndrome: from NIH criteria to ESHRE-ASRM guidelines. *Minerva ginecologica*. 2004. № 56 (1). С. 1-6.
17. Чернуха Г. Е. Современные представления о синдроме поликистозных яичников. *Consilium medicum*, 2002. Т.
18. Шилин Д.Е. Синдром поликистозных яичников: Международный диагностический консенсус (2003 г.) и современная идеология терапии. *Consilium Medicum*, 2004. № 06(9). С. 6-11.
19. Шилин Д.Е. Синдром поликистозных яичников: роль инсулинорезистентности и ее коррекция. Петрозаводск: ИнтелТек, 2004. 53
20. Olimova, N. (2023). INFLAMMATORY DISEASES OF THE PELVIC ORGANS: MODERN ASPECTS OF TACTICS. *International Bulletin of Medical Sciences and Clinical Research*, 3(10), 97-103.
21. Ismatilloevna, O. N. (2023). The Role of Immunological Factors in the Pathogenesis of HIV Infection in Women of Reproductive Age with Genital Inflammatory Diseases. *EUROPEAN JOURNAL OF INNOVATION IN NONFORMAL EDUCATION*, 3(4), 99-101.
22. Олимова, Н. И. (2023). Анализ Соматического И Репродуктивного Анамнеза Женщин С Воспалительными Заболеваниями Органов Малого Таза На Фоне Вич-Инфекции. *AMALIY VA TIBBIYOT FANLARI ILMIIY JURNALI*, 2(12), 864-872.
23. Olimova, N. I. (2023). Analysis of Somatic and Reproductive History of Women with Inflammatory Diseases of the Pelvic Organs Due to Hiv Infection. *Central Asian Journal of Medical and Natural Science*, 4(6), 1418-1426.

24. Олимова, Н. И., Юлдашев, Н. Б., Норметов, Б. Н., & Жаббаров, Х. Р. ОЦЕНКА ИММУННОГО СТАТУСА У ЖЕНЩИН С ВОСПАЛИТЕЛЬНЫМИ ЗАБОЛЕВАНИЯМИ ПОЛОВЫХ ПУТЕЙ НА ФОНЕ ВИЧ-ИНФЕКЦИИ.