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OPTIMIZATION OF EARLY DIAGNOSIS AND TREATMENT OF RECURRENT POLYCYSTIC OVARIES

Halimova Ezoza Mirzokhidovna

Annotation*: Over the years of studying polycystic ovary syndrome, various treatments for polycystic ovary syndrome have been developed. However, this pathology still occupies a leading position among the causes of endocrine infertility. Therefore, the need for further improvement of existing and development of new optimal treatment methods remains urgent.*

Keywords: *polycystic ovary syndrome, menstrual cycle disorder, chronic anovulation, hyperandrogenism, insulinresistance, body mass index, quality of life, infertility, diagnosis, treatment.*

Polycystic ovary syndrome (PCOS) is a multi – factorial heterogeneous disease characterized by menstrual irregularities, chronic anovulation, hyperandrogeny, an increase in the size and morphological structure of the ovaries [12, 15, and 16]. The urgency of the PCOS problem is determined by the high prevalence of this pathology [5, 13]. The frequency of the syndrome in the population is 11-15% [14, 24], in the structure of an ovulatory infertility– 70-75% [3, 12], among the patients of gynecologists-endocrinologists-30%, in women with hirsutism-85% [24]. PCOS is a socially significant endocrine pathology that leads to infertility, more frequent development of depression in young patients and reduces the quality of life of these women [2, 9]. The etiology of the disease is currently not fully understood. The most important factors are the following:[6, 7, 8]: 1. Pathology of the course of pregnancy and / or childbirth in the patient's mother (threat of termination of pregnancy, gestosis, premature discharge of amniotic fluid, prolonged anhydrous period, weakness of labor, premature detachment of the normally located placenta, acute fetal asphyxia, chronic fetal hypoxia). 2. Acute or chronic infections suffered by a girl at various periods of life, especially at puberty: diseases of the oropharynx and nasopharynx (frequent sore throats, chronic tonsillitis, measles, rubella, tuberculosis, viral hepatitis A), neuroinfections, and intoxication occupy the leading place. 3. Traumatic brain injuries (concussions, contusions, brain contusions). 4. Psychoemotional stress, stress, mental trauma, increased training loads and chronic information stress. 5. Chronic recurrent inflammatory diseases of the internal genital organs. 6. Congenital genetically determined defect of the ovarian enzymatic systems. 7. Hereditary factor. The pathogenetic basis of PCOS is chronic anovulation, which is caused by an overproduction of androgens and an increase in their conversion to estrogens, primarily in adipose tissue and liver. An excess of estrogens leads to a violation of the cyclicity and ratio of pituitary gonadotropins, which also supports anovulation [6]. The increased effect of luteinizing hormone (LH) on the ovaries causes hyperplasia of the internal thecatissue of the follicles, which contributes to an even greater increase in androgen production. A relatively low level of follicle-stimulating hormone (FSH) leads to a decrease in the activity of FSH-dependent aromatase, and granulosa cells lose their ability to convert androgens into estrogens [9]. High intra-ovarian the concentration of androgens interferes with the normal growth of follicles and contributes to the formation of their cystic atresia and thickening of the ovarian capsule. The process of follicle atresia occurs against the background of granulosa atrophy, which further inhibits FSH secretion. The increased pool of androgens in

peripheral tissues is converted to estrone and, thus, the vicious circle is closed [8].

Numerous studies have shown that PCOS is a diagnosis of exclusion [9,]. Leading experts of the special International Symposium of the joint working group ESHRE/ ASRM (European Society of Human Reproduction and Embryology and American Society for Reproductive Medicine), held in Rotterdam (Netherlands) in 2003 [29], came to a consensus on the need for mandatory testing of a number of hormonal parameters (17-hydroxyprogesterone, thyroid-stimulating hormone, prolactin, cortisolnormal concentrations of which can exclude other similar diseases associated with polycystic ovaries in patients with two of the three PCOS criteria. Determination of 17-hydroxyprogesterone is necessary to exclude adrenogenital syndrome [5], thyroid-stimulating hormone-hypo- or hyperthyroidism, prolactin – hyperprolactinemia, although in many patients its level may be at the upper limit of normal or slightly higher than normal, which is explained by an increase in the level of metabolic estrogens [4], cortisol-Itsenko-Cushing's disease [8]. Criteria for PCOS [7]: 1) Menstrual dysfunction with anovulation. Disorders of menstrual function are presented in the form of oligomenorrhea or primary / secondary amenorrhea, which may alternate with metrorrhagia. An indirect sign of anovulation is amenorrhea or oligomenorrhea (up to 8 menstrual cycles per year) [4]. Along with a violation of the menstrual cycle, the pathology of the reproductive system is a constant symptom of this endocrine disorder, mainly primary infertility in 71-98% [3]. 2) Clinical and / or biochemical signs of hyperandrogenism: - the level of testosterone is normal or slightly elevated; - the level of steroid-binding globulins (SSG) is at the lower limit of normal or reduced; - the index of free testosterone is increased [30]; - the level of estradiol is within the normal range or slightly elevated; progesterone in the absence of corpus luteum corresponds to early follicular - the level of dehydroepiandrosterone sulfate and 17-hydroxyprogesterone is normal or slightly elevated; - manifestations of androgen -dependent dermopathy: hirsutism, acne, seborrhea, alopecia and other symptoms of hyperandrogenism. Hirsutism, assessed on the Ferriman and Hollwayscale, occurs in 45-60% of patients with PCOS; – primary or secondary infertility of anovulatory origin; – obesity in 40-50% of patients [1]. 3) The presence of polycystic ovaries according to an ultrasound study [8]. Ultrasound semiotics of PCN (there must be at least one of the following signs): - detection of 8-12 follicles or more with a diameter of 2-10 mm; - increase in ovarian volume > 9-10 cm3; - detection of an enlarged ovarian stroma occupying more than 25% of the ovarian volume [3]. In the presence of a follicle more than 10 mm or a yellow body, ultrasound examination (ultrasound) should be repeated on the 3rd-5th day of the menstrual cycle with regular menstruation, with opsomenorrhea or amenorrhea – on a random day or 3-5 days after the onset of progestogen-induced bleeding. Preference is given to transvaginal ultrasound examination. Calculation of ovarian volume is performed using the formula: V = 0.5 H m H n H k, where V is the volume of the ovary (cm3); m is the length of the ovary (cm); n is the width of the ovary (cm); k is the thickness of the ovary (cm). Follicles are counted in the longitudinal, transverse, and anteroposterior sections of the ovaries. It is necessary to describe the distribution of follicles, stroma echogenicity, and ovarian volume during ultrasound examination [1]. Two types of polycystic ovaries are distinguished by ultrasound examination [8]: type I is a diffuse location of follicles in relation to the stroma; type II is a peripheral location of follicles in relation to the stroma. Ultrasound is a non-invasive and highly informative method [15]. Most authors currently believe that the diagnosis of PCOS can be made as accurately as possible based on the criteria described above, which in the future largely determines the effectiveness of therapy, especially in patients with menstrual disorders and infertility [12]. There are laparoscopic and morphological signs of PCOS. Laparoscopic signs of PCOS include [3]: - an increase in the size of theovaries in 91% of cases is bilateral

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