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IVF efficiency in different phenotypes of polycystic ovary syndrome

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Polycystic ovary syndrome occupies a leading place in the structure of endocrine infertility. This article presents the endocrine and metabolic features of the polycystic ovary syndrome phenotypes, as well as modern concepts of efficiency and complications of the use of assisted reproductive technologies, depending on the specific phenotype. The issues of polycystic ovary syndrome influence on selecting the method of assisted reproductive technologies, as well as possible complications that occur during *in vitro* fertilization and the features of the pregnancy course remain unresolved. The individualization of the approach seems to be promising when taking into account the differences in the hormonal profile and the features of metabolic disorders in each polycystic ovary syndrome phenotype. That may allow us to take one more step towards improving the effectiveness of *in vitro* fertilization and reducing the frequency of complications in patients with polycystic ovary syndrome.

Keywords: polycystic ovary syndrome; phenotype; insulin resistance; hyperandrogenemia; obesity; gestational diabetes mellitus; miscarriage; *in vitro* fertilization; ovarian hyperstimulation syndrome; efficiency.

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Эффективность экстракорпорального оплодотворения при различных фенотипах синдрома поликистозных яичников

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В структуре эндокринного бесплодия синдром поликистозных яичников занимает лидирующее место. В статье рассмотрены эндокринные и метаболические особенности фенотипов синдрома поликистозных яичников, современные представления об эффективности и осложнениях применения вспомогательных репродуктивных технологий в зависимости от конкретного фенотипа. Вопросы влияния синдрома поликистозных яичников на выбор метода вспомогательных репродуктивных технологий, а также возможных осложнений, возникающих в ходе процедуры экстракорпорального оплодотворения, особенностей течения наступившей беременности остаются нерешенными. Перспективным видится индивидуализация подхода с учетом различий в гормональном профиле и особенностей метаболических нарушений при каждом фенотипе синдрома поликистозных яичников, что, возможно, позволит сделать еще один шаг на пути к повышению эффективности экстракорпорального оплодотворения и снижению частоты осложнений.

Ключевые слова: синдром поликистозных яичников; фенотип; инсулинорезистентность; гиперандрогенемия; ожирение; гестационный сахарный диабет; невынашивание беременности; экстракорпоральное оплодотворение; синдром гиперстимуляции яичников; эффективность.

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Polycystic ovary syndrome (PCOS) is a widespread polygenic multifactorial disease that combines several characteristic symptoms and is manifested by impaired reproductive function and metabolic disorders [1].

Today, PCOS is the most common gynecological pathology among women of reproductive age, which leads to endocrine infertility [2]. The incidence of PCOS varies widely from 4% to 13% [3, 4]. In 2016, results of a systematic review and meta-analysis were published on the scientific portals Pubmed and OvidSP. Several thousand women were examined. The overall PCOS prevalence (95% confidence interval) according to the diagnostic criteria of the National Institutes of Health (NIH), Rotterdam Consensus, and Androgen Excess and Polycystic Ovary Syndrome Society (AE-PCOS) was 6% (5%–8%, *n* = 18 studies), 10% (8%–13%, *n* = 15 studies), and 10% (7%–13%, *n* = 15 studies), respectively. The incidence of PCOS according to the Rotterdam Consensus criteria can reach up to 21% [5, 6].

The most common clinical manifestations of PCOS include hyperandrogenemia (HA), oligo- and anovulation, as well as polycystic ovarian morphology on ultrasound examination (US) (ESHRE, 2018). In PCOS, pathological conditions, such as insulin resistance (IR), type 2 diabetes mellitus, ischemic heart disease, atherogenic hyperlipidemia, cerebrovascular insufficiency, anxiety and depressive disorders, endometrial hyperplastic processes, and ovarian and endometrial cancer, are common [1, 2, 5, 7–12].

Patients with PCOS had HA in 72.1%–82.0% of cases. Its main clinical manifestation in such patients is androgen-dependent dermopathy, often in the form of hirsutism, acne, seborrhea, and, much less often, acanthosis nigricans and alopecia. Defeminization (reduction of mammary glands, changes in skeletal architectonics, and hypoplasia of the external genital organs) and masculinization (male-type fat deposition, baryphony, and increased muscle mass) are not characteristic symptoms of PCOS but can be present with untimely correction of HA [3, 13]. A meta-analysis by Gurkan Bozdag et al. (2016) revealed that in PCOS, the incidence of HA is on average of 13% (8%–20%, *n* = 14), hyperinsulinemia (HI) is 11% (8%–15%, *n* = 9), polycystic ovarian morphology is 28% (22%–35%, *n* = 12), and oligo- or anovulation in 15% (12%–18%, *n* = 9) [6].

In 1990, the United States of America NIH held a conference, which resulted in the adoption of standardized criteria for PCOS diagnosis, including menstrual irregularities due to anovulation and clinical or biochemical signs of HA, without taking into account the morphological changes in the ovaries. Later in Rotterdam, a group of experts from the American Society for Reproductive Medicine and the European Society of Human Reproduction and Embryology (ESHRE/ASRM, 2003) supplemented the diagnostic criteria for PCOS with US signs of polycystic ovarian morphology [14, 15].

As an alternative, the AE-PCOS (2006) proposed to consider two aspects for the syndrome diagnosis, namely HA clinical or biochemical and ovarian dysfunction (oligo- or anovulation and/or polycystic morphology according to US data). Table 1 presents a comparative description of the criteria used for PCOS diagnosis.

Multicenter randomized trials demonstrated the polymorphism of the clinical manifestations of PCOS. It turned out that this pathology covers a wide range of signs and their combinations, significantly exceeding the initial criteria for establishing the diagnosis. The approaches to diagnose PCOS that existed at that time (NIH, 1990) were revised, and since 2012, the ESHRE/ASRM criteria (2003) with the obligatory indication of the syndrome phenotype are considered correct. Due to the adopted additions, PCOS can be diagnosed in women with both polycystic ovarian morphology and HA, and menstrual irregularities in combination with polycystic ovarian morphology [1, 5, 16]. In a multifaceted examination of patients with PCOS, differential diagnostics and exclusion of other etiological causes of menstrual irregularities and HA are important, which is also indicated in all the above-mentioned consensuses [16].

The structure of PCOS generally distinguished four phenotypes. Their comparative characteristics, depending on the signs, are presented in Table 2.

HA, overweight, and severe menstrual irregularities in PCOS can be assumed as independent predictors of metabolic disorders [17, 18]. Thus, R. Azziz (2018) noted that the severity of menstrual irregularities correlates directly with the IR level [1].

Research results indicate that patients with PCOS with phenotypes A and B have more pronounced menstrual

Table 1. Criteria to diagnose polycystic ovary syndrome

National Institutes of Health, 1990	Rotterdam Consensus, 2003	Androgen Excess and Polycystic Ovary Syndrome Society, 2006
• Chronic anovulation. • Clinical and/or biochemical hyperandrogenemia	• Oligo and/or anovulation. • Clinical and/or biochemical signs of hyperandrogenemia. • Polycystic ovarian morphology	• Clinical and/or biochemical signs of hyperandrogenemia. • Ovarian dysfunction (oligo-, anovulation, and/or polycystic ovarian morphology)
The presence of all criteria is obligatory	The presence of two of the three criteria are mandatory	The presence of all criteria is obligatory

Note. PCOS — polycystic ovary syndrome.

Table 2. Comparative characteristics of polycystic ovary syndrome phenotypes

Phenotype variants		Phenotype A (classic)	Phenotype B (anovulatory)	Phenotype C (ovulatory)	Phenotype D (nonandrogenic)
Incidence		44–65%	8–33%	3–29%	18.5–23%
Signs	Clinical/biochemical hyperandrogenemia	Present	Present	Present	Absent
	Oligo- and/or anovulation	Present	Present	Absent	Present
	Polycystic ovarian morphology	Present	Absent	Present	Present

irregularities, they are characterized by HI and IR, and they develop metabolic syndrome 2 times more often compared to patients with phenotype D [19–26]. Excess body weight is more typical for women with androgenic phenotypes of PCOS, and it occurs in 53.1% of cases with the classic phenotype, 33.3% with anovulatory phenotype, and 30.0% with nonandrogenic phenotype [19, 22]. The frequency of obesity reaches 86.0% in patients with phenotype A, 27.9% with phenotype B, 46.6% with phenotype C, and 38.8% with phenotype D. The most pronounced forms of atherogenic hyperlipidemia were registered in patients with phenotype A (>65.9%); this phenotype is also characterized by hypercholesterolemia and hypoalphaproteinemia [19]. A group of Italian researchers showed that the ovulatory phenotype is more typical for patients with PCOS with high socioeconomic status [27]. This dependence can probably be due to differences in insulin levels and body mass index values [27]. However, other researchers did not register any significant differences in the incidence of high body mass index, IR, and dyslipidemia in different PCOS phenotypes. C.N. Wijeyaratne et al. (2011) and A.S. Melo et al. (2010) revealed no differences in the prevalence of metabolic syndrome in women with different PCOS phenotypes [28, 29].

The prevalence of impaired glucose tolerance in patients with androgenic phenotypes of the syndrome is 62.5% in phenotype A, 55.6% in phenotype B, and 20.0% in phenotype C. Nonandrogenic phenotype D, on the contrary, is favorable for the prognosis of impaired carbohydrate metabolism, as impaired glucose tolerance is almost not revealed in this category of patients, HI and IR are registered in 8% and 12%, respectively. The percentage of these indicators with classical (14.1%, 27.1%, and 30.6%, respectively), anovulatory (9.1%, 9.1%, and 9.1%, respectively), and ovulatory (14.3%, 28.6%, and 35.7%, respectively) phenotypes confirm this conclusion. The comparison of indicators of carbohydrate metabolism in all phenotypes of PCOS found no significant differences, but an increased level of immunoreactive fasting insulin by 28.5%–34.3% in the androgenic phenotypes, especially in the classical one. In the phenotype D group, the blood level of immunoreactive insulin increased by 14.3% [24, 26].

The data presented that patients with PCOS with phenotypes A and B have an increased risk of hepatic steatosis compared with patients with nonandrogenic PCOS phenotypes and healthy women [30].

The assessment of the hormonal spectrum revealed that HA in phenotypes A and B develops primarily due to increased levels of dehydroepiandrosterone sulfate and free testosterone. Every third patient has a decreased level of testosterone-estrogen-binding globulin (TEBG), thus an increased free androgen index in all phenotypes. In ovulatory phenotype C, a lower blood level of free testosterone was found compared to other androgenic phenotypes. Ovarian HA is believed to adversely affect the quality of oocytes and embryos and the success of their development [31].

A. Jamil et al. (2016) revealed a significantly higher ratio of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) is in phenotype A than in phenotype D and women without PCOS. The level of total testosterone in serum is higher in phenotype A compared to patients with other PCOS phenotypes, as well as healthy women [32]. The level of anti-Müllerian hormone (AMH) in the blood serum of patients with PCOS exceeds 8.7 ng/ml [33]. The highest blood AMH level is determined in phenotype A. In addition, studies were conducted, in which no significant differences were obtained in the blood levels of FSH, AMH, prolactin, estradiol, dihydrotestosterone, 17α-hydroxyprogesterone, and progesterone in patients with PCOS having anovulatory phenotypes [3, 33].

Most studies found that endocrine and metabolic disorders are insignificant in patients with nonandrogenic phenotype D in PCOS and, accordingly, the incidence of metabolic syndrome is low [26, 34, 35]. In these women, compared with women with the classic PCOS phenotype, the LH/FSH ratio is almost unchanged, a lower level of total and free testosterone in the blood, as well as a lower value of free androgen index, and a higher level of testosterone estrogen-binding globulin [32]. In phenotype D, opsomenorrhea alternates with regular menstrual cycles more often than in other PCOS phenotypes [36].

In general, the published data indicate that more than half of patients with PCOS have phenotype A, whereas the other three phenotypes (B, C, D) are almost equally common. Patients with phenotypes A and B account for approximately two-thirds of the total number of patients with PCOS [37].

Understanding the distribution of PCOS phenotypes is of great importance to determine the syndrome epidemiology in the population, as well as to apply a personalized approach to the treatment and management of patients with this syndrome in *in vitro* fertilization (IVF) protocols, which aimed to reduce the risk of complications and increase the effectiveness of the procedure.

One of the major problems associated with PCOS is the loss of fertility in these patients. PCOS causes endocrine infertility in 55%–91% of cases [38]. In addition, up to 70% of PCOS cases remain unverified [39]. Due to the rapid improvement of assisted reproductive technologies, IVF is the most effective method to implement reproductive function [38]. Despite alternative approaches when choosing a protocol, in clinical practice, patients with PCOS have several problems, such as immature oocytes, ovarian hyperstimulation syndrome (OHSS), and increased risk of obstetric and neonatal complications [40]. The effectiveness of PCOS in the increased frequency and severity of pregnancy complications after IVF remains disputable and controversial.

The efficiency of IVF can be assessed by considering several indicators, namely the number and quality (maturity) of aspirated oocytes, the number of obtained embryos, the frequency of pregnancy, and early gestation, as well as the frequency of spontaneous abortions.

Ovary stimulation of patients with PCOS is challenging because the ovaries tend to overreact. Several systematic reviews and meta-analyses showed an increased risk of OHSS in patients with PCOS. Both mild and severe forms of OHSS were identified [41, 42]. This complication is assumed to be associated with both the selected IVF protocol and the PCOS phenotype [43]. Different ovarian responses were described depending on the PCOS phenotype, thus phenotype A is associated with a higher risk of OHSS, and phenotype B is associated with a lower risk of this complication [42]. The study by T. Sha et al. (2019) revealed that when a fresh embryo is transferred into the uterine cavity of patients with PCOS, OHSS develops more often [38].

L.S. Tselkovich et al. (2017) revealed a negative effect of metabolic syndrome on the results of IVF in PCOS. Thus, the total number of high-quality embryos obtained from patients with PCOS without metabolic disorders was 72.5%, which decreased to 61.4% in the presence of metabolic disorders, whereas 50.7% and 30.0% were suitable for cryopreservation, respectively [43]. In their study, V. Cela et al. (2017) found no statistically significant differences in the number and quality of transferred and frozen embryos. Nevertheless, a slightly larger number of embryos from the patient with PCOS having phenotype A were obtained and frozen [42]. In groups with phenotypes characterized by polycystic ovarian morphology, the highest incidence of implantation, biochemical, and clinical pregnancy is registered in comparison with phenotype B [44]. The percentage of mature MII oocytes in the stimulation cycles using gonadotropin-releasing hormone agonists as an ovulation trigger was 65.0%, whereas when using human chorionic gonadotropin, this indicator did not exceed 49.9% [42]. In the presence of metabolic disorders in PCOS, the frequency of hospitalizations after IVF increased threefold, reaching 70.6% of cases [43].

The frequency of pregnancy in the programs of assisted reproductive technologies in patients with PCOS and without it did not differ [38]. In addition, patients with PCOS without metabolic disorders showed a significantly higher pregnancy rate (50.7%) than women with such disorders (33.3%) [43]. A correlation was established between the presence of phenotype B and the absence of pregnancy after embryo transfer [42]. A higher incidence of carrying a pregnancy in patients with this syndrome was found compared with other causes of infertility [38]. However, M. De Vos et al. (2018) noted a lower rate of carrying a pregnancy in hyperandrogenic phenotypes A and C (16.7% and 18.5%, respectively) compared with phenotype D (33.7%) [45]. The incidence of spontaneous abortion in PCOS in combination with a tubal factor of infertility reached 64.7%, whereas no significant difference with the control group of healthy women was found without this burdening factor [38]. PCOS, including metabolic disorders, is the cause of pregnancy termination in 34.6% of cases, in the absence of metabolic disorders does not exceed 5.9% [43]. No differences were found in the frequency of ectopic pregnancy in healthy women and women with the described syndrome [38].

The incidence of isthmic-cervical insufficiency and premature birth in patients with PCOS reaches up to 28%. One of the probable pathogenetic grounds for such outcomes may be increased levels of proinflammatory cytokines (tumor necrosis factor-α and interleukin-1 and -6) that maintain chronic inflammation. One-third of all cases of miscarriages occur in the second trimester [40].

Earlier studies indicate that polycystic ovarian transformation, IR, and HA can be potential causes of gestational diabetes mellitus, preeclampsia, and arterial hypertension [40]. Based on a 2019 meta-analysis by T. Sha et al., a relationship was found between the low TEBG concentration and an increased risk of gestational diabetes mellitus in female patients with PCOS [38]. According to L.E. Kjerulff et al., a low concentration of insulin-like growth factor-binding globulin-1 may contribute to the development of arterial hypertension [46].

The authors of a 2015 cohort retrospective study compared the prevalence of obstetric complications, including gestational diabetes mellitus, gestational hypertension, proteinuria, and fetal growth retardation syndrome in women diagnosed with PCOS, isolated polycystic morphology, and with other causes of infertility, who entered the IVF protocols. No statistically significant difference was found in all groups [47].

Granulosa cell-produced AMH represents a marker of functional ovarian reserve, also called a small growing follicular pool, which ultimately determines the quantity and quality of oocytes/embryos obtained by IVF. AMH is a reliable marker of ovarian response to controlled ovarian stimulation. The basal level of AMH in the blood serum correlates closely with the US data of the antral follicle count [48].

The classical phenotype A is characterized not only by a higher level of estrogens, the number of 8–12 mm follicles, but also by a high level of AMH. According to some data, the level of AMH in the blood in patients with phenotype A is a threefold increase compared to patients with phenotype B. Serum AMH concentration is a more reliable marker to predict the ovarian response and the probability of OHSS than the patient's age and body mass index [49]. Thus, prior knowledge of high AMH levels allows planning strategies to reduce the risk of OHSS. In addition, a retrospective study by G. Bozdag et al. (2019) demonstrated a low significance of AMH determination in diagnosing three PCOS phenotypes (B, C, D), except for the classic A phenotype [53].

Metformin showed to help restore ovulation through a variety of mechanisms [50]. These include increased insulin sensitivity, direct inhibition of androgen-producing ovarian enzymes, and decreased secretion of vascular endothelial growth factor which plays a key role in OHSS pathophysiology [51]. In the group of patients with PCOS who were treated for infertility using IVF and metformin, an increased frequency of pregnancy and decreased incidence of OHSS was observed, and at the same time, no significant differences in the number of obtained oocytes, days of stimulation, or frequency of cycle cancellation, and the incidence of pregnancy and miscarriages [51].

In 2016, a group of American researchers described in sufficient detail a previously non-highlighted PCOS-like phenotype characterized by a high level of AMH, but with an atypically low level of testosterone. This pathology affects young women without obesity and is associated with low levels of dehydroepiandrosterone sulfate. The low testosterone level, in this case, is probably of adrenal origin and arises from autoimmune adrenal insufficiency, since it is accompanied by low cortisol levels. The authors note that supplementation of dehydroepiandrosterone in these patients normalizes the androgen levels and improves the outcomes of the IVF cycle [33, 52].

CONCLUSION

To date, the problem of PCOS is widely covered in the literature, and questions of pathogenesis, clinical manifestations, diagnostics, and treatment of the pathology are discussed in numerous works. The ongoing research to date will possibly expand the phenotypic range of PCOS.

However, an analysis of the literature data presented in our review enables us to conclude that most of the studies published on the aspects of the treatment of fertility disorders in patients with PCOS, particularly the use of assisted reproductive technologies, show conflicting results, which indicates an insufficient degree of familiarization of the problem and a small number of studies.

More recently, research started to develop a personalized approach to the management of patients with PCOS, depending on the phenotype. This will increase the efficiency of diagnosis and treatment of the disease itself, as well as predict the risks of failure and increase the chances of a successful pregnancy, including with the use of IVF, as well as carrying and gestational outcomes.

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