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POLYCYSTIC OVARY SYNDROME AND ENDOTHELIAL FUNCTION

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Annotation*: Modern medicine is characterized by the process of updating ideas about the factors of formation of polycystic ovary syndrome (PCOS), even those that were previously considered insignificant from a clinical point of view. In this regard, in recent years, more and more attention has been paid to the study of the role of endothelial function in the formation of pathological processes in the reproductive system in young patients. The endothelium, according to the classical definition, is a single-layered layer of specialized cells that lines the blood, lymphatic vessels and heart cavities from the inside [1].*

Keywords: *polycysticfibrosis, endothelium, syndrome.*

The most studied are the structure and function of the vascular endothelium. According to modern concepts, the endothelium is not just a semipermeable membrane, but an active endocrine organ, the largest in the body, diffusely dispersed throughout all tissues [6]. The first reports on the features of the structure and function of the endothelium appeared in 1985, when Hiskey et al. [8] showed that the vascular endothelium secretes a substance (endothelin) that has the properties of a vasoconstrictor with a long vasoconstrictor effect. There are three forms of endothelin. In addition to the main form of endothelin-1 (ET-1), there are two isoforms: ET-2 and ET-3 [13]. ET-1 is the main isoform of the human cardiovascular system. The role of isoforms other than those involved in embryogenesis remains unclear. The main amount of ET-1 is produced in the vascular endothelium and makes the greatest contribution to the activity of the endothelial system [9]. ET-1 acts as a paracrine and autocrine mediator, rather than as a circulating endocrine hormone, so its plasma concentration may not fully reflect its physiological action. It is important to take into account the fact that the plasma concentration of ET-1 depends not only on its production, but also on renal clearance and enzymatic metabolism. Determination of ET-1 plasma concentration can be useful as a marker of endothelial synthesis [4]. Under physiological conditions, ET-1 is formed in small amounts. The damaged endothelium synthesizes a large amount of ET-1, which causes vasoconstriction [5]. With prolonged endothelial damage, it begins to play a key role in the pathogenesis of a number of systemic pathologies [3]. It is suggested that an increase in the concentration of endothelin in the blood serum can be used as markers of pathological processes in the human body. ET-1 has recently been found to increase in patients with PCOS [11]. Patients with PCOS are characterized by disorders offolliculogenesis and steroidogenesis, which determine the formation, selection and development of the dominant follicle and lead to anovulation. Violation of the development of the dominant follicle depends on the state of blood supply to the follicle at each individual stage, on the quality of the endothelium and the balance of endothelial factors that control vascular tone. A defect in the selection mechanism of the dominant follicle leads to the accumulation of small antral cells. Follicles that have a significant contribution to the formation of PCOS [10]. In the literature, there are data on the content of ET-1 in the follicular fluid, and its content in various forms of infertility is considered. However, the role of ET-1 in the pathogenesis of PCOS is still poorly understood. Some authors suggest that the existing hormonal changes are involved in the formation of endothelial dysfunction in women with PCOS [17, 18], and the degree of endothelial damage undoubtedly plays an important role in the pathogenesis of the disease and,

possibly, affects the development, course and prognosis of PCOS [14]. Since the question of the presence and clinical significance of endothelial dysfunction in PCOS is debatable, it is advisable to conduct studies aimed at assessing the role of hormonal factors in the genesis of endothelial dysfunction. To study the content of endothelin-1 in the blood serum of young women with PCOS and determine the presence or absence of a relationship with hormonal parameters. Materials and methods:. The analysis included an assessment of the normality of the distribution of the studied variables using the Kolmogorov-Smirnov test, and the main statistical parameters of the series were calculated: Me, Q25, and Q75. The revealed hormonal disorders once again confirm their significance as the leading endocrine characteristics of PCOS, and their role in the pathogenesis of the disease [9]. In the course of studying the marker of endothelial functional activity-ET-1, it was found that its content in the blood serum of young women with PCOS significantly exceeded ($p<0.05$) the indicators of healthy women (median 0.83 fmol / ml, Q25-0.64 фмofmol / ml, Q75-1.02 fmol / ml).The correlation analysis made it possible to establish the presence of a direct correlation between ET-1 and LH ($r = 0.33$; $p<0.05$), the T/E2 coefficient ($r = 0.41$; $p<0.01$), and an inverse correlation with E2 ($r = - 0.39$; $p <0.02$). Therefore, the formation of endothelial dysfunction in young people is not possible. In women with PCOS, it is caused not only by metabolic disorders, with which it is often associated, but also by hormonal disorders, manifested by an increase in androgen levels and a decrease in estrogen secretion. However, the mechanism by which hyperandrogenism can affect vascular response is not yet known. Androgen receptors may exist in the vascular wall, and a direct effect of androgens on the vasculature is not excluded [15]. Until oday, it is believed that the main feature of PCOS formation is impaired folliculogenesis, in which the lack of development of the dominant follicle leads to anovulation and cystic degeneration of the ovaries. Follicle growth from the primordial stage to ovulation of the dominant follicle remains the most important part of research in human reproduction. The parameters of folliculogenesis were monitored under ultrasound observation. It was found that the volume of ovaries in patients with PCOS significantly exceeded that of healthy women (13.5±0.3 cm3, 12.9±0.4 cm3, 6.2±0.2 cm3, respectively; $p<0.001$). Individual analysis showed that the ovarian volume of more than 10 cm3 occurred in 28 (70%) patients with PCOS, while the remaining 12 (30%) had less than 10 cm3. The number of antral follicles in patients with PCOS was also significantly higher than in healthy women (6.4±0.9; $p<0.001$). The average follicle diameter was 6.2±0.3 mm. The data obtained indicate the presence of cystic ovarian degeneration in all patients with PCOS. The study of factors that affect folliculogenesis, can prevent ovulation and lead to cystic degeneration of the ovaries is of paramount importance today [12]. Recently, AMH, which is secreted by growing follicles, can be tested in blood serum, is a marker of ovarian functional activity and a diagnostic criterion for the preservation of the follicular apparatus, and has been considered as one of the diagnostic criteria for PCOS [7]. In patients with PCOS, the AMH level was 4.5 times higher than in the control group. Despite the fact that some authors talk about the influence of the endothelial system on the process of human folliculogenesis and point out the presence of a link between endothelial dysfunction and AMH secretion [16], we did not find a statistically significant link between AMH and ET-1 ($r = 0.19$, $p>0.05$). The reason why PCOS patients with PCOS are not affected by the presence of endothelial dysfunction and AMH the follicles remain at the pre-dominant stage, still unknown. The study of the endothelial system as one of the links of closely interrelated pathogenetic mechanisms of PCOS formation opens up new prospects in the development of diagnostic and treatment methods for this pathology. Conclusions. In young women with PCOS, there is an increase in serum endothelin-1, which positively correlates with the level of luteonizing hormone, the testosterone-estradiol coefficient, and is inversely related to the level of estradiol.

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