

Modern Directions in the Study of the Etiology and Pathogenesis of Endometriosis

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Summary: Endometriosis is one of the most common gynecological diseases, which reflects the medical and social significance of the problem of effective diagnosis and treatment. To date, the causes, diagnosis and treatment of this disease remain the subject of controversy. The pathogenesis of the disease is multifactorial and has not been studied enough, non-invasive examination methods have a relative diagnostic value, so modern therapeutic approaches often do not provide a complete cure.

Keywords: endometriosis, diagnostics, proteins, genes, mikro RNK.

Relevance. Endometriosis is a tumor pathological process in which benign tissue growth occurs outside the uterine cavity, similar in morphological and functional properties to the endometrium. In the structure of gynecological diseases, endometriosis is in third place, following inflammatory diseases of the uterine appendages and fibroids. If at the beginning of the twentieth century. S. Menge and E. Ortiz in the “Manual of Gynecology” (1914) wrote that “...genital endometriosis is not of particular practical importance,” then already at the end of the century, due to the widespread increase in the frequency of this pathology, endometriosis began to be considered as a new disease of civilization. Currently, endometriosis is reasonably recognized as the most common and severe disease of women of reproductive age, negatively affecting the general condition, performance and quality of life of patients [1–4]. According to population studies of the World Endometriosis Research Foundation (WERF), currently more than 176 million women in the world suffer from endometriosis (up to 10% of women of reproductive age) [5], and the economic costs of its treatment, rehabilitation and compensation for disability amount to more than \$76 billion/year. Moreover, the costs associated with compensation for loss of ability to work are 2 times higher than the costs directly for medical care. Clinical manifestations of endometriosis depend on the localization of the process, the degree of damage to the genitals and adjacent organs, and the individual pain threshold. Painless (asymptomatic) forms of endometriosis occur even with severe infiltrative lesions, but this is always the exception. The “calling card” of endometriosis is symptoms directly related to menstruation and most pronounced during this period: dysmenorrhea (mainly algomenorrhea) - 82.7–83.4%; pain syndrome of varying severity - 48.3–50%; dyspareunia - 33.4–34.5%. Bleeding with endometriosis is also predominantly cyclical in nature - as a rule, hyper- and polymenorrhea are noted, and prolonged perimenstrual bleeding and anemia are also characteristic. Dysuria (urinary disorders) and dyschezia (painful and/or difficult defecation) are observed with infiltrative lesions of adjacent organs (bladder and/or ureters and intestines, respectively). The literature describes the “four dis”

syndrome (dysmenorrhea, dyspareunia, dysuria, dyschezia), observed during menstruation in patients with endometriosis. Infertility is one of the most significant (including socially) and painful symptoms of endometriosis; its frequency is 35–40%. Thus, the fertility rate (the ratio of the number of childbirths to the number of women of reproductive age) in healthy women is 0.15–0.20, in patients with endometriosis – 0.02–0.1, i.e. it is an order of magnitude lower [6].

Endometriosis is the only disease in which a benign proliferative process affects the unchanged tissue of neighboring organs. The characteristic features of endometriosis are the ability for infiltrative growth, the absence of a pronounced capsule around the endometrioid lesion (with the exception of endometrioid cysts), and the possibility of metastasis to distant organs. The severity of the disease, the ambiguity of tactical approaches and the lack of pathogenetic treatment methods have predetermined the interest of many domestic and world researchers in a comprehensive study of this disease, however, despite numerous studies, the results remain unsatisfactory: 35–50% of patients with endometriosis suffer from pain or infertility, relapses are observed in almost half (40–45%) of patients during the first 5 years after surgical treatment [7]. Although several pathogenetic hypotheses have been proposed, the exact pathogenesis of endometriosis remains unclear. At the same time, it is obvious that endometriosis develops as a result of dissemination of the endometrium into ectopic areas with the subsequent formation of ectopic endometrial complexes [1]. It is the presence of these ectopic lesions that is thought to give rise to the symptoms associated with the disease.

Ectopic tissue of endometrial origin consists of glandular epithelium and stroma and is characterized by a predominance of benign characteristics combined with features (invasion and neoangiogenesis) that make it similar to malignant neoplasms. Many theories have been proposed to explain the causes of the development of endometriosis [5], depending on the factors causing its development: retrograde menstruation, genetic predisposition, peritoneal cell metaplasia and impaired immunological control.

Sampson's theory is the most popular and generally accepted theory of the origin of endometriosis: it is assumed that endometrioid implants arise as a result of retrograde menstrual reflux of endometrial tissue into the abdominal cavity through the fallopian tubes, which is confirmed by the detection of endometrial fragments. The spontaneous development of endometriosis only in humans and primates supports the assumption that this disease can only affect menstruating species. At the same time, retrograde menstruation is probably a very common phenomenon, but endometriosis develops only in a part of women, which indirectly indicates the role of other factors in its development (immunological, genetic, disturbances in the biochemical composition of the environment in the abdominal cavity). In addition, the alternative embryonic germ theory suggests that endometriosis may develop from Müllerian cells scattered in the peritoneal cavity, which, when exposed to certain biochemical stimuli, can become activated and form endometrial tissue. Another hypothesis, which also helps explain the extraperitoneal localization of the disease, suggests that the origin of endometrioid lesions may be associated with metaplasia of peritoneal cells and differentiation of mesothelial cells into endometrial cells. Finally, it has been suggested that blood and lymphatic vessels may play a role in the dissemination of endometrial cells over long distances. The human endometrium contains a small population of cells that have functional stem cell properties with corresponding markers and functional characteristics, which has suggested a role in the pathogenesis of endometriosis. Under physiological conditions, endometrial stem cells undergo cyclical monthly regeneration after menstruation. These cells are presumably responsible for the proliferation and cyclic regeneration of endometrial tissue after menstruation. According to this hypothesis, endometriosis may result from the migration of endometrial stem

cells into the peritoneal cavity, their proliferation, peritoneal invasion, and differentiation into endometrial cells in ectopic locations.

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