



Epidemiology, Theories Of The Development, Conservative And Operative Treatment Of The Endometriosis.

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Abstract: According to modern ideas about the nature of endometriosis, this disease should be considered as a pathological process with a chronic, recurrent course. Endometriosis is formed and develops against the background of impaired immune, molecular genetic and hormonal relationships in the female body.

Key words: Adenomyosis, adenomyoma, epithelial metaplasia, endometrioid heterotopias, endometrioid fragments, intracrine interaction, ovariectomy, HLA system.

Endometriosis is an overgrowth similar in structure to the lining of the uterus, beyond the usual localization of the endometrium. The endometrioid substrate has signs of autonomous growth and disorders of cell proliferative activity. Endometriosis can be localized both in the body of the uterus (adenomyosis, or internal endometriosis) and outside the uterus (external endometriosis). The first morphological description of endometriosis in the medical literature was published by V.Rokitansky (1860), who called the pathological formation he found in a woman's pelvis an adenomyoma. The term "endometriosis" in clinical practice was proposed by J.Sampson in 1925. Regardless of the localization and size of endometrioid foci, histologically endometriosis is characterized by a benign proliferation of glandular epithelium resembling functioning endometrial stroma glands. However, the ratio of glandular epithelium and stroma in endometrioid heterotopias of different localization is not the same. In recent years, the opinion has been expressed that "internal endometriosis of the uterus" should be considered a completely independent disease, designating it with the term "adenomyosis", and not "endometriosis" (Haney A.F., 1991). It is emphasized that the clinical picture, diagnosis, prevention, treatment methods for adenomyosis have significant features. In addition, adenomyosis cannot occur as a result of "retrograde menstruation" through the fallopian tubes, according to the most recognized implantation theory. Adenomyosis develops from the basal layer of the endometrium, which takes into account the translocation hypothesis of the occurrence of uterine endometriosis. Certain successes in the study of certain aspects of pathogenesis, diagnosis and treatment of endometriosis of various localization allowed us to define by the term "endometriosis" only an anatomical substrate, and the disease (symptom complex) associated with this substrate is called endometrioid disease. However, in modern literature and practical medicine, the term "endometriosis" is widespread, and most researchers mean a nosological unit by endometriosis.

EPIDEMIOLOGY.



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In the structure of gynecological morbidity, endometriosis occupies the 3rd place after inflammatory processes and uterine fibroids, affecting up to 50% of women with preserved menstrual function. Endometriosis leads to functional and structural changes in the reproductive system, often negatively affecting the psycho-emotional state of women, significantly reducing the quality of life. Currently, many clinicians testify that endometrioid lesions occur at any age, regardless of ethnicity and socio-economic conditions. Epidemiological studies indicate that 90-99% of patients with endometrioid lesions are detected at the age of 20 to 50 years, and most often in the reproductive period.

THE MAIN THEORIES OF THE DEVELOPMENT OF ENDOMETRIOSIS.

The variety of localizations of endometriosis has led to a large number of hypotheses about its origin. A significant number of concepts are trying to explain the origin and development of this disease from various positions. The main statements are:

- the origin of the pathological substrate from the endometrium (implantation, lymphogenic, hematogenic, iatrogenic dissemination);
- epithelial metaplasia (peritoneum);
- violation of embryogenesis with abnormal residues;
- violation of hormonal homeostasis;
- changes in immune balance;
- features of intercellular interaction.

The iatrogenic moment of the disease development is sufficiently proven by a retrospective analysis of the etiology of endometriosis in women who underwent certain operations. Of considerable interest is the possibility of metastasis of endometriosis through blood and lymphatic vessels. This type of dissemination of endometrial particles is considered one of the most important causes of known variants of extragenital endometriosis, such as endometriosis of the lungs, skin, muscles. The spread of viable endometrial cells along the lymphatic pathways is not uncommon, as evidenced by the fairly frequent detection of significant foci of endometriosis in the lumen of lymphatic vessels and nodes. Researchers continue to search for the causes of implantation and further development of endometrial elements in pelvic tissues. Although retrograde flow of menstrual blood is probably a common occurrence, endometriosis does not develop in all women. In some cases, the prevalence of endometrioid lesions is minimal and the process may remain asymptomatic, in others endometriosis spreads throughout the pelvic cavity and causes various complaints. Moreover, in some cases of endometriosis, self-healing is possible, and in other cases the disease persistently recurs, despite intensive therapy. A number of authors believe that cases of "weak" endometriosis should not be considered a disease requiring special treatment. In their opinion, this is a physiological phenomenon associated with regular retrograde withdrawal of menstrual blood. However, it is unclear what is the boundary between this condition and endometriosis as a disease. These problems are currently in the center of study. It is obvious that, in addition to the general signs of immunodeficiency and autoimmunization, there are some other factors (perhaps a combination of them) that determine the perception of endometrial particles from the pelvic peritoneum, which creates conditions for the implantation of these particles, instead of identifying them as foreign and contributing to their destruction. Disorders of cellular and humoral immunity in endometriosis have been identified with HLA antigens. It can be



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assumed that endometriosis is hereditarily determined by genes associated with certain antigens of the HLA system, namely NA, A10, B5, B27.

Of course, it is impossible to explain the whole variety of clinical and morphological manifestations of endometriosis only by a primary genetically determined immune defect. The nature of local violations of tissue homeostasis directly in the pelvic region is also important. These processes attract the attention of researchers, and the analysis of the results constantly expands knowledge about the mechanisms of control of tissue proliferation, inflammatory and dystrophic reactions. A significant place is given to macrophages that directly react to the presence of foreign elements. Macrophages "move" red blood cells, damaged tissue fragments and, possibly, endometrial cells that enter the abdominal cavity. It was found that in endometriosis, the total number and activity of peritoneal macrophages increase. The dependence between the severity of the course of endometriosis and the macrophage reaction of the peritoneal fluid was noted, and an increase in the content of macrophages in the foci of endometriosis was also proved. At the present stage, the concept put forward by W.P. Damowski et al. is of interest. (1988), later somewhat modified by R.W. Shaw (1993):

- retrograde movement of endometrioid fragments during menstruation occurs in all women;
- rejection or implantation of these fragments depends on the function of the immune system;
- endometriosis reflects the insufficiency of the immune system, which is inherited;
- immune insufficiency can be both qualitative and quantitative, leading to endometriosis;
- the production of autoantibodies is a reaction to ectopic the endometrium and it, in turn, can contribute to infertility in endometriosis.

An increase in the concentration of prostaglandins in a woman's blood plasma predisposes to the formation of the disease, affecting the cytoproliferative activity and differentiation of endometrioid tissue cells. Prostaglandins may stimulate the growth of the endometrium, manifest the main clinical symptoms – dysmenorrhea and infertility. Prostaglandins and immunocomplexes are not the only physiological regulators of intercellular interaction. Other factors determining the fate of ectopic endometrial tissue are cytokines and growth factors. In addition to the cells of the immune system, other cells are able to secrete similar signaling molecules, which have come to be called cytokines. Cytokines are mediator peptides that promote cell interaction. Certain material has been accumulated on the role of cytokines providing favorable conditions for the introduction and development of viable elements of the endometrium. The biological potential of cytokines is to regulate the interaction of macrophages with tissue elements. It is known that different cell populations are able to secrete the same cytokines. Macrophages, B cells, and some subpopulations of T lymphocytes produce similar sets of cytokines. Obviously, activation of a certain group of cells leads to the synthesis of a set of cytokines and the induction of related functions. With endometriosis, the concentration of cytokines such as interleukin-1 and interleukin-6 increases in the peritoneal fluid, the main producers of which are macrophages. There was a correlation between the level of interleukin-1 and the stage of endometriosis spread. Cytokines accumulated during the local activation of macrophages close the feedback loop, which ensures the involvement of new mediators in the process. In addition, it is believed that interleukin-1 has a number of properties that may be associated with endometriosis. It also stimulates the proliferation of B cells and the induction of autoantibody formation. It has been established that along with sex hormones and cytokines, growth factors are important regulators of cell proliferation and differentiation.



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These factors are produced by non-specialized cells present in all tissues and have endocrine paracrine, autocrine and intracrine effects. Of particular interest from the point of view of the pathogenesis of endometriosis is one of the ways of action of growth factors, called intracrine interaction. At the same time, growth factors are not secreted and do not need surface receptors mediating their activity. They remain inside the cell and act directly as intracellular intermediaries, regulating cellular functions. There are epidermal, platelet, insulin-like and other growth factors. Growth factors together with cytokines, hormones, neurotransmitters are considered as the most important means of intercellular interaction. The release of growth factors complements the effect of other active agents, contributing not only to proliferation, but also to dystrophic changes in tissues. The process of accumulation of growth factors and cytokines is facilitated by the fact that they are also produced in tissue cells attacked by macrophages, primarily in epithelial cells, fibroblasts, etc. This seems to be very important for understanding the pathomechanisms of endometriosis, the spread of which is closely related to the proliferation of elements of heterotopias, the proliferation of connective tissue.

• PRINCIPLES OF TREATMENT OF ENDOMETRIOSIS.

Treatment of endometriosis is one of the most difficult problems in modern gynecology, which, despite a significant amount of research, remains unresolved. Both surgical and hormonal methods of endometriosis treatment are being actively studied, but their combined use remains ineffective. After the widespread introduction of endoscopy into gynecological practice, organ-sparing operations involving the removal of all endometrioid implants while preserving pelvic organs and childbearing function in young women have become one of the main therapeutic methods for endometriosis. A number of authors consider organ-preserving surgical interventions to be the first stage of treatment of endometriosis, others defend the uniqueness of this method of treatment, pointing out that subsequent suppressive hormone therapy is ineffective and may have an undesirable side effect. At the same time, the clinical effectiveness of surgical interventions remains insufficient, since the risk of recurrence of endometriosis and its clinical symptoms (pain syndrome) is quite high. Discussions continue about excision of retrocervical infiltrate by endoscopic access, radical interventions for endometrioid cysts localized in the left ovary (ovariectomy), in order to prevent the formation of adhesions between the sigmoid colon and the left appendages. We believe that this is due to the impossibility of carrying out high-quality endoscopic destruction of all foci of endometriosis, which is especially difficult with its widespread forms, as well as due to insufficient training of the surgical team. The study of the pathogenesis of endometriosis has made it possible to achieve significant success in the development of methods of drug therapy of the disease. A number of authors believe that hormone therapy should be the method of choice of treatment, and only in case of its failure recommend surgery. However, the effectiveness of hormonal treatment is assessed by many experts as very limited. The modern and optimal approach to the treatment of patients with endometriosis consists in a combination (combination) of the surgical method and suppressive hormone therapy. This point of view has many supporters.

- Thus, according to modern ideas about the pathogenesis of the disease, the mandatory stages of therapy for external genital endometriosis are:
 - surgical removal of endometrioid heterotopias as a factor initiating the functioning of a vicious circle and the progression of the pathological process;



- therapeutic effect aimed at stopping the entry of viable endometrial cells into the abdominal cavity for a time sufficient to normalize impaired intercellular interactions and immune status as the main mechanisms for the occurrence and development of endometriosis;
- treatment of background diseases of inflammatory and dishormonal nature.

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