

**PREDICTION OF REPRODUCTIVE DISORDERS IN WOMEN WITH  
ENDOMETRITIS**

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**Abstract:** The review outlines the main role of chronic endometritis in the genesis of reproductive disorders in women. Etiological risk factors for the development and trigger mechanism of chronic inflammatory processes in the endometrium, features of the pathogenesis and clinical picture of chronic endometritis have been identified. The importance of diagnosing chronic endometritis using the immunohistochemical method, the feasibility of immunohistochemical research to assess receptor expression and the severity of local immunity in the endometrium, and the need to determine the alpha-2-microglobulin fertility protein (AMGF) to predict reproductive disorders are shown.

**Keywords:** Infertility, receptor expression, chronic endometritis, alpha-2-microglobulin.

**INTRODUCTION**

Chronic endometritis is a clinical and morphological syndrome in which, as a result of persistent damage to the endometrium by an infectious agent, multiple secondary morpho-functional changes occur that disrupt the cyclic transformation and receptivity of the mucous membrane of the uterine body.

**MATERIALS AND METHODS**

The prevalence of chronic endometritis among the female population has not been fully studied and, according to various sources, ranges from 0.8 to 70% [1]. According to A.V. Shurshalina, the proportion of patients with clinical manifestations of chronic endometritis does not exceed 3.3%, however, the frequency of the disease depends on the nature of the nosological pathology: for example, chronic endometritis is found in 72% of histological samples of endometrial biopsy in of women with sexually transmitted infections, 68% - in patients with tubo-peritoneal infertility [2].

It should be noted that 97.6% of all cases of chronic endometritis occur in the age range from 26 to 35 years, which is the most important in the implementation of reproductive function [3].

**RESULTS AND DISCUSSION**

There is an opinion that the presence of microorganisms in the uterine cavity does not necessarily have etiological and diagnostic significance. Some authors in their works indicate that the uterine mucosa cannot be sterile. It is believed that the endometrium is continuously exposed to ascending infection from the lower parts of the genital tract, and during menstruation the endometrium is physiologically protected from microorganisms. This point of view assumes the presence of microorganisms in the endometrium in almost all women. However, in some cases, this mechanism may not be sufficient to protect the uterine mucosa from pathogens, which may result in the development of a chronic inflammatory process in the endometrium [2].

Undoubtedly, immune factors play a large role in this. There is an assumption that the development of the inflammatory process depends on the state of the immune system of the macroorganism, and this can be determined by genetic predisposition and the presence of risk factors.

The effect of sex steroids on the endometrium is normal and during a chronic inflammatory process. Morphological changes in the constituent components of the endometrium normally occur during the menstrual cycle under the influence of estrogens and progesterone. Estradiol is the most active estrogen, binding to estradiol  $\alpha$ -receptors, causing an increase in the thickness of

endometrial tissue. In addition, estradiol increases tissue sensitivity to progesterone by inducing the expression of progesterone receptors, since the effects of progesterone require previous or simultaneous exposure to estrogens on the endometrium. Endometrial epithelial cells are estrogen-sensitive, but they do not proliferate as a result of the direct action of estradiol. Estrogens act on stromal cells, promoting the synthesis of growth factors in epithelial cells, which leads to an increase in DNA synthesis and the proliferation of neighboring epithelial cells. In contrast to the proliferative effect of estrogen, the action of progesterone promotes endometrial differentiation. It can inhibit and have an effect opposite to the proliferative effect of estrogens on the functional layer.

The role of chronic endometritis in reproductive losses.

The endometrium contains a large number of immunocompetent cells, which is of great importance for the immunological dialogue between the endometrium and the fertilized egg, creating optimal conditions for implantation and placentation. The most numerous population among leukocytes is large granular lymphocytes - CD56 cells. In the proliferation phase, the proportion of CD56 is 8% of all endometrial cells, and in the secretory phase - 60–70%. Normally, NK cells, under the influence of progesterone, migrate to the endometrium and, during physiological pregnancy, express receptors that bind to molecules of the HLA-G locus. This interaction ensures the suppression of the cytotoxic activity of NK cells [2]. NK cells localized in endometrial tissue produce IFN- $\gamma$  (interferon- $\gamma$ ), which is necessary for vascular remodeling during pregnancy at the local level. It is assumed that progesterone produced during pregnancy binds to the receptors of CD8 T cells, which leads to the production of progesterone-induced blocking factor (PIBF), which, acting on NK, implements the maternal immune response directed towards the embryo, towards less active NK-large granular lymphocytes carrying markers CD56, CD16. The maternal immune response is normally realized through Th2, which produces regulatory cytokines (interleukins IL-3, IL-4, IL-10, IL-13) [2]. In chronic autoimmune endometritis, the immunoregulatory index changes due to a decrease in cytotoxic T-lymphocytes and an increase in the number of T-helper cells, which leads to the predominance of a Th-1 type immune response.

## **CONCLUSION**

Chronic endometritis and the morphofunctional disorders caused by it are one of the causes of infertility, miscarriage, and placental insufficiency. An analysis of scientific publications showed the existence of a wide variety of diagnostic methods, which indicates the absence of a unified approach to the diagnosis of this pathology.

Thus, at present, the issues of improving methods for diagnosing chronic endometritis, making it possible to verify the diagnosis, avoiding repeated entries into the uterine cavity, and methods for preventing chronic endometritis after intrauterine interventions, remain relevant. It is also relevant to search for new technologies for the treatment of chronic endometritis, preconceptional preparation in women with morphofunctional disorders of the endometrium, which will reduce the incidence of reproductive function disorders.

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