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TITLE

FEATURES OF MOLECULAR AND BIOLOGICAL PROCESSES AT ADENOMYOSIS

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ABSTRACT

Objective: to estimate the role of matrix metalloproteinase-9 and proliferation marker Ki-67 in clinical expression of adenomyosis

Methods. 62 women with adenomyosis. Patients with moderate and severe pain and with hyper polymenorrhea and anemia of average and serious degree belonged to 1A group (n=32), with a mild pain, absence of anemia or hyper polymenorrhea and with anemia of mild degree — to 2A group (n=30). Control group (n=23) - without pathology endo - and myometrium.

Immunohistochemical research has been done according to the standard technique.

Results. Active adenomyosis was characterized by nodal form or diffusion form of II and III degree of distribution, prevalence of stromal component, expressed hyperplasia and hypertrophy of myocyte, frequent combination with cellular and mitotic active myoma of uterus, complicated hyperplasia with atypia and without it. Inactive adenomyosis was characterized by diffusion distribution of the II degree with prevalence of a ferruterous component. The product of reaction of Ki67 is found in nucleus of stromal, ferruterous and epithelial cells of endometrium and the centers of adenomyosis, fibroblast and macrophages. In 1A group expression of Ki-67 in sites of adenomyosis 12,1±1,3, in autological hyperplased endometrium - 11,5±1,2, in 2A - respectively 3,5±0,7 and 2,8±0,6 points (?<0,05). MMR-9 was found in cells of stroma of the centers of adenomyosis, sometimes in cytoplasm of apical parts of epithelial cells. MMR-9 expression (respectively in strom and in epithelium) in 1A group: adenomyosis - 4,2±1,0 and 0,1±0,02, autological endometrium - 4,3±1,0 and 0,1±0,01, 2A group: adenomyosis — 1,8±0,6 and 0,2±0,01, unchanged endometrium - 1,9±0,6 and 0,1±0,01.

Conclusion. Clinical expression of adenomyosis is caused by functional activity of endometrioid heterotopias, intensity of invasion and proliferation.

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