

Role of endometrial hyperplasia-related inflammation and apoptosis in atypical endometrial transformation

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The development of endometrial hyperplasia (EH) is considered as a multistep process, with slow progression from normal endometrium to hyperplastic due to an imbalance of estrogens and progestogens, oxidative stress, and dysregulations of apoptosis. Our objective was to examine the level of several factors involved in progression of EH such as role of apoptosis, inflammation (using local inflammatory cytokines and nonspecific protease levels), CD 45, CD 56, CD 95, VEGF expression, and histological examination. In the study 115 patients (ages 29–50 years) were examined. The patients were randomized into groups: normal endometrium (n = 18) as the control, simple hyperplasia (n = 42), complex hyperplasia without atypia (n = 39), complex hyperplasia with atypia or adenocarcinoma (n = 16). The following were evaluated for patients: steroid hormone levels in blood serum and uterine flushings, expression estrogen and progesterone receptor, CD 45, CD 56, CD 95, VEGF

and markers of apoptosis (CD 95, Bcl-2) as well as the local levels of the IL-1b, IL-6, TNF- α , and nonspecific proteases and their inhibitors. Results were shown that the level of estradiol in uterine flushings was elevated ($p < 0.05$) dramatically in simple EH as compared to that of controls, but there was no significant difference between estradiol levels among the different forms of EH. The level of CD 95 decreased significantly ($p < 0.01$) in the cancerous group compared to control and simple hyperplasia. The estimation of CD 45, the levels of the IL-1b, IL-6, and TNF- α , and the activity of elastase-like proteases showed that levels of nonspecific inflammatory markers increase with EH progression. We suggest that development of inflammatory changes in endometrial hyperplasia may be considered as a factor in the promotion and progression of pathology, as well as an attributed risk factor for malignancy in endometrial hyperplasia