

Regulation of apoptotic pathways during endometriosis: from the molecular basis to the future perspectives.

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ABSTRACT

PURPOSE: Endometriosis is defined as the presence of endometrial-like endometrial cells, glands and stroma outside the uterus, causing a strong inflammatory-like microenvironment in the affected tissue. This may provoke a breakdown in the peritoneal cavity homeostasis, with the consequent processes of immune alteration, documented by peripheral mononuclear cells recruitment and secretion of inflammatory cytokines in early phases and of angiogenic and fibrogenic cytokines in the late stages of the disease. Considering the pivotal role of interaction between immune and endometriotic cells, in this paper, we aim to shed light about the role of apoptosis pathways in modulating the fine-regulated peritoneal microenvironment during endometriosis.

METHODS: Narrative overview, synthesizing the findings of literature retrieved from searches of computerized databases.

RESULTS: In normal conditions, endometriotic cells, refluxed through the fallopian tubes into the peritoneal cavity, should be attacked and removed by phagocytes and NK cells. During endometriosis, the breakdown of peritoneal homeostasis causes the failure of scavenging mechanisms, allowing the survival of endometriotic cells. The consequent so-called "immunoescaping" of endometriotic cells could be due, at least in part, to the reduction of apoptotic-mediated pathways previously described.

CONCLUSION: Considering the large amount of evidence retrieved from in vitro as well as in vivo models, the reduced apoptosis of endometriotic cells together with the increased apoptosis of peritoneal fluid mononuclear cells may address the peritoneal homeostasis to a permissive environment for the progression of the disease.