

Apoptosis in maternal-fetal interface may affect the fetal survival during early pregnancy

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OBJECTIVES: Apoptosis, programmed cell death, is a concept of natural turnover of cells necessary for proper maintenance of a healthy organisms and is controlled by multiple signaling and effector pathways that mediate active responses to external growth, survival or death factors. Recent studies demonstrated that apoptosis occurs in the reproductive organs of several mammals including human. Therefore, aberrant apoptosis pathway can results in pathological defects. In this study, to investigate a role of apoptosis at the maternal-fetal interface, we examined the apoptotic cells at the maternal-fetal interface in normal and recurrent pregnancy loss.

METHODS: Tissues were collected from five normal pregnant women who underwent an elective termination and five aborters who had history of two or more consecutive spontaneous abortion during the first trimester. They are at the gestation stages of 6-8 weeks and 7-8 weeks, respectively. All tissues showed a normal karyotype. Apoptotic cells were detected in formalin-fixed, paraffin-embedded chorionic villi and decidual tissue by direct immunoperoxidase detection of digoxigenin-labelled genomic DNA (Apop Tag Kit, Intergen). Apoptotic index was calculated by the percentage of positive cells per total number of cells

RESULTS: Apoptosis occurred at the maternal-fetal interface both normal and recurrent pregnancy loss during early pregnancy. However the incidence of apoptosis in syncytiotrophoblast and cytotrophoblast of chorionic villi and decidual stroma is significantly higher in recurrent pregnancy loss than in normal pregnancy ($p < 0.05$).

CONCLUSIONS: This study showed that apoptosis occurred in maternal-fetal interface in natural course and we concluded that apoptosis in the maternal-fetal interface during early pregnancy might play a pivotal role in maintenance of successful pregnancy. The different incidence of apoptosis between therapeutic abortion and recurrent abortion can explain the pathological process of early fetal loss.