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Morphological Changes in the After Birth Tissues in Intrauterine Chlamydial Infection

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Editorial

Bovine chlamydia causes great economic damage to agriculture due to the fact that the pathogen is widespread in the population, is extremely virulent and pathogenic for fetal intrauterine development, while in adults the disease has little symptomatic or latent course. Intrauterine infection is characterized by a vertical route of infection - from mother to fetus, and the shorter the gestational age, the more dangerous the pathogen to the forming tissues of the fetus. In this regard, the study of afterbirth tissues (placenta, umbilical cord, fetal membranes) in order to clarify the point of "break" in the system mother - placenta - fetus, when the pathogen spreads freely to various organs and fetal tissues.

Bovine placenta belongs to the category of desmochorial by its histological structure. Structurally - its functional unit is a caruncle, where the fetal part of the placenta is in direct contact with the mother. It is in this area carried out the most active processes, vital to the forming fetus.

Given the multifaceted links of pathogenesis of infection, we should keep in mind the multi-organ nature of morpho-structural changes, when immature organs are affected, where the need for oxygen and nutrients is high, which is provided by the adequate function of the placenta. Thus, when infected early chlamydia can cause the formation of birth defects, often incompatible with life. Later intrauterine chlamydia leads to stillbirth or the birth of a premature, non-viable fetus.

Damage to the placenta consisted of specific changes and processes associated with secondary changes in its structures in the persistence of the pathogen and its vital activity. Microscopically most often there were lesions of the extraplacental membranes, consisting in the appearance among the peripheral trophoblast of decidual cells with small vacuoles in the cytoplasm, in which there were basophilic or oxyphilic inclusions with a characteristic rim. Nuclei of affected cells were either large, with preserved nuclear structure, or in a state of pycnosis and rexis. In the membranes we could see not sharply or significantly pronounced predominantly lymphocytic infiltration with an admixture of macrophages and neutrophilic leukocytes.

Structural changes characteristic of chlamydia was also observed in vascular endothelium, amniotic epithelium, decidual and trophoblastic cells of basal plate, smooth and villous chorion as small vacuolization of cytoplasm and presence of small basophilic and SHIK-positive inclusions in vacuoles. In addition, in the placenta revealed vascular disorders of varying severity, various variants of pathological immaturity, disorders of maturation of placental structures, leading, according to Borovkova L.V. (2009), to fetoplacental insufficiency.

In the material we studied macroscopically placental tissues were characterized by uneven expression of cotyledons, hyperemia or pale anemia of them. Vartona jelly of umbilical cord was unevenly expressed, thickened dense walls of arterial vessels with gaping lumen were seen through it.

Microscopically, changes involved smooth chorion, villous chorion, as well as umbilical cord and maternal part of the placenta. The epithelium of the inner lining of the uterus was preserved outside the caruncle space. The decidual cells underwent rather significant changes.

Thus, our studies revealed morphological changes in the tissues of the placenta, umbilical cord and fetal membranes when diagnosed with chlamydial infection in adults in cases of stillbirth or birth of premature off spring.

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