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MORPHOLOGICAL CHANGES IN PREGNANT PLACENTA HIGH RISK INTRAUTERINE INFECTION

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Abstract. The Revealed deflections in womb, structure of the placenta are indicative of disadvantage influence intrauterine infections on practically all from-divide share placentas, causing in them change, which in the following, in the event of failure adaptive-compensatory reserve, bring about functional fetoplacential insufficiency and hang from period gestation.

Keywords: Risk of fetal infection, the latter, adaptive-compensatory reserve.

Relevance . Intrauterine infection (IUI) of the fetus is one of the most urgent problems of obstetrics and perinatology due to the high level of infection in pregnant women, possible impaired fetal development and the birth of a sick child. The frequency of intrauterine infection in newborns ranges from 2 to 12% and does not tend to decrease. In the structure of causes of perinatal mortality, the share of intrauterine infection is more than 30% [1, 2, 4]. At gestation periods of more than 15-16 weeks, the causes of non-developing pregnancy (NB) of an infectious nature come to the fore. The question of the etiological role of the bacterial-viral factor remains open. Some researchers believe that infection is one of the most significant causes of both sporadic and recurrent pregnancy loss (RNP); others believe that infection plays a key pathogenetic role for sporadic but not RPL [2, 8, 9]. In most women, against the background of chronic endometritis, the presence of 2-3 or more types of obligate anaerobic microorganisms and viruses was noted in the endometrium, in 86.7% of cases, the persistence of conditionally pathogenic microorganisms in the endometrium is observed, which can cause activation of immunopathological processes [1, 9] . Particular attention is paid to viral infections, the persistence of which in the body leads to changes in the immune system, the development of a chronic thrombophilic condition, and autoimmune diseases [4, 8]. Mixed persistent viral infection (herpes simplex virus, human papillomavirus, Coxsackie A, Coxsackie B, enteroviruses 68-71, cytomegalovirus, parvovirus) occurs in patients with RPL significantly more often than in women with a normal obstetric history [3, 4, 5]. Bacterial-viral colonization of the endometrium is usually a consequence of the inability of the immune system and non-specific body defenses (complement system, phagocytosis) to completely eliminate the infectious agent [7 , 8].

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Target: Morphostructural analysis of the placenta in women at high risk of intrauterine infection

Material and methods. The study of the afterbirth was carried out on the basis of the methods described earlier by A.P. Milovanov and A.I. Brusilovsky (1987), modified by E.P. Kalashnikova (1988). The material was taken from women afterbirth after premature birth.

Results and discussion . Morphostructural analysis of the placenta in women with a high risk of intrauterine infection (IUI) whose pregnancy ended in premature birth revealed an inflammatory process with massive deposits of fibrinoid masses in the intervillous space, total fibrinoid transformation of individual villi, the presence of calcifications in combination with areas of fibrinoid necrosis indicates their role in premature termination of pregnancy. Fibrinoid degeneration of the villi should be considered the result of an immunological reaction leading to rejection of the fetal-placental complex [4,6,7].

Marked pathological immaturity of the placenta in the form of dissociated development of villi with areas of increased branching (Fig. 1), inflammatory and sclerotic changes with phenomena of giant cell transformation of all layers of the placenta, lympho-macrophage infiltration of the basal plate (Fig. 2) of the chorion, circulatory disorders of an involutive-dystrophic nature , the phenomena of basal decicuitis indicate in favor of the ascending route of infection. And as you know, changes in blood vessels and trophoblast in the form of its vascularization, plethora and stasis lead to microthrombosis, impaired microcirculation, insufficiency of placental trophism and poor nutrition of the fetus [2,3,5].

In addition to the noted structural disorders in all placentas, signs of a compensatory-adaptive nature were revealed, the severity of which turned out to be different. So, with weakly manifested changes, compensatory reactions were expressed only by the presence of single syncytial (Fig. 3) nodules and syncytiocapillary membranes.

Thus, the state of the fetus and the outcome of pregnancy with UGI depends both on the degree of structural changes in the placenta (violation of its maturation, involutional dystrophic and inflammatory changes), and on the intensity of the development of compensatory devices in it, the development of syncytial nodules and syncytiocapillary nodules and synithiocapillary membranes, as well as the volume in the intervillous space of circulating blood. The recurrence of infection in the II and early III trimester of pregnancy contributes to an increase in the phenomena of fibroplasia , foci of necrotic changes, reduction of the vascular bed, an increase in inflammation processes in it, signs of circulatory disorders (characteristic of herpetic,

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viral lesions, chlamydia), which may be accompanied by the development of an infectious process in the fetus, since damage to the vessels of the villi and trophoblast leads to microthrombosis , impaired microcirculation [1,4,5,8].

Fig. 1. Placenta in IUI. Remission. Decidual plate with destructively altered decidual cells, nuclear hyperchromia . Focal lymphohistiocytic infiltration with fibrinoid deposits . Hematoxylin and eosin. Uv . 100×

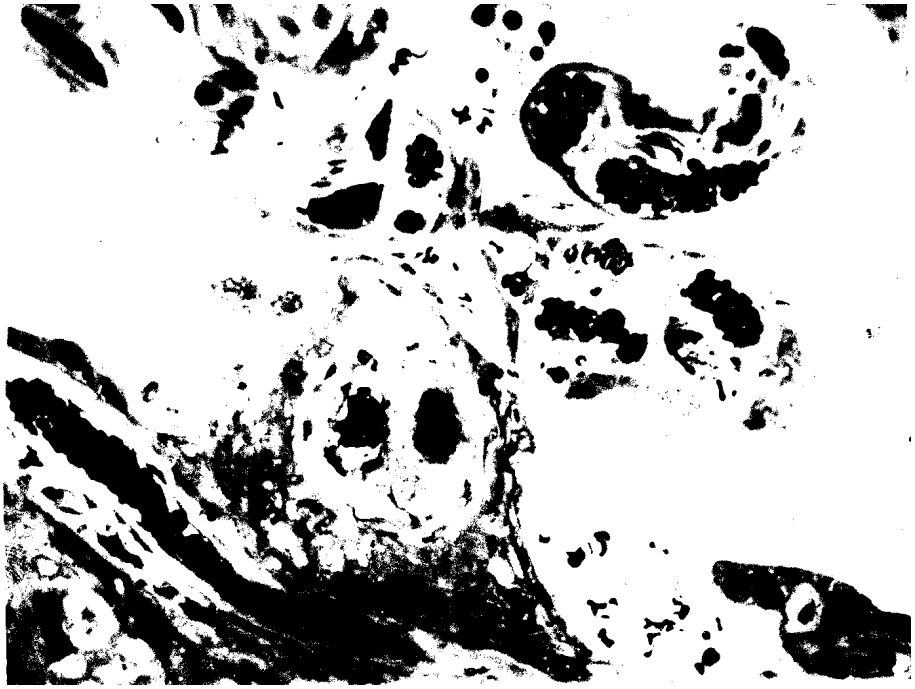

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Fig. 2. Placenta in IUI. Remission. Expansion of the lumen of the vessels in the decidual tissue of the mucus and the stroma of the terminal villi with formation of blood clots in some of them. Half cut. Uv . 400×

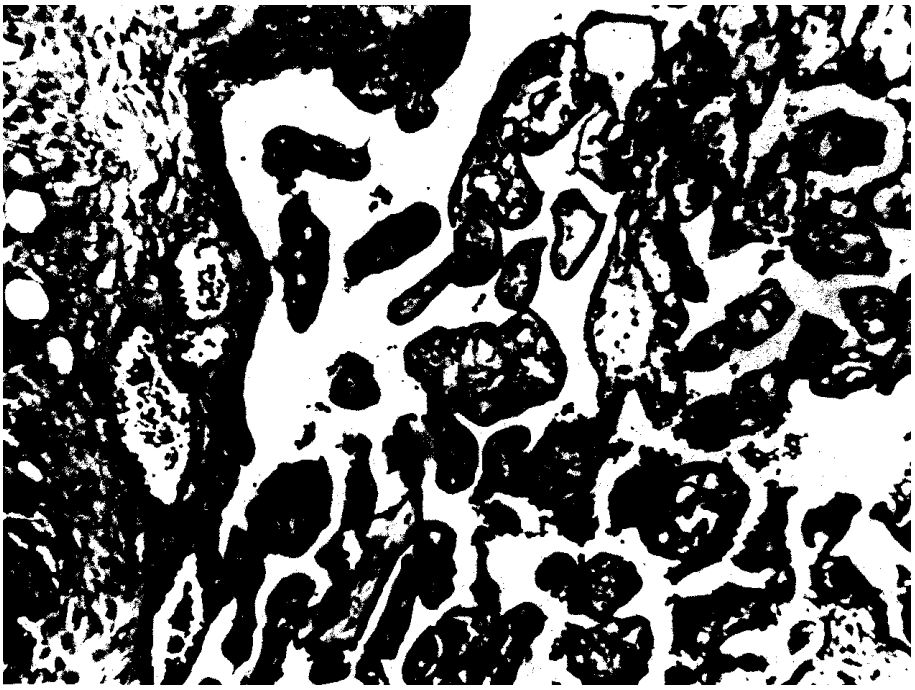

Fig. 3. Placenta in IUI. Dense arrangement of villi, stromal edema, some with single Kashchenko-Hofbauer cells and infiltration of others. Hematoxylin and eosin. Uv . 100×

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Fig. 4. Placenta in IUI. Remission. immature intermediate villi, avascular villi with pronounced edema of the stroma and the content of Kaschenok-Hofbauer cells . Hematoxylin and eosin. Uv . 200×

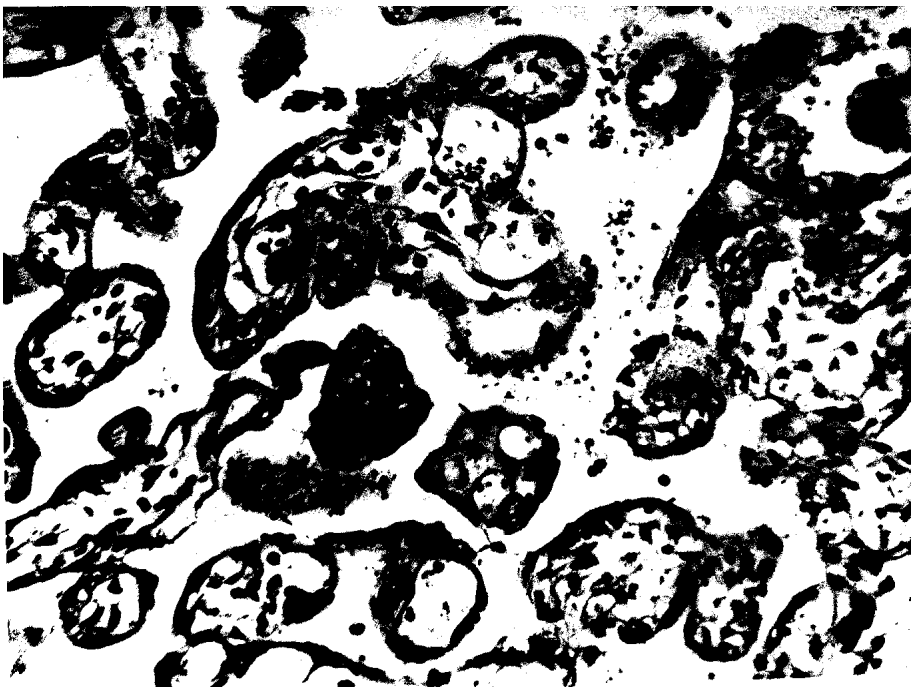

Fig. 5. Placenta in IUI. Mature, round shape villi with many blood vessels and syncytiocapillary membranes. Hematoxylin and eosin. Uv . 200×

The recurrence of infection in the II and early III trimester of pregnancy contributes to an increase in the phenomena of fibroplasia , foci of necrotic changes, reduction of the vascular bed, an increase in inflammation processes in it, signs of circulatory disorders (characteristic of herpetic, viral lesions, chlamydia), which may be accompanied by the development of an infectious process in the fetus, since damage to the vessels of the villi and trophoblast leads to microthrombosis , a violation of microcirculation.

Compensatory-adaptive processes were insufficient to ensure adequate nutrition of the fetus with such pronounced structural disorders of the placenta, indicating the severe nature of its infection with signs of a decompensated form of chronic placental insufficiency.

Small mass of the placenta, inflammatory changes in the decidua with necrosis of decidual cells, pericapillary sclerosis of the stroma of the villi, fibrinoid necrosis of the chorionic villi leads to placental insufficiency, which plays a leading role in the development of preterm labor in pregnant women against the background of UGI.

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