FEATURES OF THE PLACENTA LESION WHEN THE FETUS IS INFECTED WITH A HERPESVIRUS INFECTION.

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https://doi.org/10.5281/zenodo.13922719

ARTICLE INFO

Qabul qilindi: 01- Oktyabr 2024 yil
Ma'qullandi: 05- Oktyabr 2024 yil
Nashr qilindi: 12- Oktyabr 2024 yil

KEYWORDS

herpes virus, genital herpes, pregnancy, placental insufficiency, intrauterine infection.

ABSTRACT

The article presents the features of changes in placental tissue in pregnant women with herpes virus infection. A microscopic description, organometry, and macroscopic examination of 63 subsequent samples taken from women in labor with a genital form of herpes virus infection were carried out.

In recent years, there has been a tendency to increase the infection of pregnant women with herpes simplex virus (HSV). Great attention is paid to genital herpetic infection, since its ability to actively multiply in the placenta and embryonic tissues, penetrate the placental barrier and cause intrauterine damage to the fetus leads to the formation of placental tissue insufficiency and the development of fetal malformations. The increasing role of intrauterine infections among the causes of adverse perinatal outcomes determines the relevance of a comprehensive study of this problem.

The objective of our study was to present the features of changes in placental tissue during herpetic infection.

Research Methods. A macroscopic description, organometry, and light-optical microscopic examination of 63 afterbirth 38–40 weeks of gestation were carried out.

In our study, the 63P placenta with lesions of the herpes simplex virus type 1 and 2 was studied. In observations, type 1 herpes was found in 22.5%, type 2 herpes in 42.8% and double herpes infection of types 1 and 2 in 34.7%. Therefore, we divided them into 3 groups.

Results. Macroscopically, the appearance of the placenta did not differ much from the usual one. It looked like an oval disk. The fertile surface of most placentas was smooth. Only one had thickenings of the chorionic plate. The maternal surface, as a rule, had distinct lobules. In 13.8% of the placentas, their sizes were not the same. In these cases, the small lobules were compacted and whitish in the section. The surface of the lobules was often smooth, and in 27.4% of the placentas, plaque whitish thickenings were determined on it. As with other viral infections, the villous chorion tissue had a spongy appearance, depending on the blood supply of gray-red or cherry-red color. Multiple small foci of dense white consistency were detected in the placenta tissue, in 6 placentas they were larger, however, not exceeding 8-12% of the total volume, and were located mainly on the periphery of the placental disc. In addition, in 32.1% of cases, hemorrhages were found in the maternal part of the placenta, the villous

chorion, and less often in the membranes and umbilical cord. In one placenta, nodes of capillary hemangioma were identified. The umbilical cord had properly formed vessels. In 11 observations, its marginal or eccentric attachment was noted. The extraplacental membranes of all the placentas, with the exception of 43, were thin, smooth. In 27.4% of cases there were plaque-like focal thickenings, and in 16.9% of cases green staining. In the 1st group, the placental-fetal index was 0.13, in the 2nd group 0.16 and in the 3rd group 0.17.

A histological examination in all cases in the presence of a full-term pregnancy in the placenta showed varying degrees of severity of structural differentiation. In this case, fields of villi located at different stages of development were revealed. Terminal villi predominated. Along with them, areas were determined that contained immature and mature intermediate, chaotic, sclerosirope, and single embryonic villi. Immature intermediate villi were characterized by irregular shape, large sizes (diameter 110-180 μm), single small branches extending from them, loose stroma, weak vascularization (5-8 centrally located small capillaries), and the presence of Kashchenko-Hoffbauer cells. The presence of a significant amount of such villi in the placenta during a full-term pregnancy indicates a violation of the development of villous chorion with a gestational period of 19-25 weeks. Thus, in all the observations, dissociated development of the villous chorion with a predominance of terminal villi was noted. In all cases, widespread lesions of the walls of the vessels of the villi were observed in the form of focal endovasculitis, sometimes with necrosis of part of the inner lining and the formation of blood clots. Intervillitisitis was common and was focal in nature. Hemorrhages or foci of thrombosis in the intervillous space were more typical. Basal deciduitis of varying severity occurred in 31.7% of cases. Necrotic changes were found both in individual cells and in the form of foci. In addition, areas of sclerosis, diffuse deposits of calcium salts, fibrinoid in the villi and basal lamina were constantly revealed.

Changes in the placenta largely depended on the time of occurrence of relapses and their number. The degree of destruction was more pronounced in patients with 4 relapses or more.

Exacerbation of the disease in the I and II trimesters of pregnancy was morphologically manifested by fibroplastic changes. In the vessels of stem, mature intermediate and terminal villi, there were manifestations of productive vasculitis, with fibrosis of the walls, obliteration and stenosis of the lumen of some of them. The chorionic plate looked thickened, intensely stained with fuchsilin according to Van Gieson. Fetal membranes are edematous, their epithelium is thickened in places, forms several rows. Inflammatory changes were focal in nature. It was morphometrically established that the number of avascular terminal villi was 15.3 ± 0.62%; 46.0 ± 3.24% contain 1-2 capillaries, 24.8 ± 0.96% - 3-5 capillaries, 9.4 ± 0.86% - 10 capillaries, 4.5 ± 0.11% - more 10 capillaries.

The occurrence of relapses of herpetic infection in the III trimester of pregnancy was combined with alternative processes in the structures of the afterbirth. In the basal plate, septa, large and small villi, amnion, smooth chorion, pronounced dystrophic changes, necrobiosis, foci of fibrinoid necrosis are found. At the same time, there was a lymphoid-plasmacytic and lymphoid-macrophage inflammatory reaction, often with giant cell metamorphosis and focal hemorrhages. Inflammatory changes and necrosis of the membranes were accompanied by rupture of the fetal bladder, premature outflow of amniotic fluid, and the occurrence of an ascending infection. In this case, infiltration by polymorphonuclear leukocytes penetrating from the vessels of the decidual or amniotic

membranes and the polymorphous space prevailed microscopically in the amnion. nuclear leukocytes that migrated from the vessels of the chorion plate. In the intervillous space, inflammatory changes were of limited focal nature and represented by scanty lymphoid-plasmacytic infiltration, and the presence of fibrin strands. In the villi there were deposits of calcium salts. In 19 observations, focal hemorrhages were detected in the intervillous space. It was morphometrically established that the number of avascular terminal villi is 13.8 + 0.3%; 37.2 ± 1.22% contain 1-2 capillaries, 24.0 ± 1.1% - 3-5 capillaries, 7.6 ± 0.31% - 6-10 capillaries.

Along with the described changes characterizing the violation of the maturation of the placenta, its damage and inflammation, there are varying degrees of severity of compensatory-adaptive processes. The latter are determined by the degree of vascularization of the villi, depending on the time of relapse, the presence of functional syncytial nodules and the amount of circulating maternal blood in the intervillous space. Functioning syncytial nodules, reflecting the proliferative capabilities of trophoblast, are available in all cases. They are a cluster of basophilic nuclei closely adjacent to each other. Often these structures form “bridges” connecting terminal nap and supporting their internal mutual arrangement.

Conclusion. Genital herpetic infection leads to the development of chronic placental tissue insufficiency, the severity of which depends on the time, frequency and duration of the disease recurrence. Changes in the placenta are characterized by the presence of both destructive and compensatory processes, the degree of preservation of which determines the further development and condition of the fetus.

Pregnant women with this pathology should be assigned to a high-risk group for the possible development of placental insufficiency. The examination plan for patients with this pathology should include modern diagnostic methods for the status of the fetoplacental complex and morphological examination of the placenta. The revealed morphological changes in the placenta allow timely initiation of pathogenetically substantiated therapy for newborns, which helps to reduce the incidence of complications of herpesvirus infection.

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