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HERPES VIRAL INFECTION AND STRUCTURAL CHANGES IN WOMEN'S PLACENTA

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Introduction

One of the basic problem of modern medicine is the perinatal and neonatal mortality [1,3,4]. Among it various cause important place belong to infections, in particular Herpes simplex virus infection (HSVI). The incidence of HSVI has been revealing into different groups of population, but most dangerous occurs the case of determination of HSVI among women of reproductive age and pregnant. It is connected with the high risk of transmission of the virus from mother to their fetus (babies) during pregnancy [5,6]. Then, perinatal HSV infection has been reason considerable number of diverse disabled disease. One of the reserve, which can be influence on this index are morphological study of placenta for definition infectious damage and different variant of placental deficiency [1,4].

Materials and methods

Preparations from placentae of pregnant women with clinically and virologically established HSVI were used as material investigation. Material taking was performed in accordance to the established order from patients of Maternity hospital № 6 (Kharkiv, Ukraine). All the investigated pregnant women (from the moment of pregnancy diagnosis and its term establishment till the date of delivery) have been observed in the outpatient department of the Maternity hospital.

In each case of the histological investigation was made using traditional methods of light microscopy. Histological analysis was preceded by placental macroscopical examination for such high-informative indexes determination as: organ mass and fetoplacental index. In all the observations the state of extraplacental coats, fetal and maternal placental surfaces were described; the cases of haemorrhage, “infarcts” were fixed, the slices of placentae (0.5x0.5 cm) were taken all over the thickness of the organ both from central and peripheral regions of placental disk. Received samples were fixed in 12 % formalin on phosphate buffer (pH = 7.0-7.2), were installated through spirit battery with increasing concentration, were poured into celloidin wax. In received samples stained by haematoxylin-eosin and Van Hison method in microscope LOMO Magnification x c.x300; x c.1350. The state of vascular canal, shaggy chorion cellular pull was analyzed.

For HSVI detection the immunoenzyme test-system HerpesScreen for determination of HSV antigen of the form NEARMEDIC PLUS (Moscow, Russia) was applied. Basic working components of the test-system were monoclonal antibodies (MA) for HSV and conjugate (MA, marked with horse-radish peroxidas). Registration of the results of immunoenzyme test was performed with the spectrophotometer for AIF-TS-01C. Optical density (OD) was measured with the wave-length 492 nm on investigated and control (positive and negative) samples. Investigated sample was considered to be positive, if its OD value was equal or two times higher than one of negative control sample.

Discussion and results

Structural changes revealed in placental tissue of investigated women with HSVI, formed an integral picture of lesions, were of the same tendency, sequence and ubiquitous. Vascular canal, shaggy chorion tissue, basic membrane were involved in pathological process.

Material of structural change wasn't revealed with the help of macroscopic examination of placentae and its coats. The placentae had center in or near the median plane. The umbilical cord, usually attached near the center of the organ. Only one placentae variation was presented, in which its margin is undercut by a deep groove.

Extraplacental coats were thin, translucent, whitish, in a number of cases (2) they proved to have lobule approximately the same dimension and shape. The placentae lobes were demarcated by the grooves on its maternal surface and they correspond to the major branches of distribution of umbilical vessels.

Shaggy chorion tissue depending on the degree of blood filling was spongy, cherry, reddish-brown. Practically, in all the examined cases (7) there were regions of haemorrhage, of ischemic infarcts.

The mass of the organ varied from 260 g till 610 g. Fetoplacental index 0.07-0.09 which was lower than the normal one (0.12-0.20).

Placental vascular canal disturbanes were of degenerative and destructive nature; they were accompanied by their wall disorganization, development of stasis and thrombosis. Vascular endothelium was much thinner than normal one, with marked signs of exfoliation. Endothelial fragments with unequal, festongat contours were hanging down into the vascular lumen. Degeneration of endothelial layer was accompanied by structural disorders of basic membrane and vascular wall as a whole. Thrombosis presence with subsequent formation of thrombuses was seen in all the investigated preparations. Long-lasting character of the lasion, its depth (pyknosis, caryorrhexis followed by decomposition and lysis of the cell) were proved by fibrosis and vascular canal reduction. It's possible that disorders of the microcirculation with its probable complete stopping in examined fragments led to their disconnection from the functional activity of the whole system. Perivascular oedema as a result of hyperpermeability, led to vascular tissular isolation and posterior development of tissular hypoxia. It's probable that this process has created complementary causes, which aggravated structural changes in vascular wall. Signs of hydratation increase, gradual augmentation of internal oedema, laminar border dissolution of cytoplasmal membranes indicated, doubtlessly, at the penetration of liquid into cells from endothelial area.

Microvesiculation, directed at the excessive and substances dissolved in it, elimination, from tissue into blood played the role of protective mechanism. But at the same time, it helped numerous pores formation in the endothelium, which was changing considerably its barrier functions and increasing cellular membrane permeability, led to exhaustion of membrane reserves. That's why we had such a typical macroscopic picture of placentae, Vascular canal changes provoked corresponding reactions in perivascular space. The vasodilatation could be observed. To the damaged external walls of damaged vessels numerous small leukocyte accumulations confirmed to be adjacent. Such a destruction was accompanied by a considerable leukocyte infiltration. Morphological changes corresponded to virologic parallels, accordingly to Herpes Simplex Virus detection in placental emulsion.

At the same time shaggy chorion involvement in pathological process occurred. Focal villous dissemination lesion of different size was seen. Terminal and anchoring villi suffered the most considerable changes. The stroms of the latter ones presentated oedems and intervilliferous space dilatation. Productive and proliferative villesitis accompanied focal retardation of villous maturation. At the same time numerous pale infarcts and focal haemorrhages were accompanying fibrinoid necrosis (villi fibrin deposit). This last phenomenon was considered to be a protective reaction which blocked massed causative agent penetration into the fetal part of placentae. Also it is possible to observe villi is

adhesion with further disturbance of their trophism, autonomic homeostasis and, as a probable consequence of this, infiltrative inflammation development of different degree. As it was demonstrated before, the composition of infiltrations was formed by predominant lymphoid cells. Were observed at the same time plasmocytes and isolated macrophages. Nevertheless, lymphoid cells spectrum expansion far from compensate pulls losses by means of cellular decomposition, neither affet remarkably increased destructive process. That's why repopulation reconstractions in placentae can't be considered as compensative. As a matter of fact, in examined cases the compensative phenomens development wasn't remarkable, excepting the vascular canal, that fact, in our opinion, means a bad unfavorable prognosis for a fetus.

Conclusion

1. Morphological Placental investigation can be used as an important complementary diagnostic criterion of HSVI.
2. Histological placental changes in HSVI were accompanied by inflammatory process development (with signs and pronounced leukocylal infiltration); by stasis and thrombosis in microcirculatory canal, by sclerosis with small-focal infarction.
3. Blood vessels and chorion tissue were structural placental mark of HSVI.
4. Blood canal damage and outcome of the tissue infringement can be examined as a clear factor of the prognosis for the fetus.

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Рефераты

“Герпесвирусная инфекция и структурные изменения в плаценте женщин”

Институт микробиологии и иммунологии имени И.И. Мечникова АМН Украины. Харьков. Украина

В настоящей работе представлены результаты гистологического изучения (гематоксилин- эозин, по Ван-Гизону) препаратов плаценты женщин- родильниц с герпесвирусной инфекцией. Морфологически были установлены типичные деструктивные изменения в структуре сосудов плаценты, выраженная дезорганизация эндотелия, возникшие на ее фоне явления стаза, тромбоза (8 препаратов). С помощью иммуноферментного метода в гомогенизате плаценты был детектирован вирус простого герпеса (I тип).

Герпесвірусна інфекція та структурні зміни у плаценті жінок

Інститут мікробіології та імунології імені І.І. Мечникова АМН України. Харків. Україна

В даній роботі представлені результати гістологічного дослідження (гематоксілін- еозін, по Ван- Гізону) препаратів плаценти жінок- породіль з герпесвірусною інфекцією. Морфологічно було встановлено типових деструктивних змін у структурі стінок судин плаценти, вираженої дезорганізації ендотелію, явищ стазу, тромбозу (8 препаратів). За допомогою імуноферментного методу в гомогенізаті плаценти було детектовано I тип вірусу простого герпесу.

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Placental preparations stained with hematoxylin-eosin and according to Van Hison were studied by histological method. Typical destructive vascular lesions with pronounced disorganization of endothelial wall, signs of stasis and thrombosis in 8 preparation of placenta were established in morphological investigations. In placental emulsions with the help of immunofermental method Herpes simplex virus of the 1-st type was detected.