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CONTEMPORARY INTERPRETATION OF PATHOGENETIC TREATMENT AND PREVENTION OF HERPETIC STOMATITS IN PATIENTS INFECTED WITH TORCH (LITERATURE REVIEW)

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SUMMARY

The high prevalence of TORCh-group infections affects both the demographic situation and the health of the mother and child. The most medically and socially significant feature of TORCh-group infections is their destructive effect on all organs and systems of the fetus, and first of all on its central nervous system, this effect can cause miscarriage, stillbirth and stillbirth of the child. increases the risk of disability, the formation of defects in its development and disability. From this point of view, toxoplasmosis, rubella, cytomegalovirus infection, as well as infections caused by herpes simplex virus types 1 and 2 are of particular epidemiological importance among infections of the TORCh group.

Key words

TORCh, toxoplasmosis, rubella, Cytomegalovirus, Herpetic infection, herpes, replication

Toxoplasmosis is a zoonosis caused by intracellular parasite Toxoplasma gondii. In adults, toxoplasmosis is usually asymptomatic, but congenital pathology can cause miscarriage, stillbirth, or severe neurological damage. The way of transmission of the pathogen is usually alimentary. The main host of the parasite is cats, which are infected by eating infected mice and other animals. Also, farm animals can be carriers of pathogens. For example, in the Republic of Uzbekistan, the main source of toxoplasmosis is large and small horned cattle, the degree of damage by toxoplasmosis is 25.7% and 24.6%, respectively; in which the frequency of injuries of the population is 17.8% on average [1.3.4].

Cases of transmission by human contact, for example, when the skin is damaged during the grinding of raw meat, as well as when infected organs are transplanted or, more likely, through blood transfusion, have been described.

The vertical (mother-to-fetus) mechanism with toxoplasmas is of particular importance, in which a pregnant woman experiencing an early phase of primary damage (manifest or asymptomatic), in combination with a low level of G-class antibodies, develops placentitis and subsequently infects the fetus with

toxoplasmas. This path occurs in 40-50% of cases when therapy is not carried out during pregnancy, and fetal damage usually occurs in the antenatal period. The route of transmission of the causative agent is largely determined by the severity of toxoplasmosis. Thus, if the infection process occurs step by step, in parallel with the development of immunity, in the case of alimentary damage, then in a vertical position - toxoplasmas directly enter the vascular flow of the fetus, and the invasion is of a general nature from the beginning of the process, and no matter how immature the immune system of the fetus is the more severe it appears [2.4.6].

In pregnant women, the frequency of infection and the clinic of toxoplasmosis do not differ significantly from the clinic of matched non-pregnant women living in the same area. In most cases, the disease is asymptomatic. If the early phase of toxoplasmosis in a pregnant woman has a manifest character, it is usually mild and may remain undiagnosed. In both cases, the diagnosis of toxoplasmosis can be carried out by systematic laboratory control during pregnancy. Moreover, it is necessary because the main risk of fetal damage is associated with the primary infection of women with toxoplasmosis during pregnancy, and congenital toxoplasmosis due to placental transmission always takes the form of a general process. Its severity is determined by the infecting dose of the causative agent, the number of protective antibodies from the mother to the fetus, and the period of pregnancy when the injury occurred [1.3].

Children and adolescents may develop late symptoms of congenital toxoplasmosis. Thus, retinopathy develops in more than 85% of children who are asymptomatic. In other cases, rapid fatigue, lymphadenopathy, hearing impairment, and endocrine disorders are observed. Vascular seizures and mental retardation are detected in a number of cases at 2-4, epileptic seizures - at 7-12 years of life. Due to the formation of organic damage to a number of vital organs, specialized therapy is ineffective at this stage of the disease. For this reason, it is clear that it is necessary to increase the time and stages of laboratory diagnosis of toxoplasmosis.

Rubella is an air-capillary infection caused by an RNA virus, which belongs to the rubovirus family of togaviruses. Children aged 7-14 are the main source of infectious agents. It has a pronounced teratogenic effect. It is known that rubella in pregnant women is extremely dangerous for the fetus, especially in the first trimester of pregnancy. In 60-85% of cases, damage during this period leads to general and persistent development of the fetus, which later passes as a multisystem disease with many developmental defects. In this case, about 3/4 of children are born with congenital rubella syndrome (CRS): congenital heart defects, cataracts, blindness, deafness, microcephaly, mental retardation, damage to other

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organs. A very high percentage of perinatal deaths is noted among such babies. Children of the first year of life with passive immunity from the mother form an exceptional group. A clear sign of the epidemiological importance of rubella is the case described in the United States, in which 50,000 pregnant women were infected with rubella in 1960-1964. brought [5.6].

It is less dangerous for pregnant women to get sick in the late periods, during this period, damage to the fetus rarely develops (in 25-30% of cases) and they are less pronounced.

People infected with rubella form lifelong immunity. The development of continuous immunity is also caused by vaccination against rubella. However, there is a possibility of re-infection and re-infection after vaccination. In such cases, rubella is usually asymptomatic and is manifested by a sharp increase in the titer of antibodies after close contact with the patient. Such cases are more common in people who develop immunity after vaccination than in people with naturally acquired immunity [2].

Cytomegalovirus infection (CMVI) is a disease caused by cytomegalovirus (CMV) belonging to the family of herpesviruses. Its main target cells in the human body are monocytes, macrophages, granulocytes, epithelial and endothelial cells, fibroblasts, and smooth muscle cells. In most cases, the infection occurs either without any clinical symptoms or only non-specialized symptoms - fever, fatigue, inflammatory processes in the nose and mouth-throat, palatal tonsils, which are infectious in nature. can be observed in plab diseases.

Compared to other viruses, CMVI is often transmitted from mother to fetus. The frequency of development of CMVI varies from 0.3 to 3.0% of newborns in different countries. In contrast to other infections of the TORCH group, severe damage to the fetus in CMVI can develop in any trimester of pregnancy [1.3].

In pregnant women, primary sMVI usually passes without symptoms, and secondary infection can also pass without symptoms. Manifestation in pregnant women is often similar to mononucleosis or ARVI. About 40% of women with primary infection during pregnancy transmit the virus to their fetus. At least 5% of pregnant women have sMVI reactivation, but the number of newborns with these symptoms does not exceed 1-3% even in underdeveloped countries, probably due to the presence of high levels of protective antibodies in pregnant women. In a number of cases, infection of the fetus in the early stages of the child's development leads to disability and death of the fetus or newborn. In the late stages, the damage does not destroy the structure of the organs, and in the postnatal period it is manifested in the form of jaundice, hepatosplenomegaly, thrombocytopenic purpura, damage to the central nervous system, and pneumonia. Infection is

actively transmitted through infected breast milk, which determines 60% of all cases of perinatal infections. Secondary (recurrent) sMVI is the cause of one-third of congenital infections in economically developed countries and most cases in many developing countries with a very high percentage of seropositive women of childbearing age.

The frequency of development of congenital sMVI in different countries, according to available data, is from 0.3 to 3% of the total number of newborns. In most cases (95%), congenital infection in babies born at term is asymptomatic and without negative consequences. In babies born prematurely, especially as a result of hemotransfusion damage, the infection is severe, often passes as sMV-sepsis and has a negative prognosis. In almost 10% of children, unilateral or bilateral neurosensory deafness, retardation of mental development or movement disorders occur as a result.

Infection of blood recipients with sMV is a serious problem, as it is known that 15 to 40% of children and 2-3% of adults are infected from seropositive donors. Even more serious problems are related to organ transplantation, because the factor of transmission of infection can be not only the transfused blood, but also the transplanted organ [2.5].

Hepatic infection - an infection caused by herpes simplex viruses of type 1 and 2 (VPG-1 and VPG-2), which belong to both the sMV and herpes virus groups, and with tegmental lesions, that is, skin and mucous membrane lesions, nervous system, it is also characterized by damage to other systems in the body. Its manifestation is often associated with immunosuppression, in immunocompromised individuals it can have a desiccated, septic course. The ability of pathogens to cause congenital pathology of the fetus and newborns, to cause latent, acute and chronic forms of infection in the host organism, to damage almost all organs and systems [3].

When the mother has genital herpes, the risk of damage to the fetus during childbirth is about 40% [2.4]. Most people (almost 80%) become infected with HPV-1 before the age of 6 years, with people of higher socioeconomic status usually becoming infected later in life, and some older people not. Clinical symptoms of herpes infection are very different both in terms of location and severity. Infection in the first trimester of pregnancy causes the development of micro-, hydrocephalus, heart defects, gastrointestinal tract, reproductive system, skeletal defects, cataracts, and deafness in the fetus. Infection in II and III trimesters leads to hepatosplenomegaly, anemia, jaundice, hypotrophy, pneumonia, meningoencephalitis, sepsis. The risk of pregnancy loss is 5 times higher in VPG-2 seropositive women than in seronegative women, and heavy bleeding is 10 times

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higher. Pregnancy failure (early and late miscarriage, undeveloped pregnancy) is recorded in 29% of seropositive pregnant women [5].

In the presence of primary herpes simplex in a pregnant woman, the risk to the fetus is significantly higher (up to 50%) than in the case of recurrent disease (less than 3%). Perhaps this is related to the protective effect of maternal antiherpetic antibodies. At the same time, these antibodies do not affect the development of clinical symptoms and the exacerbation of the disease. The highest risk for newborns occurs as a result of exposure to VPG-2 during primary or recurrent genital infection in a pregnant woman. In this case, VPG is present in the birth canal and surrounding skin areas, and the newborn becomes infected during delivery. Perinatal herpetic infection, which is clinically manifested in half of the patients, occurs in the disseminated form, in 30% - in the primary neurological form (meningoencephalitis), and in 20% - in the form of skin and mucous lesions [6].

The variety of clinical forms of herpes simplex makes its diagnosis much more difficult. Since urogenital diseases associated with herpes infection have many clinical signs both in terms of character, intensity (severity) of the pathological process, and location, methods of its laboratory diagnosis are of great importance.

In recent years, against the background of the increase in the number of allergic, autoimmune and immunodeficiency cases in our Republic, there has been a constant increase in socially significant and opportunistic infections of bacterial, fungal, parasitic and viral nature. Inflammatory, chronic, slow-healing processes accompanied by the persistence of infectious agents of viral nature represent one of the main problems in medicine today [3.4].

The small importance of herpes infection is that when the reactivity of the macroorganism decreases, it becomes disseminated. Herpes infection plays an important role in the development of secondary infertility, carcinogenesis, damage to internal organs and nervous system. According to WHO data, the diseases caused by herpes infection take the second place (15.8%) after the flu (35.8%) in terms of the cause of death [...]. In healthy people, herpes infection leads to the development of primary acute infection and the development of chronic, recurrent diseases in the skin and mucous membrane. Herpes infection can be fatal in immunocompromised individuals and newborns [5.6].

Herpetic stomatitis in patients infected with TORCh is a common acute infectious disease, the causative agent of which is various types of herpes simplex virus.

The chronic and frequent course of the disease is recorded in 20% of the population of the Earth. In herpetic stomatitis in patients infected with TORCh, the infection is transmitted in different ways: horizontal way - from a sick person to a

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healthy person, vertical way - occurs in the case of transplacental infection of the fetus comes, autoinoculation - the patient himself transfers the virus from the source of infection to the damaged areas of his body. Herpes infection remains in the human body throughout its life and causes recurrence of the disease in varying degrees of severity. The possibility of infection with herpes infection exists when the virus is released without symptoms and when there are visible lesions on the mucous membrane and tissues [2.3].

Clinical signs of herpetic stomatitis in patients infected with TORCh are associated with the replication of herpes infection type 1, and now the frequency of disease cases caused by herpes infection type 2 has increased [.....]. Numerous studies have shown that 50-70% of patients have antibodies to herpes infection, but most of them have not previously suffered from this disease and are asymptomatic [1.4].

In patients with subclinical or atypical forms of the disease, the diagnosis of herpes infection is not always achieved, and they are characterized by rare reactivation of herpes infection.

High tropism for herpes infection is characteristic of neural and epithelial cells and tissues. Replication of the virus begins at the moment of passage through the damaged areas of the skin and mucous membrane of the oral cavity. For the first time, getting into the nerve nodes, the herpes infection is called a latent infection. Hematogenous way of spread of infection as a result of erythrocytotropism is typical for herpes infection. In the lymphatic system, the herpes virus is either associated with cells of the immune system or not. Regardless of the clinic, the spread of the virus occurs in a volume sufficient to apply to the affective or autonomic nerve endings. After integration into the nerve cell, the virus travels to the normal nerve root, where it is stored in a "dormant" state, either as circulating viral DNA or integrated into the DNA of the cell's chromosome.

When herpes infection enters cell neurons, viral genomes have a weakening effect, bringing them into a state compatible with the normal activity of the cell, and there is a possibility that the virus will go into a latent state in the tissues of the central nervous system, as evidenced by the detection of viral DNA in the brain. After the completion of replication, the reshaped virions leave the cell and simultaneously damage the surrounding cells.

After reactivation of the virus and replication, its subsequent migration along affected peripheral nerve endings occurs. The long-term persistence of the virus in the human body and the frequent recurrences of the disease can be explained by two opposing and most popular theories: static and dynamic. According to the static theory, OVG is in a free state in the cells of the pre-spinal efferent nerve

ganglion, and under the influence of a "triggering" factor, it migrates centrifugally from the nerve ganglion along the peripheral nerve of the axon, and is actively formed in epithelial cells. will be done. The contradiction of the theory is that it does not explain that recurrence occurs after 1-3 days, because the speed of movement of the virus along the nerve tissue is on average 4 days. On the other hand, according to the dynamic theory, the separation from the nerve node and the creation of a large amount of virus are carried out continuously, without stopping. If the virus particles exceed a certain threshold, they return to the mucous membrane of the oral cavity along the peripheral nerves, where they multiply, which leads to frequent relapses or asymptomatic shedding of the virus. Both mechanisms are valid [1.3].

Today, it has been reliably proven that virions of herpes infection are detected in epithelial cells in the places where the infection occurs most often. As a result of exposure to triggering factors (high and low temperature, sunburn, stress, hormonal changes, medical procedures, alcohol consumption), control over viral replication occurs and the virion directly develops into a mature herpes infection in epithelial cells. there is a possibility of change. The primary symptoms of herpetic stomatitis in patients infected with TORCh are explained by virus activation in the cells in the areas where primary symptoms appear, and the appearance of a cytopathic effect 2-3 days after the onset of the manifestation is explained by the migration of the virus from the nerve nodes. An increase in the amount of the virus in the body leads to the restoration of control over its reproduction and the gradual reduction of recurrences due to the stimulation of all immune systems.

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