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A SPECIALIST'S VIEW ON THE EPIDEMIOLOGICAL DESCRIPTION OF TORCH-GROUP INFECTIONS (REVIEW ARTICLE)

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ABSTRACT

The high comparative weight of TORCH-group infections affects both the demographic condition and the health of the mother and child. The most medically-socially significant specificity of TORCH-group infections is their destructive effect on all organs and systems of the fetus and, above all, its central nervous system, this effect increases the risk of miscarriage, stillbirth and the child's integrity, the formation of defects in its development and leading to disability. In this regard, among TORCH-group infections, toxoplasmosis, rubella, cytomegaly viral infection, as well as infections that call the simple herpes virus Type 1 and 2 are of particular epidemiological importance.

Key words: TORCH-group, toxoplasmosis, rubella, cytomegaly viral infection.

INTRODUCTION

Epidemiologically, infections of this group are characterized by mortality, as well as a variety of mechanisms, pathways and factors of transmission of pathogens, which makes it difficult to implement preventive and anti-epidemic measures. Since effective means of specific immuno-prophylaxis have been developed only for rubella, the susceptibility of the population to the pathogens of the TORCH group is high. Population immunity is mainly formed as postinfectious and does not always have protective activity. In addition, the possibility of lifelong asymptomatic persistence of TORCH-group infectious agents in the human body

has been shown, and, consequently, the presence of conditions for maintaining the epidemic process for an indefinite period of time [2,27,28]

At the same time, the epidemic process of TORCH infections is characterized by the absence of a clearly defined periodicity, seasonality and cyclicity, since the main target of pathogens are people with immunodeficiency conditions. The presence of asymptomatic forms and the impossibility of differential diagnosis of manifest forms only by their clinical manifestations have caused the official data on the incidence of TORCH group infections to not reflect their actual spread among the population, which makes it difficult to timely make adequate management decisions and carry out preventive and anti-epidemic measures.

These features determine the need to improve the system of epidemiological surveillance of TORCH-group infections among the population, and, above all, among risk groups, which include women of childbearing age, pregnant women and newborns. The basis of supervision should be modern sensitive and specific methods of laboratory diagnosis of TORCH-group infections, which allow timely detection of markers of actively ongoing and latent infection in order to further develop a set of adequate preventive measures [10,17,30].

Modern laboratory technologies for the diagnosis of infectious pathology, based on immunochemical research methods, make it possible to assess the presence, level, and avidity of species-specific antibodies, which makes it possible to dynamically monitor, evaluate, and predict the course of epidemic and infectious processes (Adieva A.A. et al., 2009; Roberts C. et al., 2011) [10].

At the same time, the existing logistical support for laboratory diagnostics of TORCH-group infections does not allow for effective monitoring of risk groups, primarily due to the relative cost of screening research methods. In addition, there are no unified means of computer registration and recording of results suitable for all research methods used. The need to use reagent kits from different manufacturers also affects, since currently none of the domestic companies produces the entire list of kits necessary for laboratory diagnosis of TORCH group infections. At the same time, as practice shows, there is an inevitability of certain discrepancies in the results of the analysis of the same pool of samples with the use of test systems from different manufacturers.

Thus, the problem of the discrepancy between the needs and possibilities of surveillance is urgent and requires a systematic solution by developing scientific, methodological and organizational foundations for improving its diagnostic component, which allows us to assess the real situation of TORCH group infections.

Toxoplasmosis is a zoonosis called simple Toxoplasma gondii, which parasitizes intracellular space. Toxoplasmosis in adults usually shrinks without signs, but congenital pathology can lead to abortion, stillbirth of a child or severe neurological damage [1]. The path of transmission of the pathogen is usually alimentary. The main owner of the parasite is cats, which are invasive, eating infected mice and other animals. Also agricultural animals can be carriers of pathogens. For example, in the Republic of Tajikistan, the main source of toxoplasmosis is large and small-Horned moles, the degree of their Toxoplasma damage is 25.7% and 24.6%, respectively; in which the frequency of damage to the population is on average 17.8% [2,23,29].

Cases of infection have been described by human contact, such as when the skin sheaths were damaged during grinding raw meat, as well as when the affected organs were transplanted or, more likely, in a blood transfusion [33].

Of particular importance is the vertical (mother – to-fetus) mechanism with toxoplasmas, in which a pregnant woman experiencing an early phase of primary injury (manifest or asymptomatic) develops placentitis in harmony with a low level of Class G antibodies, as well as subsequent invasion of the fetus with toxoplasmas. This pathway occurs in 40-50% of cases when there is no therapy during pregnancy, while fetal damage usually occurs during the antenatal period. The path of transmission of the pathogen is largely determined by the weight of the Toxoplasma's rejection. So, if the process of infection in Alimentary injury occurs in stages, in parallel with the development of immunity, then in a vertical position-toxoplasmas fall directly into the vascular stream of the fetus, and the invasion takes on a general character from the beginning of the process, and the more immature the immune system of the fetus, the more severe it manifests itself [4, 7,15,28]

The frequency of invasion in pregnant women as well as the toxoplasmosis clinic does not differ significantly from the clinic in non-compatible pregnant women living in the same area. In most cases, the disease is accompanied by no symptoms. If the early phase of toxoplasmosis in a pregnant woman has a manifest character, it is usually mild and can remain undiagnosed. In both cases, the diagnosis of toxoplasmosis can be carried out by conducting systematic laboratory control during pregnancy. On top of that, it is necessary because the main risk of fetal injury is associated with primary female invasion with toxoplasmas during pregnancy, while congenital toxoplasmosis caused by placental transmission always occurs in the form of a general process. Its severity is determined by the infectious dose of the causative agent, the number of protective antibodies dropped

from the mother to the fetus, as well as the period of pregnancy during which the injury occurred [6, 9].

Children and adolescents are likely to develop late symptoms of Congenital Toxoplasmosis. Thus, more than 85% of children who experience invasion without signs develop retinopathy. In other cases, rapid exhaustion, lymphodenopathy, hearing impairment, endocrine disorders are observed. Vascular gravity seizures and mental retardation are detected in a number of cases at the age of 2-4, epileptic seizures - at the age of 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 required to increase the duration and stages of laboratory diagnostics of toxoplasmosis [8, 11,23,33].

Rubella is an air-capillary infection called by a virus with RNA, it belongs to the genus of ruboviruses of the togavirus family. The main source of the causative agent of infection is children aged 7-14 years. The trigger has expressive teratogenic properties. it is known that rubella in pregnant women is extremely dangerous for the fetus, especially in the first trimester of pregnancy. In this process, the injury leads to the general and persistential development of the fetus in 60-85% of cases, which is then accompanied by many defects in development as a multithysmic disease. In this case, children around 3/4 are born with congenital rubella syndrome (tqs): congenital heart defects, cataracts, blindness, deafness, microcephaly, mental retardation, damage to other organs. A very high percentage of perinatal deaths are reported among such infants. Children who have passive immunity from the mother, who are at the first age of their life, form an exceptional group. A bright sign of the epidemiological importance of rubella is the case described in the United States, in which 50 thousand pregnant women with rubella were infected in 1960-1964, which led to the fact that there is a vat of 20 thousand children to be born, as well as 10 thousand children to be born and have a dead child [10, 14].

The incidence of pregnant women in late periods leads to less risk, during this period, fetal damage rarely develops (in 25-30% of cases), and they are less pronounced.

Rubella patients form a stable immunity to their entire life. The development of persistent immunity is also caused by vaccination against rubella. However, there is a possibility of both re-infection and transmission after vaccination. In such cases, rubella usually goes without symptoms and is manifested by a sharp increase in the titers of the antibodies after close contact with the patient. Such cases are more pronounced in individuals who have developed immunity after vaccination than in people with naturally acquired immunity [12, 15].

Cytomegalovirus infection (SMVI) is a disease called cytomegalovirus (SMV) from the family of herpesvirus, the main target of which in the human body is monocytes, macrophages, granulocytes, epithelial and endothelial cells, fibroblasts, smooth muscular cells. Infection in most cases either goes without any clinical signs at all, or only non – specialized symptoms-temperature rise, snoring, inflammatory processes in the nose and mouth-ringum, enlargement of the tonsils of the palate, which can be observed in many diseases of an infectious nature [34].

Compared to other viruses, SMVI is often given from mother to fetus. The frequency of development of SMVI varies from 0.3 to 3.0% of newborns in different countries. In this case, unlike other infections of the TORCH-group, severe fetal damage at SMVI can develop in any trimester of pregnancy [13].

In pregnant women, primary SMVI usually goes without signs, secondary – can also go away as a recurrent infection without signs. In pregnant women, a manifesto evening often gives a clinic similar to mononucleosis or O'RVI. Around 40% of women with primary infection during pregnancy transfer the virus to their fetuses. At least 5% of pregnant women experience reactivation of SMVI, but the number of newborns with these signs does not exceed 1-3% even in underdeveloped countries, most likely it is 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 Child Development leads to cases of miscarriage and death in the fetus or newborn. Late-term injury does not disrupt the structure of the organs and is manifested in the postnatal period in the form of yellow disease, hepatosplenomegaly, thrombocytopenic purpures, lesions of the central nervous system and pneumonia. The infection is actively given through the affected breast milk, which determines 60% of all cases of perinatal infections. Secondary (recurrent) SMVI is the cause of a third of cases of congenital infection in economically developed countries as well as the majority of cases in many developing countries where the percentage of seropositive women of childbearing age is extremely high [16].

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. Congenital infection in babies born at the time of their term occurs in most cases (95%) without signs and negative consequences. In premature babies, especially those born as a result of damage in hemotransfusion – the infection is severe, often accompanied as SMV-sepsis and has a negative prognosis. In almost 10% of children, this results in unilateral or bilateral neurosensory severe earache, lagging psychic development, or impaired movement functions [18].

Damage to blood resipients with SMV is a serious problem, as it is known that between 15 and 40% of children and 2-3% of adults are affected by seropositive donors in blood transfusions. Even more severe problems are associated with organ transplantation, since the factor of the infection being given can be not only the blood poured, but also the transplanted organ [19].

Hepetic infection-type 1 and 2 (VPG-1 and VPG-2) is an infection called by simple herpes viruses, which belong to both the SMV and herpes-virus group and are characterized by tegmental lesions, that is, skin and mucous membrane lesions, damage to the nervous system, as well as other systems in the body. Its manifestations are often associated with immunosuppression, in immunocomprometated individuals it may have a dessiminated, septic dilation. Of particular importance are the innate pathology of the fetus of the causative agent, as well as the ability to call diseases in newborns, damage almost all organs and systems, while the master calls for latent, acute and chronic forms of infection in the body [17].

When the mother has genital herpes, the risk of fetal injury during childbirth is around 40%. The vast majority of people (almost 80%) infect VPG-1 until the age of 6, when people with higher socioeconomic living standards usually become infected later in their lives, while a percentage of older people do not. The location of clinical signs of a herpetic infection is also**,** also quite different by weight of RET-fog. In the I - trimester of pregnancy, it is established that infection leads to the development of micro -, hydrocephalus, heart defects, gastrointestinal tract, venous system, skeletal defects, cataracts, deafness in the fetus. In trimesters II and III, infection leads to hepatosplenomegaly, anemia, yellow disease, hypotrophy, pneumonia, meningoencephalitis, sepsis. The risk of pregnancy interruption is 5 times higher in women seropositive to VPG-2 than in women seronegative women, with high water retention – 10 times higher. Failure to complete pregnancy (premature and late baby miscarriage, undeveloped pregnancy) is noted in 29% of seropositive pregnant women [13].

In the presence of primary simple herpes in a pregnant woman, the risk to the fetus (up to 50%) is significantly higher than in the case when the disease recurs (less than 3%). Perhaps this is due to the protective effect of maternal antiterpetic antibodies. At the same time, these antibodies do not affect the development of clinical signs and the progression of the disease. The highest risk for newborns occurs as a result of the action of VPG-2 in a primary or recurrent genital infection during pregnancy in a pregnant woman. In doing so, the VPG will be in the birth paths and the skin areas that surround it, and the newborn will be infected in the birth phage. In half of patients, clinically expressed perinatal herpetic infection

occurs in a disseminated form, in 30% – in a primary neurological form (meningoencephailt), and in 20% – in the form of skin and mucous lesions [20].

Such an expressive variety of simple herpes clinical forms makes its diagnosis much more difficult. Since urogenital diseases associated with herpes infection have multiple clinical signs, both in character and in intensity (degree of severity) and location of the pathological process, methods of its diagnosis in the laboratory are of paramount importance [21].

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