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CLINICAL AND LABORATORY FEATURES OF THE COURSE OF CONGENITAL CYTOMEGALOVIRUS INFECTION

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Abstract
According to experts' estimates, 30-40 thousand children with congenital CMV infection are born each year in the USA alone, i.e. it is far ahead of all other congenital infections in terms of frequency and significance. The nature of fetal damage, as in other infectious diseases, depends on the timing of infection [3,4,5].

Keywords: Primary infection, pregnancy, infectious diseases, extrauterine life, fetal damage.

Introduction

In the case of primary infection during pregnancy, the incidence of fetal infection averages 40%, and in most cases the child is born with clinical manifestations of infection. If the disease recurs (against the background of existing specific immunity), the risk of infection of the foetus is significantly lower - from 0.5 to 1.5%. If the foetus is infected early in pregnancy, foetal death and spontaneous abortion are possible; in the first 3 months of pregnancy, the teratogenic effect of cytomegalovirus is often manifested. When infected later in pregnancy, the child may have congenital cytomegaly, which is not accompanied by malformations. In this form, symptoms of the disease are detected from the first days of extrauterine life [4,5,6].

Congenital cytomegaly is characterised by intrauterine developmental delay, jaundice, enlarged liver and spleen, decreased platelet count, sometimes up to 50×109 /L, thrombohaemorrhagic syndrome, reticulocytosis and progressive anaemia. There is an enlargement of the liver and spleen, which persist sometimes for a year, while the haemorrhagic syndrome and thrombocytopenia disappear after 2-3 weeks. The intensity of jaundice increases during the first 2 weeks and then decreases slowly, sometimes in a wave-like manner over 2-6 months. In addition to jaundice and liver enlargement, there is an increase in the activity of serum enzymes - ALT, AST, alkaline phosphatase [8,9,10,11,12].

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In the liver biopsy, characteristic cytomegalic cells can be detected. The most severe manifestation of this form is encephalitis, which is practically not found in acquired cytomegalovirus infection [13,14,15,16].

Microcephaly, enlargement of the ventricles of the brain, sensorineural hearing loss and chorioretinitis often develop. Foci of necrosis with subsequent formation of calcinates are more often located in the perivascular zones of the large hemispheres. They are usually detected on CT scan. Microcephaly, hydrocephalus, etc. may result from intrauterine encephalitis. Changes in the central nervous system are often combined with eye damage in the form of chorioretinitis, cataracts, optic atrophy, and kidney damage. The cardiovascular system is much less frequently affected. It is important to note that congenital cytomegalovirus infection is always generalised, whereas acquired infection may be localised with isolated lesions of the salivary glands. If signs of the disease appear 3 months or more after birth, cytomegalia can be considered acquired. Infection of newborns can occur during labour, during blood transfusion, 40-60% of children are infected through breast milk of seropositive mothers. In these cases, the symptoms of the disease are manifested by gradually developing anaemia, lymphocytosis, liver enlargement, interstitial pneumonia is often detected, poor body weight gain. However, a number of authors consider infection through breast milk as a variant of natural primary immunisation. Acquired CMV infection never leads to neurological complications and delayed psychomotor development [16,17,18,19].

The diagnosis of cytomegalovirus infection is made on the basis of clinical picture, virus isolation in cell culture or its detection by immunochemical methods. The diagnosis of cytomegalovirus retinitis is made when typical retinal changes are detected during ophthalmoscopy. It is mandatory to exclude other opportunistic infections accompanied by retinal lesions: toxoplasmosis, syphilis caused by herpes simplex virus and Pneumocystis carinii [20,21,22].

The clinical diagnosis of CMV infection is unreliable. The best way to confirm is isolation of the virus in conjunction with at least a fourfold increase in titre or a persistently high antibody titre. The virus is easily isolated from blood and biological fluids in single-layer cultures of human fibroblasts. If the test material contains many viral particles, as is often the case in generalised cytomegalovirus infection, its cytopathic effect is manifested in a few days. In other cases, such as in mononucleosis-like syndrome, the cytopathic effect can be detected only after a few weeks. To speed up diagnosis, many laboratories apply the virus to single-layer cell cultures, incubate them for 12-24 h, and then stain them with monoclonal antibodies to the cytomegalovirus super-early antigen. Detection of cytomegalovirus in urine and saliva alone does not confirm the diagnosis of current infection, as the virus is excreted into the environment for months and years after recovery, whereas its presence in the blood is not confirmed [23,24,25].

For this purpose, detection of cytomegalovirus super-early antigen (pp65) and its DNA in leukocytes is used, which allows to accelerate the results by several days compared to isolation in cell culture. In the diagnosis of cytomegalovirus encephalitis and polyradiculopathy, detection of virus DNA in cerebrospinal fluid by PCR is used. Serological methods include complement binding reactions, indirect haemagglutination and immunofluorescence methods, and enzyme-linked immunosorbent assay (ELISA). The antibody titre often rises only 4 weeks after infection and remains high for many years. Therefore, a single serological test cannot distinguish between

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current and past infection. Determination of IgM-antibodies to cytomegalovirus is more informative, but false-positive results are possible in the presence of rheumatoid factor in the blood. New molecular genetic methods (including PCR and determination of the virus antigen concentration in the blood) allow not only diagnosing but also monitoring the activity of the infection [1,2,3,4,5,6,7,8,9].

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