Volume 3, Issue 2, February 2025 ISSN (E): 2938-3765

NEW INSIGHTS INTO CYTOMEGALOVIRUS INFECTION

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Abstract
CMVI is an infection caused by a pathogen from the beta-herpesvirus group, characterised by diverse manifestations ranging from asymptomatic to generalised course with severe damage to the CNS and other organs. CMV infection is widespread [5,6,7].

Keywords: Cytomegalovirus infection, risk of infection, foetus, primary infection, secondary infection.

Introduction

The level of infection in most countries of the world ranges from 50 to 90% and varies considerably in different populations, even within the same country, depending on ethnic and socio-economic factors. The greatest risk of fetal infection with CMV and the development of severe forms of the disease is noted when the pregnant woman carries primary CMV infection. The incidence of primary CMV infection in women during pregnancy does not exceed 1%. Intrauterine infection of the foetus with CMV in women with primary CMV infection reaches 40-50%. At the same time, 5-18% of infected children develop a manifest form of the disease with a severe course [1,2,3,4,5]. Transplacental infection in the first trimester leads to CNS malformations, chorioretinitis, heart conduction block. Infection at later terms can lead to the development of progressive jaundice, haemorrhagic syndrome, hepatosplenomegaly, pneumonia. In the future, these children have a high probability (90%) of developing hearing impairment, epilepsy, mental and psychomotor development delay, optic atrophy, various speech disorders. In case of secondary infection during pregnancy, specific immunity factors in the mother (anti-CMV antibodies, anti-CMV T (SD 8)-lymphocytes) provide effective protection of the foetus from infection and development of severe forms of the disease. As a result, the risk of intrauterine infection with CMV does not exceed 2%. It should be emphasised that reactivation of latent CMV infection and superinfection with another strain can only be differentiated by molecular analysis of isolates [16,17,18,19,20].

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Approximately 50% of children with congenital CMV infection have ante- and postnatal signs of the disease: intrauterine developmental delay, microcephaly, hepatosplenomegaly, petechial exanthema, jaundice, chorioretinitis, thrombocytopenia, anaemia. Up to 30% of such children die in the first years of life (three quarters - at the age of 12 months) against the background of increasing disseminated coagulopathy, liver failure (primary cirrhosis), secondary bacterial complications. In 10-15% of cases of congenital CMV infection, subclinical in the newborn period, clinically significant manifestations of the disease develop later - delayed psychomotor development, sensorineural deafness, visual impairment [4,5,6].

The source of infection is a person infected with CMV. After primary infection, excretion of the virus from the body usually lasts several months. If infection occurs during the perinatal period, continuous excretion of the virus lasts 4-8 years. In both adults and children, the state of latent infection may be interrupted by periodic relapses, during which virus excretion begins again. In an infected person, the virus is found in internal organs, blood, liquor, saliva, urine, vaginal secretion, semen, breast milk, and lacrimal fluid. The main mechanisms of CMV infection are contact and airborne [7,8,9,10].

The contact mechanism is realised by natural and artificial routes. Natural routes of transmission of the pathogen are dominant. A susceptible person is infected by direct contact with the source of infection (kissing, sexual intercourse) or indirectly through virus-contaminated utensils, toothbrushes, toys. Because of the low concentration of the virus in secretions and the lability of the pathogen, transmission requires prolonged and close contact. Infection of the foetus is realised by transplacental transmission from mother to foetus, which is possible throughout pregnancy. Primary CMV infection in pregnant women is particularly dangerous for the foetus [11,12,13].

In intrapartum infection, the first clinical manifestations of neonatal disease may manifest after 20 days of life (up to 6 months of age). 12 10-30% of pregnant women are seronegative. The actual incidence of congenital CMV infection among newborns does not exceed 0.2-2.5%, as the risk of foetal infection, severity and prognosis of the disease in congenital CMV depends not so much on the presence of the virus in the organism as on the activity of the infectious process during pregnancy [14,15,16].

In antenatal infection of the foetus, in the vast majority of cases there is a transplacental route of transmission of CMV. In intrapartum infection, the virus enters the body through aspiration, ingestion of infected amniotic fluid or secretions of the mother's birth canal. The newborn may be exposed to CMV infected milk, which is of particular importance in extremely low birth weight infants. Infection of children with CMV in labour or immediately after birth is not usually associated with clinically significant disease, but preterm infants with ENMT react differently, in whom postnatal infection leads to respiratory tract damage (pneumonia, bronchiolitis). At the same time, even with asymptomatic course of congenital CMVI, 5-17% of children may have various health disorders in the future. The artificial route is realised by infection of recipients of blood components and organs with CMV [17,18,19,20].

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