

**FEATURES OF THE COURSE OF CYTOMEGALOVIRUS INFECTION IN INFANTS  
IN THE ANDIJAN REGION**

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**ABSTRACT:** Currently, this topic is again attracting the attention of doctors of various specialties in light of the emerging problems of perinatal pathology and opportunistic infections. Clinically manifest forms of CMV develop in individuals with immunodeficiency conditions (especially AIDS), as well as in newborns due to age-related characteristics of antiviral immunity [1]. Researchers also explain increased susceptibility to cytomegalovirus (CMV) and reactivation of latent infection in pregnant women by the development of physiological immunosuppression. Acute primary CMV infection in a pregnant woman poses a particular danger to the fetus [4]. Congenital cytomegalovirus infection is the most common congenital infection, occurring in 0.6–5% of newborns worldwide and considered the leading nongenetic cause of sensorineural hearing loss in children. The majority of newborns with a manifest form of the disease develop psychomotor and cognitive disorders, and approximately half have visual impairment [6].

**Keywords:** Cytomegalovirus infection, intrauterine infection of the fetus.

**RELEVANCE.**

There are congenital and acquired cytomegaly. Congenital cytomegaly often occurs as a generalized form affecting many organs and systems. Acquired cytomegaly in young children occurs as a mononucleosis-like syndrome, sometimes with predominant damage to the lungs, gastrointestinal tract, liver, or as a generalized form. Both congenital and acquired cytomegaly can be asymptomatic. It is generally accepted that the number of manifest and asymptomatic forms is expressed as 1:10. In addition, according to the current. There are acute and chronic cytomegaly. Infection of the fetus occurs from a mother suffering from a localized form or an acute form of cytomegalovirus infection. The virus enters the placenta hematogenously and causes its damage. Newborns can become infected through mother's milk. However, not all those infected in utero are born with manifest signs of the disease. More often in newborns, the infection occurs latently with giant cell metamorphosis in the salivary glands [5]. With age, the frequency of detection of cytomegalovirus cells decreases while the number of people with antibodies to cytomegalovirus in their blood increases. A humoral immune response is developed and during latent infection, complement-fixing and virus-neutralizing antibodies appear in the serum [4].

Cytomegalovirus (CMV) is a widespread pathogen in all socioeconomic groups and is a DNA virus with high seroprevalence. CMV infection is endemic and has no seasonal characteristics. In developed countries, the prevalence of CMV among women of childbearing age ranges from 50–85%, while in developing countries it approaches 100% [8]. Congenital CMV infection (CMVI) is one of the most common congenital infections, occurring in 0.6–5% of newborns worldwide, being the leading nongenetic cause of sensorineural hearing loss in children and often causing delays in the development of the nervous system [6].

This pathology is considered as a result of transplacental transmission during primary or recurrent infection in the mother, and the risk of transmission to the fetus is 30–35% during

primary infection, and up to 1.1–1.7% during repeated infection [1, 6]. Vertical transmission increases with gestational age, but the risk of fetal infection is greater if infection occurs early in pregnancy. The majority of infected newborns (85–90%) are born without symptoms [7]. The clinical picture is very diverse, the most common manifestations are: intrauterine growth and developmental delay, prematurity, microcephaly, jaundice, petechial rash, hepatosplenomegaly, pneumonitis, hepatitis, sensorineural hearing loss, various neurological symptoms (suppression of unconditioned reflex activity, up to coma, weakly induced reflexes, convulsions) [9]. Paraclinical findings include thrombocytopenia, anemia, leukopenia, isolated neutropenia, increased transaminase levels, direct hyperbilirubinemia, chorioretinitis, and neuroimaging features [4]. Isolation of the virus from urine and/or saliva is the gold standard for confirming infection, and the diagnosis of congenital CMV infection is established based on detection of the virus in the child during the first 3 weeks of life [6].

**Purpose of the study.** Studying the course of cytomegalovirus infection in infants.

#### **Materials and methods.**

During the period from 2021 to 2023, 74 children aged from 14 days to 3 years with various manifestations of CMV were under the supervision of the intestinal infection department of the children's infectious diseases hospital in Andijan and the diagnostic center. To verify the diagnosis, in addition to general clinical tests, biochemical parameters were examined and serological markers of perinatal infections were determined, including hepatitis B, C, and CMV viruses using ELISA. If necessary, CMV DNA was determined by PCR. Etiological diagnosis was carried out using dynamic ultrasound examination (US) of the abdominal organs and neurosonography (NSG). In addition to the usual parameters, the ratio of the lobes, the I segment index, and the histodensity of the liver parenchyma were quantitatively assessed [3]. At the same time, mothers were examined using the same program.

**Results and discussion.** The mothers of all children were infected with CMV and had a burdened obstetric history. During pregnancy, 69 (93.4%) women suffered various forms of respiratory infection, 3 (4.3%) noted malaise and low-grade fever in the 8th trimester. 59 (80%) children were born with low gestational weight and signs of intrauterine malnutrition. In 43 (59%) patients, cutaneous hemorrhagic syndrome was observed after birth. 19 (26%) newborns had respiratory disorders; these children subsequently suffered from frequent bronchopulmonary diseases. The main reasons for referring a child for examination for CMV infection were hepatosplenomegaly, prolonged jaundice, and neurological symptoms with somatic disorders. Children were first hospitalized or referred for gastroenterology appointments between the ages of 14,142 days and 5 months. 59 (80%) of them had a protracted course of newborn jaundice, which began, as a rule, on the 3-5th day of life and persisted until 1.5-2 months of age. The unconjugated fraction of bilirubin predominated. The liver damage occurred according to the type of perinatal hepatitis. Liver function tests were abnormal in all patients. In 48 (65%) children, the number of red blood cells and hemoglobin was reduced to the level of mild to moderate anemia. All children were under the supervision of a neurologist. The most frequently diagnosed syndromes were psychomotor development delay syndrome, hypertensive-hydrocephalic syndrome, and somewhat less frequently, motor impairment syndrome. In NSH, an increase in the echogenicity of the brain parenchyma, its heterogeneity and granularity, perivascular infiltration, and manifestations of mixed or external hydrocephalus were noted [2]. A relative feature of the neurological manifestations of CMV infection was their resistance to traditional therapy (vascular, nootropic, vitamin preparations). A number of children had an undulating course (unexpected resumption of symptoms, increased severity of intracranial

hypertension after a period of well-being), which apparently corresponded to periods of latency and reactivation of CMV. Serological examination of children in the first 3 months of life revealed antibodies to CMV of the IgG class in all cases (65%), and antibodies to CMV of the IgM class were also found in 48 (65%) people. When examining children older than 3 months, IgG antibodies were detected more regularly with an increase in their titer in paired sera. In 28 (40%) patients, blood sera were examined by PCR; in 12 (42%) patients, CMV DNA was detected.

During serological examination of mothers, antibodies to CMV of the IgG class were detected in all cases, and in 48 (65%) - of the IgM class. In the observed group of children, the following clinical manifestations of perinatal CMV were noted: congenital malnutrition, hemorrhagic syndrome, jaundice, hepatitis, neurological disorders, mainly delayed psychomotor development and hypertensive-hydrocephalic syndrome, anemia. Late diagnosis led to late treatment and low effectiveness. Conclusions. 1. A burdened somatic, obstetric and gynecological history, as well as pathological delivery indicate a high probability of intrauterine infection of the fetus. 2. The results of PCR for the presence of TORCH markers in children under 1 year of age revealed a high degree of infection of children born to mothers with chronic latent cytomegalovirus infection.

**Summary.** We have studied for CMV infection in babies. To verify the diagnosis, in addition to general clinical analyzes examined biochemical parameters were determined serological markers of perinatal infections, including hepatitis viruses B, C, CMV by methods IFA. If necessary, we determined CMV DNA by PCR. Etiologic diagnosis was performed using dynamic ultrasound (US) of the abdomen and cranial ultrasonography (NSG). In the observed group of children had the following clinical manifestations of perinatal CMV infection: congenital malnutrition, hemorrhagic syndrome, jaundice, hepatitis, neurological disorders, mainly psychomotor retardation, and hypertension-hydrocephalic syndrome, anemia.

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