

# PATHOGENESIS AND PATHOLOGICAL ANATOMY OF CYTOMEGALOVIRUS INFECTION

Daminov F. A.

DSc, Ass. Professor, Head of the Department of Clinical Laboratory Diagnosis with the Course of Clinical Laboratory Diagnostics of PGD

Karimova M. M.

Assistant of the Department of Patophysiology

Ruziboyeva N. A.

Cadet of the Department of Clinical Laboratory Diagnosis with the Course of Clinical Laboratory Diagnostics of PGD; Samarkand State Medical University

## Abstract

Cytomegaly virus, having entered a previously uninfected organism, penetrates into cells and begins to replicate actively. The result of replication is the formation of daughter viral particles, which leaving the infected cell are 'covered' by the outer shell. In this case, the outer shell of virions is formed with the participation of the cell membrane of the cell damaged by cytomegalovirus. CMV reproduces in lymphocytes, blood monocytes, and persists in lymphoid organs [4,5,6].

**Keywords:** Low virulence, tropism, lymphocyte migration, salivary gland, infected cell.

## Introduction

The cytomegaly virus, having entered a previously uninfected organism, penetrates into the cells and starts actively replicating. The result of replication is the formation of daughter viral particles, which leaving the infected cell are 'covered' with an outer shell. In this case, the outer shell of virions is formed with the participation of the cell membrane of the cell damaged by cytomegalovirus. CMV reproduces in lymphocytes, blood monocytes, and persists in lymphoid organs [7,8,9,10,11,16,17,18,19].

The virus has low virulence and a pronounced tropism to the epithelial cells of salivary gland ducts, where it multiplies slowly without cellular damage. Infection of salivary glands by CMV occurs as a result of transepithelial migration of lymphocytes and histiocytes. Up to 10,000 viral particles can accumulate in lymphocytes, macrophages and epithelial cells, forming intranuclear inclusions. CMV-infected cells hypertrophy, and their nuclei increase in size. As a result, the infected cells acquire the 'owl's eye' appearance typical of cytomegaly - an enlarged cell in which the protoplasm is only visualised as a very thin band due to the large diameter of the nucleus. After infection, CMV is present in the body in a latent form, mainly in peripheral blood mononuclear





cells, periodically reactivated. CMV is characterised by considerable antigenic diversity [12,13,14,15].

Therefore, when a seropositive person is infected with another strain of CMV, the formation of specific immunity against this pathogen will occur as in the case of primary contact. It should be noted that previously developed type- and group-specific antibodies to other CMV strains will restrain active virus replication. However, effective immune defence (sufficient level of specific anti-CMV-ATs and specific killer cells) will be formed only by 14-28 days from the moment of infection with this virus strain [7,8,9,10,11,12,13]. CMVI is primary if infection with cytomegaly virus and the development of the infectious process occurs in a previously seronegative patient. If a seronegative pregnant woman is infected with cytomegaly virus, due to transient immune features, a more active replication of CMV accompanied by viraemia is possible during this period [16,17,18,19,20,21].

Viraemia, especially in conditions leading to a breach of the placental barrier, favours transplacental transmission. The virus that enters the foetus actively replicates and spreads in the body, due to the slow increase in the concentration of specific Ig G (capable of transplacental penetration) in the mother's blood and the immaturity of foetal immunity. The degree of fetal damage depends on the intensity of virus multiplication and the gestational period during which the infection develops. Accordingly, both minimal manifestations of the disease (asymptomatic, subclinical forms) and severe lesions are possible [20,21,22,23].

In secondary infection (reactivation of latent CMV infection or reinfection with a new CMV strain), CMV replication occurs under conditions of 'immunological pressure', the intensity of viral replication and the degree of viraemia are significantly restrained due to the presence of species- and group-specific antibodies. This determines a much lower risk of transmission to the foetus, as well as a milder course of CMV infection in the foetus and newborn (the foetus simultaneously with CMV receives from a seropositive mother and antibodies that prevent virus replication and limit its spread) [1,2,3].

Pathological changes in CMV infection may have varying degrees of severity and may be local or diffuse. Histopathological changes range from focal parenchymatous or periventricular necrosis to microglial nodules (gliosis) and scattered cytomegalic cells. Necrotic changes in the initial stages are accompanied by inflammatory infiltration consisting of circulating mononuclear cells, mainly monocytes. Severe CNS lesions due to early viral dissemination are more common in infections in early pregnancy. The probable reason for this is the higher sensitivity of neurons undergoing differentiation to CMV replication during the first trimester of gestation, in contrast to already differentiated neurons, which are more resistant to CMV infection late in pregnancy [4,5,6].

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