**MODERN UNDERSTANDING OF DIAGNOSIS AND TREATMENT OF INFECTIOUS MONONUCLEOSIS IN CHILDREN**

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Infectious mononucleosis (IM) is the most common, characteristic clinical manifestation of herpesvirus infections. According to ICD - 10 in the block "Infectious mononucleosis" (B27) includes: gammaherpesvirus mononucleosis, which causes Epstein-Barr virus (WEB), cytomegalovirus mononucleosis, mononucleosis of unspecified infectious nature. However, most scientists associate this disease with Epstein-Barr virus (herpesvirus type 4). In the world, infectious mononucleosis affects 16 to 800 people per 100,000 population annually. According to the WHO, more than 50% of children in the first 10 years and 80-90% of adults have virus-specific antibodies as a marker of previous infection. The level of infection in the adult population of Ukraine is almost 100%, and in children - more than 50%, with 50% of them having a recurrent course of the disease.

**Actuality** of the study of infectious mononucleosis is due to the high circulation of the pathogen among the population, the specific tropism of herpesvirus to immunocompetent cells, lifelong persistence of the virus in the body and often latent course.

New research has shown that Epstein-Barr virus can be a trigger for many hematological and oncological diseases, such as thrombocytopenia, agranulocytosis, autoimmune hemolytic anemia, acute leukemia, nasopharyngeal carcinoma, Burkitt's lymphoma and lymphoma.

Traditionally, the diagnosis of IM is based on clinical and hematological changes. Currently, the specific diagnosis of IM is to use PCR to determine the DNA pathogen and different classes of specific antibodies (AB) by enzyme-linked immunosorbent assay (ELISA). Epstein-Barr virus has specific antigens: early (EAD, EAR), capsid (VCA), nuclear (EVNA), membrane (MA).

As soon as the virus enters the body, the production of IgM and IgG antibodies against capsid antigen (VCA) begins. In the acute form of IM, early antigens appear: diffuse (EAD) antibodies disappear after 6 months, and localized (EAR) - persist for several years after the transferred IM. Nuclear antibodies (EVNA) are detected 1-6 months after the onset of IM, the titer increases during recovery. The method of immunoblotting is also used, it allows to determine antibodies to individual antigens of the pathogen. Of great importance is the study of avidity of the Ig class IgG (strength of binding of antigen to AB). At primary infection, AB with low avidity (avidity index (IA) less than 30%) is first synthesized. For the late stage of primary infection is characterized by AB with moderate avidity (IA-30-49%). Antibodies with high avidity (IA - more than 50%) is detected in 1-7 months after infection with WEB. Serological markers of the initial phase of IM are AB class Ig M to VCA, Ig G to EA. An indirect sign of infectious mononucleosis is an increase in the content of aminotransferases (ALT, AST) and organ-specific liver enzymes (LDH-5, urokinase).

Treatment of IM is mainly based on symptomatic therapy. At a high fever appoint antipyretic drugs (paracetamol, ibuprofen). In severe disease, the appointment of glucocorticosteroids in a short course is justified. The appointment of antihistamines is not justified due to the fact that the appearance of such a complication as exanthema is not associated with Ig E-dependent immune response.

Etiotropic therapy with acyclic nucleosides is theoretically justified, as the virus in the phase of the lytic cycle (which occurs in acute productive infection) secretes thymidine kinase. With this enzyme, acyclic nucleosides are converted from the inactive form of the prodrug to the active form, which disrupts the synthesis of viral linear DNA. The use of antibiotics is justified in the layering of bacterial flora, as well as in the development of complications. When prescribing antibacterial drugs, cephalosporins or macrolides should be preferred.

**Conclusions.** The key to success in the treatment of IM is timely diagnosis, correct and strictly individual approach to both etiotropic and pathogenetic therapy, as well as timely hospitalization of patients with severe form of IM.