FEATURES OF IMMUNE STATUS INDICATORS IN HERPES INFECTION

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Introduction. The clinical outcome of primary herpes infection (HI) is largely determined by the immune status of the individual. At the same time, it should be noted that the nature of pathogenetic changes in patients with herpes is largely influenced by the ability of the viral genome to integrate into the host cell genome, particularly in paravertebral ganglia, as well as the tropism of HSV and other herpesviruses for blood cells and immune cells (erythrocytes, platelets, granulocytes, macrophages, lymphocytes).

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These mechanisms contribute to the lifelong persistence of HSV structures in the human body and underlie alterations in both cellular and humoral immunity. Moreover, today HI is considered an infectious (acquired) disease of the immune system, in which prolonged viral persistence, in some cases, is accompanied by productive HSV infection in nearly all types of immune system cells. This manifests as functional insufficiency of these cells and contributes to the development of immunodeficiency [1,2,3].

Other researchers have also demonstrated that cellular immune mechanisms play a primary role in the formation of anti-herpes immunity. The state of these mechanisms largely determines both the outcome of primary infection and the frequency and severity of recurrences. The duration of immunodeficiency during viral infections depends not only on the properties of the virus itself but also on the type of immune response mounted by the patient [11,12,13,14,15].

It is believed that a relatively high level of specific antibodies in the blood of patients with recurrent herpes stabilizes viral persistence but does not prevent recurrences. Studies have shown that in severe forms of recurrent herpes infection (HI) with involvement of the skin and mucous membranes, the disease occurs against the background of high titers of complement-binding antibodies, along with a decrease in the quantitative content of leukocytes and lymphocytes in peripheral blood, and an

altered ratio of lymphocyte subpopulations [16,17,18,19].

Numerous researchers have demonstrated that in HI, the total number of CD3+ cells (total T lymphocytes) and CD4+ cells (T-helper cells) is reduced, along with a decreased immunoregulatory index (CD4:CD8). The activity of natural killer (NK) cells and antibody-dependent cellular cytotoxicity (ADCC) is diminished, and the ability of leukocytes to synthesize endogenous interferon is suppressed. These changes are characteristic of patients with moderate to severe recurrent herpes. They are most pronounced during the relapse phase, while in the remission phase, positive dynamics in the studied immunological parameters are observed, although they remain below normal values [4,5,6,7,8,9,10].

We conducted a comprehensive immunological examination of 148 patients with genital herpes (GH) caused by HSV. Among them, 42% had severe GH (recurrences at least once per month), 33% had moderate GH (1 recurrence every 2–3 months), and 25% had latent GH.

Monoinfection with HSV was observed in 22% of patients, while in the remaining cases, various microbial associations were detected.

Peripheral blood analyses included determination of immunocompetent cell counts (CD3, CD4, CD8 for T cells and CD22 for B cells), lymphocyte proliferative activity in response to T- and B-cell mitogens (RBTL), NK cell activity, antibody-dependent cytotoxicity of lymphocytes (ADCC), as well as phagocytic and metabolic activity of leukocytes using spontaneous and induced NBT tests (nitroblue tetrazolium reduction). Levels of cationic proteins, lactoferrin (LF), and myeloperoxidase (MPO) were measured, along with ceruloplasmin (CP), transferrin (TF), immunoglobulins, and circulating immune complexes (CIC). Interleukins 1 and 2 were also assessed [20,21,22,23].

The study of monocyte–macrophage system (MMS) activity is of particular interest to clinicians because MMS cells participate in multiple phases of the immune response, primarily interacting with T lymphocytes during antigen recognition and processing. Directly or indirectly activated by T lymphocytes, MMS cells play an important role in the effector phase of the immune response, including cytotoxic activity and phagocytosis of immune complexes.

Important factors of the body’s nonspecific defense, in addition to those mentioned above, are cationic proteins (CPs) of neutrophilic granulocytes (NGs). These include enzymatic CPs (myeloperoxidase, lysozyme, cathepsin B, elastase) and non-enzymatic CPs (defensins, bactericidal permeability-increasing protein, lactoferrin) (Kokryakov V.N. et al., 1989). In the available literature, we did not find studies addressing the dynamics of CPs in patients with herpes infection. In our work, CP activity was assessed using the lysosomal-cationic test (LCT) [24].

It has been established that CPs contained in NG granules perform diverse

functions in the inflammatory response, including acting as inflammatory mediators, nonspecific opsonins during phagocytosis, and modulators of blood coagulation and fibrinolysis, among others. CPs provide NGs with strong antimicrobial potential through their complex effects on microbial structure and metabolism.

Low-molecular-weight CPs, such as defensins, are highly basic surface-active molecules that inhibit HSV replication by acting on the viral envelope and depriving the virus of its membrane-targeting properties. Consequently, they are active against both free virus and virus adsorbed on host cells and serve as factors for viral clearance and infection suppression.

We demonstrated that the acute phase of herpes infection is accompanied by a decrease in the total CP content, as determined by LCT (see Table 11), which may indicate intense NG secretion and active involvement of these cells in the body's defense system. As the infection progresses favorably, CP levels increase, and LCT indicators return to normal [20,21,23,24].

Nevertheless, high levels of myeloperoxidase (MPO) in serum throughout the disease course may indicate persistent NG activation, supported by accelerated NG circulation and faster renewal of the NG pool in the blood. Data from the NBT test and NG phagocytosis further confirm active involvement of NGs in the pathological process, which is accompanied by functional exhaustion of these cells during the remission phase.

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