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Chronic recurrent herpetic stomatitis as a disease of immunity (Literature review)

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Abstract

Modern aspects of the pathogenesis of chronic recurrent herpetic stomatitis have been studied. It is indicated that the selected disease can be described as a chronic multi-link recurrent process with the characteristics of a combined secondary immunodeficiency with damage to the T- and B-links of immunity, oppression of the functional activity of NK cells and monocyte-macrophage cells, as well as damage to immune-controlling changes in the immune system. In any of the above cases, the herpes simplex virus has the possibility for persistence, mutation and, as a result, latency in the human body.

Keywords: chronic recurrent herpetic stomatitis, herpes simplex virus, herpesvirus infection

Recently, there has been an increase in the number of infections of a bacterial, viral and fungal nature due to an increase in immunodeficiencies, namely allergic and autoimmune diseases. Slow chronic inflammatory processes which are associated with the presence of infectious agents of a viral nature represent a major medical and social problem. (1-4)

Herpes infection is the most common infection. Among infectious diseases that cause death, diseases caused by the herpes simplex virus (HSV) are in the second place (15.8%) after influenza (35.8%). (5)

The cause of mortality in cases of illness with these viruses is that they begin with an inflammatory process, but over time they can turn into a disseminated form due to a change in the reactivity of the body. Currently, it has been proven that HSV is involved in carcinogenesis, that it is the cause of secondary infertility, and affects the nervous system and internal organs. (6) HSV, which is widespread in nature, is the causative agent of such an infectious disease as herpetic stomatitis. The source of infection can be a sick person or a virus carrier. (3, 7, 8, 9)

An equally important feature of the virus under study is its tendency to a long and often recurrent course, which is associated with the location of the virus in the nerve ganglia in the form of viral DNA, which gives us the right to deny the existence of a treatment method that could completely eliminate HSV from the human organism. (10, 11)

It is believed that herpesvirus infection is a disease of the immune system due to the fact that the organism's immune system has a direct effect on the course of the infectious process by increasing or decreasing the activity of its components. (12)

A relapsing form of herpesvirus infection can occur in any age group, since the clinic of primary herpes was previously observed (13). The clinical manifestations of

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a recurrent form can be variable in nature - from asymptomatic to ulceration of wounds. (14) The relapsing form can also occur with an unexpressed general infectious syndrome, which is associated with the presence of antiviral antibodies in the blood serum. (13)

The most important goal of the treatment of herpesvirus infection is to achieve the most rapid relief of clinical manifestations and stable, long-term remission of the disease. Researchers studying the problems of herpes infection claim that a decrease in the organism's immune resistance leads to an exacerbation of chronic herpetic stomatitis, which leads to the maintenance of immunity using immunocorrective therapy based on interferon. (15)

The most preceding process in HSV infection is the formation and release of anti-inflammatory cytokines, which affect the activation of specific immunity. This is due to the abundance of macrophages and T-lymphocytes in recurrent wounds of the skin and mucous membrane of the lining of the mouth, which promote the secretion of cytokines. (16)

Cytokines are mainly involved in promoting homeostasis of the nervous system through the repair and protection of neurons from damage. An important role is played by cytokines in the withdrawal of symptoms of the disease by using anti-cytokine antibodies, which indicates their leading role in the pathogenesis of the chronic course of HSV infection. (17)

In immunity, which has an antiviral effect, TNF-α occupies an important position, the effect of which is enhanced by interferon, which is a proven fact (18). One of the conceivable mechanisms of the collaborative action of TNF-α and IFN-γ is the ability of interferon to control the expression of TNF-α receptors.

T-lymphocytes are activated with the help of TNF-α. The latter increases the proliferation of T-lymphocytes which are reactivated by antigens and lectins by enhancing the expression of the IL2 receptor. In addition, the role of the most important and effective factor belongs to TNF-α, as it is triggering mechanism of apoptosis, which is important in viral infections. (19)

A reduction in the formation of stimulated INF-γ in the cytokine and interferon positions is recorded in the case of a relapsing form of herpetic infection, which indicates the exhaustion of the antiviral defense mechanisms, and a tendency to overproduction of IL-4, which confirms the presence of an inflammatory process. (20) Excessive production of stimulated INF-γ in atypical herpesvirus infection is a consequence of small but stable antigenic stimulation. (21)

The frequency of relapses is interrelated with the stage of reduction of the formation of IFN-γ by lymphocytes and the cytotoxicity of NK cells (22). The duration of the acute stage of inflammation is completely subordinate to the synthesis of IFN-α. A direct relationship was recognized between the clearance of damage of the IFN mechanism, the cytotoxicity of NK cells and the degree of the course of the inflammatory process, in the forefront of the frequency of relapse. It was found that

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in patients with infrequent relapses (1-2 times per year) and in the proportion of patients with an average frequency of relapses (up to 3-4 times per year), immunity lesions have a transitional temperament. Deep distortions of the immune system, coinciding with a secondary immunodeficiency state, are noted with repeated demonstrations of HSV (at least 6 times a year). (23)

An antinomical type is observed in patients with HSV infection, along with a normal response to the triggering of HSV according to the cellular pattern, when the humoral link is triggered as the response to the escalation (22,23).

The mechanism of the tactics of antibodies against infected cells is closely interrelated with the suppression of the release of the virus into the environment, which leads to a reduction in the expansion of the virus, but at the same time does not rid the human organism of the virus. (23,24)

Regardless of the high content of specific antibodies in saliva and blood, restarting HSV again causes a recurrence of the disease. Humoral immune mechanisms connect extracellular viral particles, inhibit its receptors, change the physicochemical features of the external structure of the virion. As a result, the virus loses its ability to adsorb on a sensitive cell and diffuse into it. In addition, there is a conurbation and opsonization of virions, which help and accelerate phagocytosis and also push the formation of interferon by lymphocytes. (24)

Most scientists associate the defense mechanism with the triggering of complement-mediated lysis and antibody-dependent cellular cytotoxicity against infected cells. A high level of antibodies of a specific series of viruses will not be able to provoke the inactivation of a viral infection. (25) Perhaps the degree and rate of specific humoral immunity is a factor in analyzing the prevalence of the virus. (26) Exacerbations of the disease constantly occur in conditions of triggering humoral immunity. The degree of its index depends on the number of relapses of the disease. Some scientists believe that humoral immunity plays an important role in reducing the number of recurrences of infection, but does not eliminate it. (27)

Along with this, there is an unusual judgment that explains the importance of immunoglobulins in the pathogenesis of a viral disease. (28) It is based on the fact that a consequence of each demonstration of the disease is a radical increase in antibody titers. Regardless of the high content of immunoglobulins in the serum and outside the start of the viral process, with relapses, antibodies do not protect against exacerbation, but they also play a contradictory role, appearing in the formation of pathological immune complexes or the development of an allergic reaction. Rarely, antibodies do not provide a protective effect, but, on the contrary, are involved in stimulating infection, focusing on the cells of the organism. A particle of immunoglobulins G in the structure of NK cells can nonspecifically bind to cells through receptors for the Fc fragment and create the closest bonds with the cell surface and the envelope of the virus, facilitating the penetration of the virus into the cell. (29)

Consequently, in patients with a chronic form of recurrent herpetic stomatitis, there is a combined secondary immunodeficiency state with a damage of T- and B-links of immunity, suppression of the functional activity of NK cells and cells of the monocyte-macrophage link, as well as damage of immuno-control changes in the immune system. Hence, in the chronic course of herpetic stomatitis, the immune response turns out to be numerically and qualitatively imperfect, and may also not be fully specific. Ultimately, HSV has all the resources for persistence, mutation and latency in the human organism. (30)

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