MORPHO-FUNCTIONAL CHANGES IN ACUTE FORMS OF APHTHOUS STOMATITIS

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Abstract: *Disorders of endothelial function in CRAS are systemic in nature. In this regard, identifying violations of the adhesive properties of the vascular wall during the development of CRAS is of significant scientific and practical interest. Studying the content of soluble molecules of cell adhesion and migration of leukocytes in the blood serum of patients with CRAS will significantly expand the understanding of pathogenesis, diagnostic possibilities, and improve the prevention and treatment of this disease.*

Key words: *stomatitis, oral cavity, adhesion and migration of leukocytes, diagnosis, prevention, pathogenesis, oral cavity, mucous membrane.*

The modern concept of the pathogenesis of aphthous stomatitis is based on the results of the interaction of genetic predisposition factors and various trigger agents (infectious, immunological, allergic, food). The result of this is the activation of immunoinflammatory processes occurring in the oral mucosa (OM) with the involvement of pro- and anti-inflammatory cytokines, adhesion factors, etc. In the lesions of the OM, there is an accumulation of activated T lymphocytes and macrophages, which leads to the initiation of synthesis mediators that increase inflammation. After the first cytokine response, a cascade of reactions is activated, leading to predominantly increased synthesis of tumor necrosis factor alpha and proinflammatory cytokines, which leads to a pathological immunoinflammatory

response. An important role in the implementation of immune-mediated stages of inflammation is played by adhesion molecules, the main function of which is to maintain intercellular interactions, migrate cells to the site of inflammation, and initiate an immune response. There are 3 main families of adhesive molecules: selectins, integrins, immunoglobulins. Selectins are expressed on the membranes of leukocytes (L-selectins), platelets (P-selectins) and endothelial cells (P- and E-selectins). Integrins are expressed on the membranes of leukocytes, endothelial cells and ensure adhesion of leukocytes to endothelial cells and extracellular matrix proteins - fibronectin, collagen, laminin, vitronectin. The expression of cell adhesion molecules is induced by proinflammatory cytokines, free radicals, lipopolysaccharides, leukotrienes, histamine, thrombin, complement components and many other factors.

With the help of adhesion molecules (integrins and selectins), leukocytes migrate to the site of inflammation and an inflammatory infiltrate is formed: adhesion (sticking) to the vascular endothelium at the site of inflammation; penetration through the epithelium; movement towards the site of inflammation under the influence of chemotaxis.

The data obtained indicate that in patients with CRAS, an increased content of soluble adhesion molecules can influence the process of movement of leukocytes along the vascular bed, and then directly through the vascular wall into the tissue (focus of inflammation) to realize their effector potential. The above should also be confirmed by the increased respiratory “explosion” of neutrophilic granulocytes in patients with CRAS, the number of which increases. The most important role in the production of membrane antigens is played by the pro-inflammatory cytokine TNF-a, which is secreted at the site of inflammation and ensures the expression of adhesion molecules, thereby mediating the migration of effector cells through the vascular wall and their infiltration of tissues. In this regard, it was of interest to try to discover the relationship between the sequence of adhesive reactions. The specific sequence of leukocyte emigration is due to the fact that the expression of various adhesion molecules does not occur simultaneously. Initially, selectins are expressed

under the influence of inflammatory mediators. Already in the first minutes of action on the vascular wall of histamine, thrombin, bacterial endotoxins (lipopolysaccharide, LPS),phospholipid PAF (platelet activation factor, PAF) redistributes P-selectin from its intracellular depot - granules of endothelial cells (Weibel-Palade bodies) - to the surface of the plasma membrane. After 1-2 hours, under the influence of complement fragments (C5a, Bb), leukotriene B4, TNF-a, leukocyte L-selectins are expressed, and as a result of the action of bacterial LPS, IL-1, TNF-a, TNF-ß, IL-8 and other cytokines - E-selectins, as well as their ligands. Integrins, proteins of the immunoglobulin superfamily and addressins appear on the membranes of leukocytes and endothelial cells much later. In this regard, the maximum rate of neutrophil release occurs in the first 2 hours and decreases significantly after 4-6 hours. Emigration of monocytes begins along with neutrophils, but reaches a maximum after 16-24 hours.

The interaction of selectins with their oligosaccharide ligands is not very strong (low affinity) and is easily destroyed by the bloodstream (reversible adhesion). Selectins attract leukocytes to the vascular wall and hold them for a while, release them and reattach them, which creates the effect of rolling along the vascular wall. Activation of integrins is accompanied by expression on the surface of endothelial cells under the influence of TNF of adhesion molecules of the immunoglobulin superfamily. All this ensures a strong connection of leukocytes with the vascular wall, spreading them on the surface of the endothelium (irreversible adhesion), as a result of which they penetrate through the extended gaps between en

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