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FEATURES OF THE PATHOGENESIS OF INTOLERANCE TO SYNTHETIC DENTAL MATERIALS USED IN ORTHOPEDIC DENTISTRY

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A B S T R A C T

Currently, the prevalence of allergic diseases in different countries is steadily increasing. The study of the mechanisms of the immunomodulatory action of dental materials is a very relevant new scientific direction of modern materials science. This article presents literature data describing the pathogenetic features of the effect of synthetic dental materials used in the manufacture of dental prosthetic structures on the oral mucosa in patients with allergic intolerance.

K E Y W O R D S

acrylic polymers, intolerance, allergic reaction, antigen, pro-inflammatory cytokines.

An adequate biological response of the organism to the used composite material under the necessary conditions of use is called biological compatibility. This mechanism determines the functional relationship between the organism and the material used. Since the tissues of the oral cavity are normally in a dynamic equilibrium state with general and local biochemical and immunological mechanisms, and the relationship between the material, its function and the body is constant, the nature of the body's biological response to the material can also change dynamically [1, 2].

Some dental materials are capable of inducing an inadequate biological response with the subsequent development of pathological reactions. Allergy is one of the types of pathological reactions of the body's immune system [3, 10].

Such a reaction can be caused by a fairly wide range of materials, including dental alloys, filling materials, plastics, etc., used in various fields of dentistry. The possibility of acquiring allergenic properties of dental materials after the combination of their components with the protein components of the individual, forming “haptens”, is not excluded. With prolonged exposure to the body of allergens, there is an indirect increase in the body's sensitivity to these antigens. Subsequently, they acquire the ability to adhere to antibodies or T-lymphocytes - effectors without prior binding to the antigenic structures of the body. Allergens similar in amino acid composition can be found in various kinds of chemical products. In this regard, the process of sensitization to any one chemical substance

can provoke a cross-allergic reaction to other compounds that are similar in antigenic composition[19.28].

Most often, allergens contained in dental materials, such as residual monomer, dyes and stabilizers, cause delayed-type manifestations, while an immediate atopic type of body reaction, on the contrary, occurs extremely rarely. As a result of the allergen entering the body and the formation of sensitized T-lymphocytes, an inflammatory reaction develops, which carries both a protective role and signals a damaging factor [4,7].

Allergodermatosis is a group of immune-mediated skin diseases, which are united by the effect of the allergic component, as a trigger mechanism, on the pathogenesis of the development of all objective symptoms. In the body, in response to relatively harmless antigens entering the mucous membranes of the oral cavity, class E immunoglobulin (IgE) is produced, but if the immune response is disturbed, the pathogenetic mechanism of the type 2 T-helper system (Th2) is activated, which, in turn, leads to excessive production of class E immunoglobulin (IgE) [13,21].

The high-affinity IgE receptor (FceRI) is involved in the binding of allergen-specific IgE to mucosal mast cells. Upon repeated exposure, the antigen indirectly binds IgE on mast cells and causes their degranulation with the release of pro-inflammatory cytokines that induce the migration of eosinophils and Th2 lymphocytes into the mucous membranes. This process of development of pronounced tissue inflammation underlies the clinical manifestations of such diseases as allergic asthma and rhinitis, eczema, etc. [13,21]

With allergic dermatosis, activation of Th2-lymphocytes occurs and the production of IL-4 cytokines is stimulated; IL-5; IL-13, which promote the maturation and activation of eosinophils, and provoke the synthesis of IgE by B cells. Various materials used in dental practice can affect the stimulation of the activity of Th2 cells and, conversely, a decrease in the activity of Th1 cells, which are aimed at suppressing the allergic reaction of the body. Thus, the materials and drugs used in dental practice are capable of inducing or reducing an inadequate immune response to specific allergens in allergic people [14,26].

In people with a genetic predisposition, allergic manifestations develop mainly in certain tissues, in most cases these are the mucous membranes of the upper respiratory tract (allergic rhinitis, bronchial asthma), the mucous membrane of the eyes (conjunctivitis), the hearing organs (serous otitis media), the skin (urticaria, angioedema, atopic dermatitis) [13].

The magnitude of the immune response depends on the amount of antigen affecting the body and on the method of its penetration into the body. With an increase in the dose of a protein antigen and the number of its repeated exposure, the tolerance specific to a given antigen also increases. Increasing the proportion of polysaccharide antigens can desensitize antigen-specific B cells and thereby inhibit antibody formation. Subcutaneously or intradermally administered antigens usually enhance the immune response, and intravenous administration or administration of an antigen through the gastrointestinal tract often leads to specific immune tolerance. Irresponsibility of immunity is associated with the formation of T- (or B-) cell tolerance or with the stimulation of T-cells, which contribute to the inhibition of the immune response [2, 15].

The main mediator of inflammation, histamine, under the influence of specific and nonspecific stimulants, is produced in mast cells and basophils. IgE-mediated histamine release occurs under the influence of specific antigens (anti-IgE, various allergens), but not IgE-mediated histamine release most often occurs under the influence of plant lectins, drugs, complement factors, hyperosmotic

stimulants (radiocontrast agents), polymeric materials and others stimulants acting directly on the cell membrane [4,7].

Basophils circulating in the bloodstream have a morphologically homogeneous structure, however, in the event of an inflammatory reaction and tissue infiltration, their morphological change is possible. Normally, basophils circulating in the blood vessels are found during the late phase of an allergic reaction or during parasitic invasion, in contrast to mast cells, which are not detected in the peripheral blood [23]. Mast cells are less responsive to various stimuli than basophils. Mast cells are believed to play a central role in allergic reactions in tissues, however, the biological significance of basophils is associated with their wide range of mediators, preexisting or newly formed. Mediators are released in response to specific (AG-IgE) and nonspecific stimulation. Basophils predominantly synthesize and secrete histamine and leukotriene LTC4 [22].

In recent years, it has been established that histamine can be synthesized not only by mast cells and basophils, but also by hematopoietic cells that do not have specific histamine storage granules and, therefore, secrete histamine as it is synthesized. [20] For the first time, this type of histamine production by increasing the activity of histidine decarboxylase was demonstrated when myeloid cells were cultivated in a mixed culture of leukocytes and stimulated with concanavalin A, lipopolysaccharide, or staphylococcus enterotoxin [8].

Until recently, it was believed that histamine exerts its effects through three membrane receptors: HI, H2, H3, HI and H2 receptors are present on neutrophils, eosinophils, various immunocompetent cells (T and B cells, monocytes), hepatocytes, chondrocytes. Their expression can change in various pathological conditions such as allergic rhinitis, autoimmune myocarditis, rheumatoid arthritis and atherosclerosis. Through H2 receptors, histamine regulates cell proliferation and immune response, secretion of gastric juice, relaxation of vascular and bronchial smooth muscles, and basophil chemotaxis. [5] H3 receptors are found in the central and peripheral nervous system and are considered as presynaptic receptors that control the release of histamine and neurotransmitters [1] .

This receptor subtype is not found on lymphoid cells. H4 receptors have recently been found mainly on peripheral hematopoietic cells, eosinophils, neutrophils, and CD4+ T cells [12].

The expression of Hl, H2, and even H4 receptors can be modulated during the immune response. Histamine receptors recruit several secondary intracellular "messenger molecules" for signal transduction. Some of them are specific to certain receptor subtypes, while others are general [2].

Until recently, histamine was considered in immunology as a mediator that is associated with allergic reaction processes, but now more and more data are being provided that histamine can also be a modulator of the cytokine profile and a mediator of some biological processes stimulated by hemocytokines. The mediator histamine is involved in monocyte-macrophage differentiation, which is confirmed by the production of this mediator in response to the induction of progenitor cells by the macrophage-colin-stimulating factor [16]. balance of Th1/Th2 cells and the impact on the production of immunoglobulins of different classes during the immune response [Clinical comparison of histamine H1-receptor antagonist drugs. J Allergy Clinic. Immunol., 1996, 98: 307-318].

Jutel M. in 2001 revealed the modulating effect of histamine on the occurrence of an immune response, however, the effect of npo-Th2 histamine on T-cell proliferation predominates, subsequently inhibiting the action of IL-12 and increasing the production of IL-10 through antigen-presenting cells. [Jutel M. et al. Histamine regulates T-cell and antibody responses by differential expression of Hi and H2 receptors. Nature, 2001. Vol. 413. - P. 420-424].

Histamine not only enhances the expression of the co-stimulatory molecule CD86, but also modifies the secretion of chemokines by dendritic cells and also inhibits the synthesis of IL-12[6].

Thus, the extremely important role of histamine in the regulation of the body's immune functions has been proven. Therefore, the determination of histamine released from blood basophils can serve as an important tool for assessing the effect of dental materials on the oral mucosa of patients suffering from allergic dermatosis.

There are several methods for determining the release of histamine from basophils: radioisotope, fluorometric, enzyme immunoassay, gas and mass spectrometry methods [5, 17].

In recent years, with the development of computer technology, the use of various methods for the determination of histamine in clinical practice has significantly increased, especially the fluorescence analysis technique for the diagnosis of whole blood. The principle of operation of this research method is associated with the use of the laws of fluorometry and the special binding of histamine using a glass surface. Diagnosis by this method allows you to determine the released histamine in the range of 5-1000 ng / ml. The advantage of a fluorescent blood test over other methods is: the possibility of accelerated analysis (within 3.5 hours from the moment of blood sampling), a small volume of blood is required for analysis, the possibility of conducting a large number of tests due to automation and computerization of the method, obtaining results in absolute values (ng/ml), not as a percentage.

The fluorometry method allows the use of both standard and non-standard, specific (allergens) and non-specific materials. For the analysis of histamine release from basophils, extracts from foods, drugs, and any other materials suspected of being involved in the release of histamine from blood basophils can be used. [Automated and computerized glass fiber (fluorometric) method for determining the release of histamine from whole blood basophils most effectively assess the processes of histamine-releasing activity of the tested dental materials [11].

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