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ETIOPATHOGENETIC FEATURES OF INFLAMMATORY PERIODONTAL DISEASES

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ABSTRACT
The article presents scientific data concerning the role of various oral factors in the development of gingivitis and periodontitis. The article presents the results of a study devoted to the study of pathogenetic mechanisms of development of inflammatory periodontal pathology. Etiopathogenetic aspects of periodontal disease development can be used in the complex treatment of these types of pathology.
KEY WORDS: *Etiology, Pathogenesis, Gingivitis, Periodontitis, Dentistry, Treatment.*

One of the most pressing problems in dentistry to this day is periodontal disease [9, 17]. Their significant prevalence, pathological changes in the dental system, and negative impact on the patient's body determine the importance of the problem of treating this pathology.

Modern epidemiological data indicate that pathological changes occur as a result of poor oral hygiene, poor-quality dentures and fillings, dental deformities, occlusal trauma, disruption of the structure of the tissues of the vestibule of the oral cavity, medications used, and past and concomitant diseases [16, 17]. Many researchers note the high prevalence of periodontal diseases among the population; with age, the frequency of lesions increases, along with this, the severity of the pathological process in the periodontal tissues increases [2, 3, 5].

Numerous theories of the etiology and pathogenesis of periodontal diseases can be divided into several groups: inflammatory-dystrophic in the first place is the toxic effect of dental plaque; metabolic and alimentary – nutritional and metabolic disorders; endocrine – hormonal disorders of regulatory processes in pathology of the thyroid, parathyroid, pancreas and sex glands; immunological – processes of autoallergy and autoimmunization , disorders of humoral and cellular immunity [1, 17]. For example, it has been established that immunological changes and autoallergization processes develop as destructive changes in periodontal tissues worsen.

In developed forms of periodontitis, the bactericidal properties of blood serum and saliva decrease, and titers of antibodies to altered gum antigens increase. The level of these antibodies fluctuates depending on the severity of the disease [1].

According to other authors, the allergic state of the body, including in periodontal tissues, is determined not by local causes, but by systemic disorders. Researchers have established such disorders of the immune system in periodontitis as neutropenia , suppression of the functional activity of T-lymphocytes, and an increase in their spontaneous blast transformation [1]. Primary damage to polymorphonuclear leukocytes and lymphocytes in periodontitis has been noted [4, 15].

Certain influence on Chronic emotional stresses have a significant impact on the human periodontium [3, 4, 5]. A. S. Grigoryan, Yu. A. Petrovich and co-authors consider the pathogenic influence of stress factors, manifested by the activation of lipid peroxidation, to be very important [4, 14]. Increased lipid peroxidation of cell membrane structures leads to changes in the permeability and osmotic properties of membranes, and, accordingly, to increased activity and the release of hydrolytic enzymes from lysosomes and subsequent cell autolysis . According to the authors, this may be a key link in the pathogenesis of the inflammatory-destructive process in the periodontium.

There is a lot of literature data on changes in the periodontium in dysfunctions of the endocrine system. The most well-studied periodontal changes are in diabetes: 90–93% of patients with diabetes have periodontal pathology [7].

V. N. Kopeikin created the concept of vascular-biomechanical mechanisms of periodontal diseases, which indicates that unidirectional and uniform subthreshold functional loads of teeth can be the direct cause of the pathological process in the periodontium [12, 13].

According to H. A. Kalamkarov , functional overload of teeth plays a certain role, causing a complex of pathological changes in the tissues surrounding the tooth [11]. In this case, destructive changes are local in nature, and inflammatory phenomena are not

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expressed. In generalized periodontitis, functional overload is often a consequence of the pathological process in the periodontal tissues [8, 10]. It sharply increases the pathogenic effect and significantly slows down the rate of reparation [9].

Of knowledge about the etiology of inflammatory periodontal diseases often defines subgingival microflora as the dominant causative factor [6, 9, 16]. The most important factor for the development of periodontitis is supra- and subgingival plaque . The latter is especially important, localized in the space of the gingival groove and on the surface of the junctional epithelium. The microbial flora in periodontitis is diverse and depends on the severity and phase of the disease. Despite the diversity of the microbial landscape, some types of microorganisms are recognized as specific periodontopathogens . Combinations of these bacteria are found in places of greatest destruction of periodontal tissues. These include : gram -negative anaerobic microorganisms of the bacteroides group (P. gingivalis , P. melaninogenicus), to a lesser extent anaerobiospirillum , spirochetes, fusobacteria ;
gram-positive anaerobic bacteria of the actinomycete group (A. naeslundii , A. Viscosus , A. Israelii), to a lesser extent peptostreptococci .

The high pathogenicity of the above-mentioned bacteria is due to their virulence and the characteristics of energy metabolism.
Microorganisms living in the gingival groove and periodontal pockets have acquired highly developed defense mechanisms. Most periodontal pathogens have a high ability to attach and penetrate tissues. Their invasion mechanism is similar to that of enteropathogenic bacteria. This property is a significant factor in the etiopathogenesis of periodontal diseases. The ability of these microorganisms to adhere to the basal membrane is also a predisposing condition for the development of the inflammatory process. The damaging effect of bacteria can be expressed in direct toxic action, causing inflammation and destruction, as well as indirectly, by stimulating immunopathological reactions. To date, it has not been determined which of these mechanisms is the leading one [16]. As a result of damage, activation or release of certain biologically active substances occurs, which determine the rate of development and intensity of the process.

Under the influence of histamine and serotonin, microvessels expand and their permeability increases, which is manifested by hyperemia, exudation, fibrin deposition, and leukocyte migration. Heparin, released during the breakdown of mast cells, reduces cellular metabolism and blocks mitosis and phagocytosis. Prostaglandins increase vascular permeability and enhance edema caused by histamine and bradykinin .

Over time, microcirculation disorders are observed in the underlying tissues, manifested in thromboses and impregnation of vessels and perivascular tissue with blood proteins. First of all, the venous knee of the capillaries is affected. A large number of exchange vessels are switched off, avascular zones are formed [14, 15]. As a result, tissue trophism is disrupted, and the cells switch to anaerobic respiration (glycolysis), characterized by the formation of a large number of toxic products. In addition, bacterial antigens can form immune complexes with antibodies in the gingival pocket, which activate the complement system, which leads to a powerful release of tissue and cellular mediators, activation of the Hageman factor , C3a- and C5a- fractions of complement, platelet-activating factor. Among the products of cell decay and serum proteins in the inflammatory focus, a large number of substances of polypeptide nature are isolated, capable of causing tissue and cell necrosis, increasing vascular permeability and activating leukocyte migration. Under the influence of tissue and bacterial hyaluronidase and edema, the main substance of connective tissue depolymerizes and passes into a sol state, losing its viscosity. Microorganisms and their toxins can penetrate into the resulting microspaces .

It has been established that in ulcerative -necrotic gingivitis, juvenile periodontitis, and some adult periodontitis , electron microscopic examination reveals a large number of anaerobic bacteria in the periodontal tissues, although in most cases plaque bacteria are unable to penetrate the epithelium and connective tissue [17]. The development of the clinical picture of periodontitis is also associated with a progressive decrease in the content of neutral glycoprotein in the epithelium, one of the main components of the tissue barrier. The process of local loss of neutral glycoprotein in the surface layer is considered especially important, which can also be considered a condition that promotes the migration of oral microflora into the epithelial layer [3].
immunofluorescence method revealed that acute and chronic periodontitis is accompanied by focal alteration of the gingival basement membrane, facilitating the penetration of microflora into deeper tissues [17]. As a result, the epithelium of the gingival groove is destroyed , and collagenases and elastase produced by bacteria destroy connective tissue fibers, which leads to the development of an inflammatory-destructive process in the periodontal tissues.

Thus, therapeutic measures in the treatment of periodontitis should be aimed primarily at eliminating the microbial factor and reducing secondary tissue alteration.

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