Modern Ideas About the Pathogenesis of Generalized Periodontitis

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ABSTRACT

In the article, studies on the problem of the etiology and pathogenesis of periodontal diseases using biochemical, histomorphological, immunological, hormonal, radiological and other research methods made it possible to view and clarify a number of pre-existing views on the causes and mechanism of periodontitis, which allows not on ыяly to conduct earlier and earlier studies accurate diagnosis of these diseases, but also to carry out more effective treatment, to outline effective methods of prevention. The analysis of modern domestic and foreign literature allows us to conclude that inflammatory and destructive periodontal diseases are a socially significant and general medical problem due to the high prevalence of pathology among all age groups of the population.

Keywords: chronic generalized periodontitis, treatment, prevention.

Introduction: Currently, periodontal diseases have become quite widespread in many countries of the world, including in our country. According to various scientific and medical literature in the Russian Federation, in many regions the prevalence of inflammatory and destructive changes in the adult population is 80-99%. Over the past 10 years, there has been no tendency to decrease the prevalence of this pathology [3, 9]. According to the latest dental examination conducted in 2007-2008 according to WHO criteria, the prevalence of signs of periodontal tissue damage (bleeding gums, tartar, periodontal pocket 4-5 mm) reaches 80% or more in people 35-44 years old. When comparing similar indicators obtained 10 years ago, there is a tendency to decrease the prevalence of signs of periodontal tissue damage at the age of 12-15 years, however, in the age range of 35-44 years, the indicators remained at the same level. In the age group 65 years and older, more than a quarter of respondents in 2008 registration of indicators of the state of periodontal tissues was impossible due to the absence of teeth [5, 10, 17].

Thus, inflammatory periodontal diseases are not only a medical, but also a socio-economic problem associated with an increase in the number of people with periodontitis and, accordingly, a high percentage of loss of healthy teeth [6, 15]. When studying the etiology of inflammatory periodontal diseases, the focus to this day is on the hypothesis of "Nonspecific plaque infection" first proposed by Zonenvert (1958), and then confirmed by Rosebery (1963). But in an experiment on dogs, it turned out that with an increase in the biomass of dental plaque, periodontitis was not detected in all subjects. Further studies conducted by F. Slots (1978), S. Socransky (1998), W. Loesche (1972), identified a "Specific plaque hypothesis" suggesting the presence of periodontopathogenic bacteria, of which the "marker" microorganisms of periodontitis are Prevotella intermedia, Bacteroides forsythus, Treponema denticola, Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis. It was revealed that the presence of this microbiota causes the occurrence and progression of bone tissue destruction in periodontitis [3, 10, 16]. Research by a

periodontologist professor at Harvard University S. Socransky, in the field of microbiology, made it possible to isolate complexes of microorganisms located in periodontal pockets. Red and orange complexes in high concentrations are associated with periodontitis, and the presence of Aggregatibacter Actinomycetemcomitans indicates the presence of an aggressive form of the disease, the remaining complexes belong to the normobiota [6]. As symptoms increase in chronic generalized periodontitis, dysbiotic changes in periodontal pockets are observed, manifested by a decrease in the content of lactobacilli, vailonella, peptostreptococci and an increase in the number of actinomycetes in association with fusobacteria, opportunistic pathogens, fungi of the genus Candida [6, 12]. To date, it has been established that the occurrence of inflammatory and destructive periodontal diseases is due not only to the presence of pathogenic periodontal microflora, but also to a favorable environment conducive to its reproduction. These are secondary causes, including local and systemic factors of the development of inflammatory periodontal diseases [10, 13].

The main local factors stimulating the formation of plaque are: caries of the crown and root of the tooth, malocclusion, defects in prosthetics and dental fillings, features of the anatomical structure of the soft tissues of the oral cavity and teeth, quantitative and qualitative composition of saliva [5, 9]. Among the systemic factors contributing to the development of periodontitis, the presence of diseases of internal organs, vitamin deficiency and bad habits are distinguished [6]. About 88-100% of patients with reflux disease, as well as gastric and duodenal ulcers have pathological changes in periodontal tissues. The conducted studies scientifically prove that plaque is a reservoir of Helicobacter pylori, from where it can spread to other parts of the gastrointestinal tract. Moreover, against the background of diseases of the stomach and intestines, inflammatory and destructive processes in the periodontium are especially active and have a generalized character [5, 8]. The presence of endocrine pathology: diabetes mellitus, thyrotoxicosis, insufficient ovarian function in women contributes to pronounced changes in periodontal tissues. The influence of chronic renal failure on the course and spread of periodontitis due to the presence of xerostomia and impaired mineralization of bone tissue of the alveolar processes of the jaws has been established [12,14]. The occurrence and progression of inflammatory periodontal diseases is also facilitated by a deficiency of vitamins A, B, C, and E. A number of scientific studies confirm the hypothesis that the vitamin D receptor gene is one of the genetic markers of periodontitis [9, 11]. The effect of smoking on microcirculation in periodontal tissues has been proven. Smokers have vasodilation and a sharp increase in blood flow in the first minute after smoking, and after 30 minutes - vasoconstriction and a decrease in blood flow. In subsequent periods, almost all parameters return to their original values. However, activation of lipid peroxidation contributes to the damage of nucleic acids [3, 8]. Factors such as heredity, gender, age, nationality, socio-economic status play a certain role in the development and course of inflammatory periodontal diseases [2, 8]. It is known that gingivitis precedes periodontitis, and this requires not only certain microorganisms, but also certain reactions of the macroorganism. An important place in the pathogenesis of periodontitis is occupied by a delicate balance between the destructive process caused by the phlogogenic factor and the body's defenses, this is the so-called immunopathogenesis, which is a whole scientific direction - "Osteoimmunology" [1, 6]. The microbiota contained in plaque produces peptidases that determine the ability of adhesion to the surface of the epithelium and its further destruction. Phagocytes, which are the source of inflammatory response mediators, are the first to turn on in response to the action of periodontal pathogens. Histamine, serotonin, bradykinin, interleukins, arachidonic acid derivatives - prostaglandins and leukotrienes regulate the lumen and permeability of blood vessels, as well as the functional activity of phagocytes, thereby playing a key role in triggering inflammation. This process is a non-specific protection of the body. The result of

intercellular interactions is the activation of specific immunological protection, represented by cellular and humoral systems. T - lymphocytes are responsible for cellular immunity. Microbial antigens stimulate the proliferation of T -lymphocytes and their transformation into T-effectors, which are represented by two subpopulations: T4 and T8 (depending on the presence of CD4 or CD8 coreceptor molecules on the cell surface). T-effectors representing type T4 include T-helpers/indicators that secrete lymphokines and B-cell growth factors. The T8 type includes T -killers that destroy cells with an antigen, and T - suppressors that prevent excessive immune reactions by inhibiting the activity of B- and T - lymphocytes [7, 10]. Sensitization of T-lymphocytes to periodontal tissues altered by the action of microbial toxins provides a cellular mechanism of specific damage. Humoral immunity is represented by B-lymphocytes synthesizing immunoglobulins (Ig G, Ig M, Ig A) upon contact with an antigen. As a result, antigen-antibody complexes are formed that activate the complement system, which causes a cascade of interaction of proteins that provide proteolysis [6]. Immune complexes damage the vascular endothelium and perivascular space, which increases inflammation [3, 7]. There are functional connections between immunocytes and bone cells, which may explain the resorption of bone tissue in periodontitis. A key role in inflammatory resorption is played by the balance between RANK - L / RANK (from the English receptor activator of NF - Kb ligand) and OPG (inhibitory protein osteoprosterin). IL1, IL6, IL11, IL17, TNF - a and prostaglandin E2 contribute to the expression of RANK - L protein by osteoblasts and T-lymphocytes. For a certain time, the RANK - L molecule can be bound to the surface of a stromal cell or osteoblast. The precursors of osteoclasts formed from a bone marrow stem cell have receptors (RANK) on their surface that bind to RANK - L, thus forming a mature multinucleated osteoclast. At this stage, the process can be blocked by the free-moving protein osteoproterin, which is able to bind to RANK - L. Consequently, when an imbalance of RANK - L/osteoprosterin occurs in the direction of increasing the synthesis of RANK - L, the mechanism of bone resorption is triggered [5, 8, 11].According to the results of recent studies in the field of osteoimmunology, it was noted that periodontitis will have the fastest, most pronounced, aggressive course if the microbiota is especially virulent and the body's reactions are weakened (immunodeficiency state) [12, 14].

Resume: The analysis of modern domestic and foreign literature allows us to conclude that inflammatory and destructive periodontal diseases are a socially significant and general medical problem due to the high prevalence of pathology among all age groups of the population.

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