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Review Article

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MODERN ASPECTS OF ETIOPATOGENEZ OF THE GENERALIZED PERIODONTAL DISEASE (REVIEW OF LITERATURE)

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ANNOTATION

Now diseases of the parodont represent one of widespread and difficult pathologies in modern stomatology. Approximately at 50% of the population of various regions of the world at the age of 17-60 years various forms of diseases of the parodont are noted. At the same time nearly 90% of the population in the developed countries have gingivit symptoms, for 50% are diagnosed the generalized periodontal disease (GPD) of moderate severity, and for 3% of heavy. According to WHO data (2005), the functional violations of a stomatologic system caused by loss of teeth from parodont diseases develop in 5 times more often than at caries complications, and take the second place on distribution frequency among all dental diseases. At a periodontal disease there is a suppression of regulatory mechanisms in the system of microcirculation of the parodont which extent of frustration depends on severity of a disease and it leads to decrease in lability of microvessels. At easy degree of a periodontal disease inflammatory changes proceed against the background of a spasm the precapillyare of links of the microcirculator course and the increased permeability of a wall of post-capillary faded, being followed by initial rheological changes of blood, developments of stagnation in microvessels and increase in reactivity of endotelialny layer in the venule.

KEYWORDS: A generalized periodontal disease, etiopatogenez the parodontit, microcirculation of blood vessels at the parodontit, lability of microvessels.

INTRODUCTION

Currently, periodontal diseases are one of the most common and complex pathologies in modern dentistry.[26] Approximately 50% of the population of different regions of the world aged 17-60 years have various forms of periodontal diseases -.[45] At the same time, almost 90% of the population in developed countries have symptoms of gingivitis, 50% are diagnosed with generalized periodontitis (GP) of moderate severity, and 3% have severe symptoms.[43] According to who (2005), functional disorders of the dentition system caused by loss of teeth from periodontal diseases develop 5 times more often than with complications of caries, and are the second most common among all dental diseases -[52] It should be noted that the incidence of periodontitis, including GP, according to the world Health Organization, is widespread among people aged 30 to 40-44 years (55-98%), as well as those aged 15-19 years (55-89%). The author obtained information about the prevalence of local periodontal lesions of inflammatory and destructive pathology based on the analysis of accounting and reporting documentation of dental institutions, studying the structure of etiological factors, as well as the frequency of their influence on the development of local lesions, in order to form recommendations for dentists for the prevention of local forms of periodontitis. Studies have revealed the structure and nature of the main causes of the development of local periodontal lesions by an inflammatory-destructive process, among which iatrogenic factors and infectious-toxic factors are in the first place - in case of inflammation of the tooth pulp, depending on the level of quality of dental care provided to the population.

As shown by the analysis of the literature, the current level of scientific research does not fully disclose and justify the mechanisms of violations and criteria for diagnosing periodontal diseases, which determines the relevance of the problem. Information published in recent years in the scientific literature on this problem indicates that chronic inflammatory diseases of the digestive system are associated with lesions of the organs and tissues of the oral cavity and the periodontal itself. According to the authors, in diseases of the digestive system, conditions are created for the occurrence of inflammation in the periodontium, since there is a violation of a number of regulatory mechanisms: immune and endocrine imbalance, endo-toxicosis, violation of microcirculation, neurohumoral regulation, psychosomatic relationships, changes in the metabolism of connective tissue, mineral metabolism, and vitamin deficiency. All this leads to a weakening of the body's resistance and, together with external factors (microbial

colonization of dental plaque), to the development of gingivitis and periodontitis -.[1] More than 30 species of pathogenic anaerobic gram-negative microorganisms belong to the microorganisms that vegetate in the gingival plaque, among which the main role belongs to Porphyromonas gingivalis, Prevotella intermedia, Actinobacillus actinomycetemcomitans, Bacteroides forsutus, Treponema denticola -.[63] The authors proved the etiological role of these microorganisms in the occurrence of periodontitis, their relationship with the severity of the lesion, and established quantitative levels of anaerobic microflora at different depths of the lesion-[38,60,66,69,72]

Most authors consider the microflora of dental plaque to be the most important etiological factor in gingivitis and periodontitis.[16,17] Pathogenic and conditionally pathogenic microflora of the oral cavity is recognized as one of the leading factors in the etiology of inflammatory periodontal diseases.[17] The results of bacteriological studies and the study of dental plaque under the gums indicate the complex composition of the microflora in periodontal diseases. Among the microorganisms there are various types of streptococci, hemolytic Staphylococcus, Trichomonas, fusobacteria, actinomycetes, protozoa, etc. In addition, specific gram-negative bacteria are found in periodontal pockets, such as Porphyromonas gingivalis, Prevotella intermedia, Actinobacillus actinomycetemcomitans, Capnocytophags, Bacteriodis forsythus, Treponema dentiicola, Veilonella recta [Ivanov V. S., 17]. Up to 85% of all microbes are anaerobes, of which 10 to 15 bacteria have pathogenic properties. These microbes, which form a group of periodontopathogenic species, have highly adhesive, invasive and toxic properties and, moving under the gingival margin, damage the epithelium of the dentoalveolar groove. Important virulence factors of these microbes are endotoxins, which are lipopolysaccharides in their chemical composition, which interact with immuno-globulins A, G, and M and various complement components- [Ivanov V. S., 1998]. Exo-and endotoxins disrupt cellular metabolism, cause alterations in periodontal tissues, which contributes to the development of an inflammatory response. The development and course of the inflammatory process in the periodontium, its generalization and chronization are determined not only and not so much by the specific and quantitative composition of the oral microflora, as by the state of the body's own defenses and the response of the immune system. Immune reactivity of the body plays an important, if not decisive, role in the development of inflammatory periodontal diseases. The significance of the defense system (innate and acquired immunity) is associated with the induction of anti-inflammatory expression of tissue cytokines, activation of chemoatractants and involvement of anti-inflammatory cells, with violations of local and systemic metabolism, hemodynamics, immunological and neuro-regulatory disorders, and shifts in microbiocenosis- [Yonemura T. 1989; Watanabe K., et al., 1991; Firatii E., et al., 1996; Siqueira J. F. et al., 2001]. An important role in the starting mechanisms of inflammatory and inflammatory-destructive processes in the periodontium is played by the state of local and General systems of protection and maintenance of homeostasis. Changes in indicators of non-specific reactivity in periodontitis have been found by many researchers. In patients, depending on the severity of the pathological process, the complement titer and the amount of serum properdin, the level of serum lysozyme, and the phagocytic activity of blood leukocytes decreased -.[30,49,57,59] Thus, in moderate periodontitis, changes in the content of lysozyme and immunoglobulins A and sIgA in the oral fluid are observed, which are mutually compensated in nature-[41,60] It is known that in the process of phagocytosis, the degranulation process is also extremely important, during which neutrophil granules are released. These grains contain antiseptic and proteolytic enzymes with pronounced properties. At the phagolizosome stage, they are actively involved in the process of destruction, destruction and "washing out" of periodontal microorganisms, which greatly contributes to the localization and reduction of the rate of spread of microbial infection in periodontal tissues. Therefore, phagocytosis is the main protective mechanism against periodontal infections -.[75] Complement is a complex of plasma proteins circulating in the blood, which actively interact with each other, and after activation, melt down with the membrane proteins of cells, thus providing a powerful antibacterial protection against periodontal disease. The role of components in the etiopathogenesis of periodontal diseases is still not precisely defined. However, it is indisputable that the interaction between phagocytosis, the formation of antibodies and complements provides primary protection of periodontal tissues from pathogens. Despite the fact that individual components of the complement were found in increased amounts in patients with periodontal disease in blood serum, gingival fluid, and gum tissue samples, the concentration of individual components of the complement often differed. A significant role in the protection of the periodontium, especially against pathogens, is shown by the fact that many periodontopathic species of bacteria can disable some components of the complementary system. The pathogen Porphyromonas gingivalis is known to cause degradation of the component part of the complement in the gum groove fluid or their accumulation on bacterial surfaces-[27] In the same way, A. actinomyce - temcomitans probably reduces complement activation by producing Fc binding proteins that are released during microbial growth.[4] the Formation of specific antibodies from periodontal bacteria supports a specific and later type of immune response. Antibodies from periodontal pathogens deactivate various virulent factors of these bacteria, preparing them with various immune responses for effective phagocytosis. To date, the increased number of serous antibodies from periodontal bacteria is estimated as a manifestation of increased activity, i.e.,

the late stage of periodontal disease, during which there was a dissemination of periodontal bacterial antigens in the human body. This occurs when specific bacteria enter periodontal tissues, which leads to an increased inflammatory and immune response. Qualitative changes that involve the transformation of bacterial colonization into bacterial invasion of periodontal tissues are usually accompanied by qualitative changes in the host's immune responses, such changes are both local and systemic. It is known that a specific human immune response is provided by two cell lines: T-and B-lymphocytes. As you know, periodontal pockets are a constant source of bacteria, their toxins and antigens. All these substances can activate Tm-lymphocytes, which develop Th1-lymphocytes responsible for activating macrophages. The activity of macrophages in periodontal destruction is one of the possible pathogenetic pathways of the disease, since macrophages are able to destroy their own periodontal tissues, producing a large number of anti-inflammatory cytokines. Th2 cytokinin profiles (IL-1 alfa, IL-5, IL-6), which indicates the emergence of new B-lymphocytes as a significant part of local cytokines and a possible coefficient of excerbation of the inflammatory process. In another group of patients with periodontal disease, IFN-alpha and IL-6-were significantly increased.[4,42] Deregulation of cytokines and immunoglobulins in places of inflammation leads to significant destructive changes. Periodontal inflammation is represented by plasma cells that directly produce antibodies in an amount of approximately 50% of the total number of B cells. These inflammatory-immunological reactions, caused by inadequate IgG production, contribute to the destruction of periodontal tissues. Increased amounts of IL-1, IL-6, and TNF-alpha provoke periodontal-destroying processes and chemical reactions -.[42] In the literature known to us, there is evidence that the high prevalence of periodontal diseases is associated with the nutritional characteristics of the population -[61,84] Vitamin deficiency plays a certain role among nutrition factors.[71,73] Among the variety of causal factors that contribute to the occurrence of periodontal diseases, significant importance is attached to occupational hazards-.[37,40,44] Workers of industrial enterprises are exposed to the combined action of many adverse factors of the production environment, which cause a decrease in the body's resistance and an increase in the frequency of periodontal tissue pathology.[26,52] In recent years, thanks to the accumulated clinical and experimental data, the role of the oral microflora as an etiological factor in the occurrence of periodontal diseases is recognized by most researchers. The authors present data on pathogens of inflammatory periodontal diseases, especially on the biological film covering the root surface and consisting of a population of bacteria linked by a matrix (polysaccharide complex). According to the authors, the matrix is a product of the life of microorganisms and performs protective and adhesive functions, which determines the complexity of the selection of antibacterial drugs-.[17] Considering modern aspects of the etiology of inflammatory periodontal diseases, some authors conclude that the microflora of the oral cavity and "dental" plaque is only one of the adverse factors that lead to the failure of the body's defenses and the periodontal complex to inflammation of the latter -.[16] According to A. S. Grigoryan (1999), one of the main factors in the pathogenesis of periodontitis is pathogenic microorganisms that vegetate on the teeth and gums. The microflora of the gingival groove, on the teeth in the form of plaque and plaque, with its toxins, enzymes, and antigens, has a toxic and sensitizing effect on the body, changing its reactivity. As a result of violation of local protective factors of the oral cavity against the background of reduced resistance of the body, the bacterial flora can become an etiological factor of inflammatory and inflammatory-destructive processes in the periodontium -.[22] In 80% of cases, the cause of periodontal diseases is the oral microflora, which is a mechanical, chemical and biological irritant of periodontal tissues -.[32,33,47,78] The formation of plaque is affected by the frequency of food intake, its nature, the concentration of hydrogen ions in saliva, its viscosity, the speed of salivation and the process of self-cleaning.[70] Carbohydrates are a source of synthesis of intra-extracellular polysaccharides by coccal microorganisms.[32] The development of pathogenic and non-pathogenic microflora of dental plaque is also influenced by factors such as constant contact with the microflora of the environment, the ingress of microorganisms from food, favorable conditions for the development of microbes-temperature, humidity, and nutrient medium-.[32,35,61,64] Bacterial enzymes play an important role in the pathogenesis of periodontitis. A strong effect on periodontal tissues is provided by proteolytic enzymes that destroy collagen, which can have both bacterial and leukocyte origin-.[47,32,80] All anaerobes and obligate vibrions are able to break down collagen.[40,45] Generalized periodontitis, regardless of the severity of the course, is accompanied by shifts in microbial symbiosis, the manifestation of which is a decrease in the aerobic link, an increase in the General anaerobic contamination, the survival of enterobacteria from periodontal pockets, dysbiosis with a deficit or complete elimination of L. acidophilicus. Lactobacillus deficiency plays a major role in the implementation of the pathogenic action of opportunistic representatives-[33,61,79,85] In the connective basis of the gum, protein-glycosoaminoglycan complexes are destroyed, free amino acids, uranium acids, aminosaccharides, low-molecular polysaccharides, and polypeptides accumulate. Osmotic pressure increases, water retention occurs, edema, swelling, acidosis and hypoxia develop, which in turn initiate the accumulation of lactic and fatty acids. A high level of lipid peroxidation leads to the destruction of cell membranes. The described violations of the structure and function of the connective tissue of the gums are accompanied by a pronounced vasomotor reaction, prolonged expansion of blood vessels. Persistent hyperemia is accompanied by a violation of vascular wall permeability and migration of polymorphic nuclear leukocytes and macrophages into intercellular spaces.

According to the authors, an important factor in the development of periodontal diseases is Tartar, which is found in 80% of people with a healthy oral cavity and in 90-96% of both dental and somatic patients. 3-6 weeks after the formation of plaque, the first signs of mineralization appear in it.[77] At the same time, the microbial cells of dental plaque are neglected, although in some places the initial microbial structure is preserved -.[40] The resulting Tartar has a harmful effect on the gum, on the tissues of the gingival sulcus, and on the circular ligament-.[55,60] It is delayed, injures, infects and allergizes periodontal tissues, causes acute and chronic inflammation, and then destruction-.[33] Tartar, especially located at the neck of the tooth, has a mechanical irritation of the gums, impedes the circulation of gingival fluid, the outflow of exudate from the periodontal pocket, as a result of which the edge becomes inflamed, exfoliates and atrophies. The inflammatory process in periodontal tissues, in turn, can contribute to the deposition of Tartar-.[50] It is known that the metabolism of periodontal tissues is closely related to the biological environment of the oral cavity, i.e. saliva. It plays an important role in preserving the integration of oral tissues, has a huge informative value, and contains components that determine the protective function. Many researchers recognize the state of the vascular bed and the periodontal microcirculatory system as one of the factors in the pathogenesis of periodontal diseases that trigger the pathological process, which is primarily facilitated by the features of the periodontal circulatory system-.[58] In periodontitis, there is a suppression of regulatory mechanisms in the periodontal microcirculation system, the degree of disorder of which depends on the severity of the disease and this leads to a decrease in the lability of microvessels. In mild periodontitis, inflammatory changes occur against the background of spasm of the precapillary links of the microcirculatory bed and increased permeability of the wall of the postcapillary venules, accompanied by initial rheological changes in the blood, congestion in the microvessels and increased reactivity of the endothelial layer in the venules. In moderate and severe periodontitis, inflammatory changes in periodontal tissues occur against the background of pronounced arteriole spasm, significant dilatation of the venular section of the microcirculatory bed, and increased reactivity of epithelial cells in postcapillary venules.[14] In inflammatory periodontal diseases, microvessel changes are predominantly atrophic: arterioles are sharply narrowed, the number of functioning capillaries is reduced by 24-31%, the level of capillary blood flow in the gum is reduced to 29-53%, and this leads to a sharp weakening of microcirculation and trophic disorders in periodontal tissues.

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