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MODERN ASPECTS OF ETIOLOGY AND PATHOGENESIS PERIODONTAL DISEASES

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Abstract
Inflammatory-destructive periodontal disease is one of the most complex and widespread forms of pathology and is the main cause of tooth loss among the adult population. An epidemiological study conducted by the World Health Organization (WHO) in 35 economically developed countries among people aged 31-44 years showed a high prevalence of periodontal diseases - over 75%, which indicates not only a high incidence rate, but also a significant decrease in the age of patients suffering from this pathology. According to E.M. Kuzmina intact periodontium is detected only in 12% of people, and the rest have lesions of varying severity: from initial inflammatory to severe destructive changes with loss of teeth. Severe periodontitis, which leads to tooth loss, is found in 5-15% of cases in most surveyed populations [2, 12].

Keywords: periodontium, etiology and pathogenesis, inflammatory diseases, odontogenic infection.

Introduction:
Some authors believe that chronic generalized periodontitis, despite the sufficient monomorphism of the main clinical manifestations, is an etiologically and pathogenetically heterogeneous disease. Periodontal diseases can develop under the influence of both local causes (plaque microorganisms, occlusal trauma, etc.) and the combined effects of local and general (endogenous) factors against the background of changes in the body's reactivity [13, 14]. It is known that the development of periodontal diseases is influenced by such local factors as excessive consumption of soft food, a decrease in saliva secretion, overhanging edges of fillings, the presence of orthodontic appliances, anomalies in the position of the teeth, abnormal attachment of the frenulum of the lips and tongue, small vestibule of the mouth, traumatic injuries. An adverse effect on the periodontium is caused by dietary disturbances, physical inactivity, pathology of internal organs and systems, changes in water-salt metabolism, immunological, vascular, hormonal and metabolic changes, chronic intoxication and hypoxia [10]. Microorganisms of dental plaque are involved in the occurrence of the inflammatory process in periodontal tissues. It is known that

anaerobic microorganisms with pathogenic properties vegetate in a bacterial plaque. These include Actinobacillus, Actinomycetem comitans, Porphyromonas gingivalis, Bacteroidas forsythus, Campylobacter rectus, Eikenella corrodens, Streptococcus intermedius, Spirochetes, etc. [12, 15]. The pathogenic effect of microorganisms is largely due to their active enzymatic activity. Microbial enzymes are able to increase the permeability of capillaries, cause a violation of the permeability of the epithelial membrane and penetrate into the subepithelial connective tissue base of the gingival mucosa. Collagenase, hydrolyzing collagen, is able to destroy the collagen of the periodontal ligament and bone tissue of the alveolar process. Bacterial hyaluronidase, as a result of the breakdown of hyaluronic acid, contributes to the destruction of the connective tissue epithelium, fibroblasts, expansion of microvessels, an increase in the permeability of their walls, and increased leukocyte migration. The presence of collagenase enhances the local action of hyaluronidase. Along with this, the proteolytic enzymes of dental plaque catalyze the formation of highly active polypeptides - kinins, which cause the main clinical symptoms of gingivitis: increased capillary permeability, development of edema, redness and bleeding of the gums. The destruction of periodontal tissues is caused by groups of proteolytic and hydrolytic enzymes, which, together with the resorption activity of osteoclasts, cause damage to periodontal tissues and alveolar bone, and the germination of the epithelium leads to the formation of a periodontal pocket [12, 14]. Traumatic overload of periodontal tissues plays an important role in the etiology and pathogenesis of periodontal diseases. All organs of the dentoalveolar system are closely interconnected. Occlusion, periodontium, temporomandibular joint, masticatory muscles, nervous system are the main links of this system. Functional overload of periodontal tissues contributes to the development of pathological changes with a predominance of inflammatory and degenerative processes. Destructive-inflammatory processes are observed with malocclusion (deep incisal overlap, deep, open, progenic, prognathic bite, etc.); with early loss of molars and premolars; after the removal of a large number of teeth (post-extraction tooth movement); crowding; improperly formed vestibule of the oral cavity; irrational prosthetics, etc. [13]. Occlusal disorders in the dentition are aggravating factors in the development of inflammatory changes in periodontal tissues, and in some cases occlusal trauma can serve as a trigger for the occurrence of focal disorders of microcirculation processes in the periodontal ligamentous apparatus. Functional overload is accompanied by changes in the hydrostatic blood pressure in the vessels of the blood and lymph flow and, as a result, the appearance of hemo- and lymphostasis, impaired permeability of histohematic barriers, perivascular edema, diapedesis of blood cells, erythrocyte aggregation, embolism and,

vascular thrombosis, which ultimately As a result, it affects the structure of periodontal tissues [13]. Also, the cause of periodontal disease can be functional deficiency (hypofunction), which is a consequence of modern civilization. Atrophic processes develop as a result of the intake of carefully processed, soft food, which deprives the tissues of a full load and contributes to the formation of dental deposits, which are the cause of inflammation of the marginal periodontium. Inadequate functional load reduces the resistance of periodontal tissues to external influences, such as microorganisms, trauma, and contributes to the deposition of tartar, a decrease in local blood flow, which in turn leads to destructive processes. [13]. It is well known that incorrectly placed fillings, overhanging edges of crowns, located at the edges of the gums of the prosthesis clamer are factors that contribute to the deposition of food debris and plaque bacteria. Insufficiently contoured teeth contribute to mechanical irritation of the gingival margin, the ingress of food particles into the gingival sulcus, incomplete adherence of the gum to the tooth surface, and conditions are created for microorganisms to realize their pathogenic properties. Factors contributing to the development of periodontal disease include mouth breathing and bruxism. With oral breathing, there is overdrying of the mucous membrane of the gums, a decrease in resistance to infectious factors, a decrease in the antibacterial properties of the gingival fluid and saliva. Also, the tone of the circular muscles of the mouth and cheek muscles increases, which increases the pressure on the periodontal teeth of the upper jaw and contributes to the development of destructive processes in it. The development of inflammatory processes contributes to the unhygienic condition of the oral cavity. Bruxism contributes to increased abrasion of hard tissues of the tooth, periodontal trauma, impaired microcirculation and, ultimately, bone resorption. According to modern data, common factors play an important role in the occurrence and development of periodontal diseases: atherosclerosis and its complications, hypertension, diabetes mellitus, obesity, etc. [8] So, in patients with generalized periodontitis in 97% of cases, pathology of internal organs was detected, which indicates the relationship between the state of periodontal tissues and the general status of the body [2, 13]. According to scientific studies, cardiovascular, endocrine diseases, pathology of the liver, kidneys, genetic predisposition, puberty disorders contribute to the progression of inflammatory and degenerative processes in periodontal tissues, and the degree of damage to the periodontal complex is the deeper, the more severe and longer the course of somatic pathology [2, eighteen]. It should be said that the age of the patient has a significant role in the development of periodontal diseases, since the number of systemic diseases and drugs used to treat them increases, which leads to a decrease in the effectiveness

of protective mechanisms in elderly patients. With increasing age, there is a decrease in bone density and healing abilities as a result of a slowdown in metabolism [10]. One of the factors negatively affecting periodontal tissues is the use of drugs, which include: corticosteroids, immunosuppressants, hydantoin, heavy metal salts, oral contraceptives, cyclosporine [10]. In modern literary sources, the data presented indicate that metabolic disorders in periodontal tissues, resulting from a number of exogenous and endogenous influences and dysfunction of enzymatic systems, cause a violation of the blood supply to the periodontium. Blood microcirculation plays a key role in providing tissue trophism and compensatory processes in the development of inflammatory and ischemic lesions of periodontal tissues. The circulatory organs supply periodontal tissues with oxygen, nutrients, and remove waste products from them. But a decrease in stroke and minute volumes of the heart and arterial hypotension can lead to a drop in perfusion pressure in the vessels of the musculoskeletal apparatus of the tooth, and as a result, the development of dystrophic changes in them. The latter cause a decrease in the resistance of periodontal tissues [2, 9]. The results of scientific research have shown that there is a clear relationship between the blood supply to periodontal tissues and the reactivity of the cardiovascular system. The severity of compensatory reactions on the part of the latter is inversely related to the severity Pavlova, No. 3, 2013 156 compensatory reactions of the periodontal vascular bed [2, 9]. In general clinical practice, diseases associated with a violation in the hematopoietic system are often encountered. Among them, anemias are often observed, both congenital and acquired: aplastic, hemolytic, iron deficiency, etc. Anemic conditions are combined with a violation of blood coagulation, pathology of the heart, blood vessels, other organs and systems, which affects the state of the dental system and, first of all, tissues periodontal [7, 21]. According to the literature data, chronic generalized periodontitis occurs in almost all patients with peptic ulcer of the stomach and duodenum. The pathogenesis of periodontal tissue damage in peptic ulcer is not fully understood. However, it has been established that in patients with chronic generalized periodontitis, especially severe and moderate, the phenomena of endotoxicosis develop, confirmed by the dynamics of such hematological indicators as the leukocyte index of intoxication, the hematological index of intoxication, the sorption capacity of erythrocytes, the electrokinetic properties of the nuclei of buccal epithelium cells, etc. [8] . The commonality of innervation and humoral regulation of periodontal and gastrointestinal tissues create prerequisites for the mutually aggravating course of the pathological process in the oral cavity and stomach. Inflammatory changes in periodontal tissues and stomach occur under the influence of common factors of aggression: bacterial infection (in

particular Helicobacter pylori), diffuse neuroendocrine system, genetic factors that cause the development of apoptosis. Noteworthy is the information that the eradication of H. pylori in the stomach and oral cavity against the background of its complete sanitation contributes not only to achieving remission, but also to restoring the structure of the mucous membrane of the digestive tract (stomach) within 2-3 years. Inflammatory periodontal diseases against the background of chronic gastritis are accompanied by a violation of the processes of cell renewal of gingival epitheliocytes, which may be due to the direct or indirect influence of serotonin, melatonin, histamine and Helicobacter pylori infection on the processes of cell proliferation [11]. A large number of studies are devoted to the study of the relationship between periodontal diseases and endocrine pathology. Periodontal diseases have been noted with hypo and hyperfunction of the thyroid gland, parathyroid and gonads. Changes in the periodontium in diabetes mellitus have been studied in the most detail [1]. In patients with diabetes, periodontitis occurs in almost 100% of cases and is characterized by an aggressive course. This pathological process has its own unique morphological structure, which is significantly different from inflammatory periodontal disease in individuals without diabetes mellitus. At the same time, the frequency and severity of the pathology is directly correlated with the duration of carbohydrate metabolism disorders [1]. According to scientific studies, an increase in the concentration of glucose in saliva, periodontal fluid and a decrease in salivation (up to xerostomia) can adversely affect the nature of the bacterial flora, increasing the process of non-enzymatic glycation of proteins: inflammatory mediators, immunoglobulins and other mediators of immune defense, as well as cells involved in oral immune defense. This process leads to a decrease in immune defenses. In the pathogenesis of periodontal syndrome in diabetes mellitus, periodontal angiopathy is of primary importance: the lumen of the vascular bed does not disappear, but the vascular wall is affected. Pathological changes in blood vessels are reduced to primary plasma damage to the basement membrane of the microvascular bed, and then lead to sclerosis and hyalinosis of the wall. According to scientific studies, these changes have nothing to do with inflammation. Therefore, microcirculatory changes in diabetes mellitus are primary [1,2]. Among the systemic risk factors that lead to a decrease in body resistance and create a predisposition to the occurrence of periodontal diseases, rheumatoid arthritis should be noted. The pathogenesis of rheumatoid arthritis is characterized by pronounced heterogeneity, however, the generally recognized leading role belongs to combined changes in cellular and humoral immunity. According to the results of scientific research, it was found that the clinical manifestations of generalized periodontitis are due to the form

and variant of the course of rheumatoid arthritis. Pronounced inflammatory and destructive changes in periodontal tissues, characteristic of generalized periodontitis I-II degree, II degree with an aggravated course of moderate severity were detected only in patients with systemic manifestations of rheumatoid arthritis. In patients with generalized periodontitis associated with the articular form of rheumatoid arthritis (seronegative variant), clinical signs of a chronic course prevailed, in which destructive processes in the alveolar bone were the leading ones with less pronounced signs of inflammation in the soft tissues of the periodontium [5,]. Of great importance in the development of periodontal diseases belongs to the deficiency of vitamins C, B, A, E, D, which affect the condition of its tissues. With a deficiency of vitamin C in the body, the processes of formation and formation of collagen fibers are disrupted, tissues loosen, the permeability of the intercellular substance and capillaries increases, the formation of bone tissue slows down, and the resistance of periodontal tissues to infection decreases. A lack of vitamin A leads to a decrease in the barrier function of the gums (alteration of the gingival margin), thereby contributing to its inflammation. Vitamin E deficiency enhances free radical lipid peroxidation, periodontal pockets are formed, bone tissue atrophy, changes in the periodontal vascular system. Vitamin D deficiency leads to the development of osteoporosis, which manifests itself in children with osteoporosis, and in adults with osteomalacia [8]. It was revealed that one of the factors in the development of inflammatory periodontal diseases in young people is disadaptation to chronic psycho-emotional stress. Excessive vegetative support of activity contributes to the occurrence of inflammatory periodontal diseases and, just like neurotic character traits in these individuals, is a manifestation of maladaptation to psycho-emotional stress. In a state of emotional stress, people's habits change, the consumption of tobacco, alcohol, drugs increases, sleep and nutrition disorders appear, and oral hygiene worsens. All this significantly weakens the body's defenses and, against this background, local pathogens that contribute to the development of inflammation are more easily activated [7, 18]. One of the risk factors for the development of periodontitis is smoking, which contributes to a greater degree of destruction of periodontal tissues and slower healing after treatment. The combination of smoking with the genetic factor IL-1α increases the frequency of transition from gingivitis to periodontitis by 3-7 times. The impact of nicotine changes the composition of the subgingival microflora, inhibits protective mechanisms that contribute to the elimination of periodontal pathogens (reduces the amount of gingival fluid and saliva, inhibits the functions of the most important cells of the defense system - neutrophils, monocytes, macrophages, dendritic cells). Violation of blood flow and metabolic disorders that

accompany anemic conditions contribute to the development and increase in the aggressiveness of periodontopathogenic microflora [4, 6]. It follows from the above that periodontitis is closely related to general somatic pathology, however, foreign authors noted that chronic odontogenic infection is an important factor in the development of somatic pathology. Studies have shown that the focus of infection in the oral cavity is often associated with the pathology of internal organs: chronic bronchitis and bronchial asthma, arthralgia, arterial hypertension, damage to the blood system and many other diseases. [10]. The pathogenic effect of the stomatogenic focus is associated with the fact that it is a source of hetero- (microbial, medicinal) and autoantigenic persistence, and also has a depressing and disorganizing effect on the immune system. Chronic foci of infection in the oral cavity make a significant contribution to the development of immunological imbalances, constantly diverting the body's immunological resources and depleting the overall potential of anti-infective protection, which increases the number of newly emerging infectious foci and chronicizes them. On the other hand, the oral cavity in these patients becomes a permanent source of infection spread to the underlying organs, as well as lymphogenous and hematogenous throughout the body [10]. Between the occurrence of chronic foci of odontogenic infection and diseases of internal organs, there is a deep pathogenetic unity, due to mutual causal relationships mediated by immunological imbalances, impaired interleukin regulation and inferiority of nonspecific resistance of the body. The revealed regulatory shifts should be considered as potential points of application of the action of additional systemic therapeutic and preventive measures in the complex treatment of patients with combined infectious and inflammatory processes in the oral cavity and diseases of internal organs [10]. Currently, attention is drawn to the role of dental infection in the pathogenesis of diseases of the cardiovascular system. Thus, it was found that periodontitis plays an important role in the occurrence and development of atherosclerosis [8]. The presented data confirm the relevance of scientific research on the etiology and pathogenesis of periodontitis, new knowledge about which will improve the effectiveness of treatment and prevention of this pathology.

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