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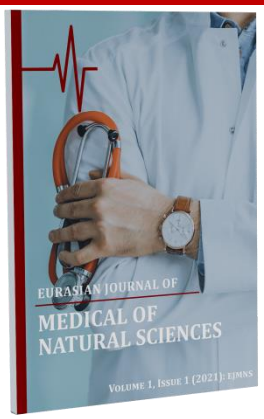

A MODERN VIEW ON THE ETIOPATHOGENESIS OF PERIODONTITIS (LITERATURE REVIEW)

Juraev Bakhrom Ilkhom ugli
Chemistry university hospital
Tashkent. Uzbekistan
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ABSTRACT

Periodontal pathology is one of the most common problems in modern dentistry, which is a serious medical and social problem all over the world. At the same time, the diagnosis and treatment of this disease is one of the most difficult problems of dentistry, since for many years the issues of etiology, pathogenesis and even the name of the disease have remained controversial, which has given rise to many classifications. It is known that any chronic disease is the final stage of a prolonged pathophysiological process that has arisen in the body under the influence of adverse factors that have a direct or indirect effect on the development of the disease. This article provides an overview of the data of domestic and foreign literature on the main etiological factors of periodontal diseases, as well as the mechanisms of their development from the point of view of modern dentistry.

Introduction. Periodontal pathology is one of the most difficult and common problems in modern dentistry, so the relevance of its solution is beyond doubt. According to the World Health Organization (WHO), periodontal diseases occur in various regions: from 55.0 to 99.0% in the age group from 15 to 20 years, and in the group aged 35 to 44 years, this indicator varies from 65.0 to 98.0% [1, 2].

According to WHO, the factors causing human diseases consist of hereditary (they account for 20%), environmental conditions (they account for 25%) and lifestyle (55%) [3].

Numerous scientific studies indicate that in the etiology of periodontal diseases, in addition to the general immune status, the presence of general somatic diseases, genetic predisposition and others, one of the main reasons is an unsatisfactory state of oral hygiene. Fedorov Yu. A. and Koren V. N. note that with regular oral care, periodontal diseases are observed much less frequently (30.4%), while with unsystematic care they occur in 37.5%, and in the absence of it - in 48.5% of cases [1, 4, 5]. This has a direct effect on the local immune status of the oral cavity, since all forms of inflammatory periodontal diseases occur against the background of progressive pathological changes in it [6, 7].

The low hygienic level of the oral cavity is the main factor in the formation of dental plaque and biofilm, which consist of specific microflora, have a high periodontopathogenic potential and are considered as the main etiological factor in the occurrence of inflammatory processes in periodontal tissues [8, 9].

In the formation of dental plaque, the main role is assigned to microorganisms and their waste products. With improper and irregular brushing of teeth, a rapid growth of microbial colonies occurs. For example, in 4 hours, the number of plaque microorganisms reaches about 103104 bacteria per 1 mm2 of the tooth surface. If the hygiene rules for the oral cavity are not followed during the day, the number of bacteria increases by an order of magnitude. Here, the main role is assigned to streptococci, which adhere to the pellicle and synthesize homopolysaccharide from sucrose, and it, in turn, is responsible for the accumulation of bacteria on the teeth. After 3 days, the accumulation of bacteria along the gingival margin leads to the subsequent creation of favorable conditions for the growth of the number of bacteria and a change in the composition of the microflora [4, 10, 11].

In addition to the microflora of plaque, anaerobic flora, namely endotoxins of periodontal pathogenic microorganisms, plays a leading role in the formation of the inflammatory process in periodontal tissues. According to WHO data, their representatives include those species that, along with the predominantly anaerobic type of respiration, have high invasive, adhesive and toxic properties in relation to periodontal tissues. At the same time, not one, but several microorganisms play a role in the formation and progression of periodontitis, which secrete several complexes - associations of microbes associated with periodontal diseases:

1. Porphyromonas gingivalis, Bacteroidies forsihus, Treponema denticola;
2. Streptococcus sanguinis, Streptococcus mitis, Streptococcus oralis, etc.;
3. Actinomyces odontolyticus, Actinomyces naeslundii, Veillonella parvulla;
4. Prevotella intermedia, Fusobacterium nucleatum, Campilobacter rectus [12].

In addition to oral hygiene, local factors predisposing to the development of inflammatory diseases in periodontal tissues include various anomalies of the maxillofacial region, attachment of the frenules of the lips and tongue, pathological bite, crowding of teeth, small vestibule

of the mouth, pronounced mucosal cords, bruxism, bad habits, etc. [13].

Separately, it is worth touching on such a bad habit as smoking, which is one of the risk factors for periodontitis. It suppresses the vascular reaction that usually accompanies gingivitis and periodontitis, as a result of which the body's immune response sharply decreases. Nicotine leads to the destruction of periodontal tissues by regulating the release of cytokinin, which probably explains the less pronounced inflammation and bleeding of the gums in smokers [14, 15].

A separate role in the pathogenesis of chronic inflammatory process in periodontal tissues is assigned to the immune system [16]. Macrophages, lymphocytes and mast cells, constantly making excursions to the surface of the gum, instantly react to any changes from the external environment, quickly turning on protective mechanisms. The inclusion of a specific immune defense link in the mechanism of pathogenesis of inflammatory periodontal diseases

is evidenced by the high frequency of detection of positive skin tests for gingival antigen in patients with periodontitis.

Many microbial products (exo- and endotoxins) have been identified as activators of an indirect pathway of action on the macroorganism. Scientists have assigned the name "modulins" to such substances of microbial nature [9].

Numerous studies by domestic and foreign scientists have proved that there is a certain relationship between inflammatory periodontal diseases and any general somatic pathology [3]. Examples of such diseases are pathologies of the cardiovascular system, diabetes mellitus, atherosclerosis, diseases of the gastrointestinal tract, respiratory tract, central nervous system, immunodeficiency, etc.

Thus, in the general structure of concomitant internal pathology in generalized periodontitis, diseases of the cardiovascular system (CVS) occupy up to 68%, depending on the type of pathology: hypertension - 26%, coronary heart disease (CHD) - 10.5%, neurocirculatory disorders - 68% [24]. It is also worth noting that with the progression of chronic periodontitis, the index of peripheral vascular resistance of the periodontal complex increases, while additional deterioration of blood flow has been reliably established in patients with various forms of coronary heart disease, including in combination with type 2 diabetes mellitus [5].

The relationship between the oral cavity and the gastrointestinal tract is a predisposing factor to the development of periodontal diseases. It was found that in patients with gastroesophageal disease, chronic pancreatitis, peptic ulcer of the stomach and duodenum, the prevalence of periodontitis is one and a half times more common than in healthy individuals [6]. In addition, modern researchers have proved that after a course of treatment for dysbiosis, patients with generalized periodontitis of stages I and II have positive dynamics of local immunological parameters, which is another confirmation of the close relationship between diseases of the oral cavity and gastrointestinal tract [7].

Pathogenetic similarity is also observed in diseases of the ENT organs and oral cavity. A significant effect is a decrease in the protective

properties of the oral mucosa in patients with bronchial asthma [8]. The relief of bronchial asthma attacks requires the appointment of medications, in particular inhaled glucocorticoids [1]. It has been proven that these drugs cause a decrease in local oral immunity, which creates favorable conditions for the reproduction of oral microflora and other pathogenic factors.

In patients undergoing treatment for chronic kidney disease due to intensive anti-inflammatory therapy, mild inflammation in the periodontium is observed. There are some changes in the metabolism in the periodontium. In more severe clinical cases, patients experience marginal periodontal recession and hyperesthesia of hard dental tissues, even with mild periodontitis. In children, nephrogenic osteodistrophy is manifested by changes in bone and cartilage tissue, which clinically resemble rickets, therefore this pathology is called "renal rickets". Osteomalacia develops in adults, in the mechanism of development of which the main role belongs to a violation of vitamin D metabolism [6].

There is also such a thing as "hepatic rickets", or "hepatic osteomalacia", which occurs in chronic hepatitis and cirrhosis. This pathology occurs with liver damage in childhood. The disease is accompanied by stunting, stunting and even dwarfism. Vitamin D is also important in

the mechanism of osteoporosis in liver lesions, namely its endogenous insufficiency. Alveolar bone atrophy increases in patients with chronic liver damage [11].

Pathology of the pituitary-adrenal system is also one of the etiological factors in the development of periodontal diseases. It is known that the use of glucocorticoids leads to partial resorption of the bone tissue of the alveolar bone. Cortisol reduces the activity of osteoblasts of the alveolar bone, causes the destruction of collagen fibers, accelerates osteoclastic resorption of bone tissue [4].

The increased atrophy of the alveolar bone under the influence of glucocorticoids is explained by their catabolic effect. Inhibition of bone tissue formation, an increase in glucocorticoid production may be due to a hyperplastic process in the adrenal glands or increased production of adrenocorticotropic hormone by the anterior pituitary gland (Ku-shing disease) [7].

A similar effect occurs as a result of a primary or secondary increase in the activity of the parathyroid glands, which produce parathyroid hormone.

This pathology often develops with hypoestrogenemia as a result of early extinction or shutdown of the function of the genital glands in women. Under the conditions of this endocrinopathy, changes in periodontal disease primarily reflect osteoporosis in the skeleton [6].

The importance of the nervous system in the dynamics of inflammation is beyond doubt. Under the action of any pathogenic factor, the nervous system and its trophic function are activated primarily or secondarily. It has been established that with short-term negative emotions in periodontal tissues, a prolonged increase in vascular tone occurs [12]. Blood pressure increases with-

the retention of glucocorticoids, which have a catabolic effect, which leads to a slowdown in the regenerative processes in cells. Lipid peroxidation is activated in tissues, as a result of which the content of free radicals increases, which have a toxic effect on cells [14]. Chronic emotional stress also has a negative effect on periodontal tissues. At the same time, there is a violation of metabolism in the periodontium and, first of all, lipids, the formation of lipid peroxides increases. They have a pathogenetic effect on periodontal tissues and accelerate involution [8].

As you know, acquired immunodeficiency syndrome affects many organs and systems, and, of course, this disease has manifestations in the oral cavity. Periodontal lesions are a typical manifestation in HIV-infected people [11]. Usually, periodontal lesions develop at low values of the CD4/CD8 ratio, their severity depends on the degree of decrease in the number of CD4 cells to a greater extent than on the degree of development of dental plaques or the presence of certain microorganisms [1].

Many authors note that microcirculation disorders are a trigger in the development of periodontal diseases [3]. The microcirculatory bed reacts to the appearance of various pathogenic factors long before the appearance of clinical symptoms of inflammation, as it is a very sensitive system. The development of chronic microcirculatory changes in the periodontium is associated with a violation in the leukocyte-platelet-endothelial balance. In pathology, when blood cells pass through the microcirculatory bed, their aggregation and adhesion to the vascular endothelium are observed, which possibly leads to blockage of the

capillary network with the development of hypoxia of periodontal tissues. It is worth noting that the pathogenetic factor is not morphological, but functional changes in periodontal vessels, leading to hypoxia. Under such conditions, oxygen delivery to periodontal tissues is significantly reduced to such a level that it will not be sufficient to maintain cell function, metabolism and structure [2].

Conclusions: In recent years, the planned sanitation of the oral cavity among the population of our country has significantly given way to individual sanitation in terms of circulation, especially of the organized contingent. Considering that the treatment of periodontal diseases is a time-consuming, step-by-step process that requires great effort and knowledge, it is obvious that timely prevention and elimination of local and general etiological factors will help reduce the likelihood of this pathology. In addition, the interest and literacy of the population in relation to the state of oral health and the independent detection of periodontal diseases in the early stages of their occurrence and development plays a significant role. All patients who initially sought dental care should be motivated to observe careful oral hygiene using various personal hygiene products, followed by quality control of the manipulations performed. It is also important to tell patients about the leading role of the microbial factor in the occurrence and development of inflammatory periodontal diseases.

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