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PATHOGENETIC RELATIONSHIP OF PERIODONTAL DISEASE AND INSULIN RESISTANCE

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ABSTRACT: Features of periodontal disease pathogenesis in the context of diabetes mellitus and their influence on treatment tactics are actively discussed in the scientific literature, although the nature of the effect of endocrine pathology on the development and course of generalized periodontitis and the mechanisms associated with its exposure as a risk factor cannot be considered conclusively clarified

Key words: periodontal diseases, diabetes mellitus, mononuclear phagocytes

INTRODUCTION: Periodontal pathology in insulin resistance is associated with impaired hemodynamics and the development of angiopathies, impaired cellular metabolism, immunological and neuroregulatory disorders in the body, as well as chronic tissue damage by the end products of enhanced glycosylation.

The development of the inflammatory process in periodontal disease, its generalization and chronization are determined by the specific and quantitative composition of the oral microflora, as well as the state of the immune system, altered by diabetic pathogenetic factors

Currently, the prevalence of periodontal diseases has sharply increased among people of both sexes who are in the reproductive period. According to WHO (1990), the presence of periodontal diseases (compared with complications of caries) increases by 5 times the development of functional disorders of the dental system, leading to tooth loss.

It is well known that in patients with DM periodontitis occurs in almost 100% of cases and is characterized by an aggressive nature of the course of This pathological process has a unique morphological structure, significantly different from inflammatory periodontal diseases in people without DM. The frequency and severity of this process has a direct correlation with the duration of carbohydrate metabolism disorders.

Etiology and pathogenesis of periodontitis in type I diabetes mellitus

Currently, there is no accurate epidemiological data on the frequency of periodontal diseases in patients with DM. The results of previously published studies trying to reflect the relationship between periodontitis and disorders of carbohydrate metabolism are contradictory, which is probably due to the large variability in the distribution of patients into groups, as well as the severity of differences in epidemiological methods of registering oral diseases. Nevertheless, in most studies, a high prevalence of periodontitis in various age periods is found in patients with impaired carbohydrate metabolism.

Type 1 patients who are in the stage of decompensation of diabetes and have concomitant somatic pathology, increased gingivitis intensity was determined in a significantly larger number of cases compared to their peers without diabetes. Therefore, the next stage in the study of this problem was the analysis of the effect of metabolic control disorders on the progression of oral diseases.

In patients with DM duration of 10 years or more, the loss of periodontal structures was more significant than in patients with impaired carbohydrate metabolism for less than 10 years. Examination of patients aged 40-49 years with a duration of DM for more than 20 years revealed a more significant loss of periodontal attachment and alveolar bone with a depth of periodontal pockets > 6 mm in comparison with patients without DM. The authors concluded that the aggravation of this pathological process could also be influenced by smoking, which contributes

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to the formation of pathological periodontal pockets, loss of periodontal attachment, increased risk of development and progression of periodontopathies, as well as vascular disorders.

An attempt to study the effect of sex steroids on the frequency and structure of oral diseases has not been successful at present. However, it is generally accepted that androgens and estrogens have a predominant anabolic effect on stimulating the synthesis of the matrix involved in the restoration of periodontal tissues, as well as on the gum growth induced by medications. At the same time, both estrogens and progesterone can lead to the development of gingivitis in pregnant women, and long-term use of hormonal contraception in women can contribute to an increase in the frequency of periodontal diseases. In addition, high levels of cortisol associated with stress factors significantly increase the risk of necrotic ulcerative gingivitis.

Vascular disorders and microbiological disorders

Periodontal angiopathy, along with retinal and renal angiopathy, refers to the earliest and most common lesions detected in 90-93% of patients with diabetes of both sexes. In the pathogenesis of periodontal syndrome in angiopathy, one of the dominant roles is given. Dysproteinemia plays a primary role in the genesis of lesions of small vessels.

Pathomorphological changes in blood vessels with disorders of carbohydrate metabolism are manifested by a lesion of the vascular wall against the background of an unchanged lumen of the vascular bed. It is known that diabetic microangiopathy is based on the processes of plasmorrhagia, which are reduced to primary plasma damage to the basement membrane of the microvascular bed, sclerosis and hyalinosis of the vascular wall; microcirculatory disorders are of a primary nature.

Against the background of violations of transcapillary metabolism, increased permeability of connective tissue structures, the microflora of the gingival furrow (its endotoxins, enzymes) causes inflammatory and destructive changes in the periodontal, the secondary overload of its tissues further aggravates the pathological process.

Etiology of periodontitis in patients with type 1 diabetes on the background of decompensation of carbohydrate metabolism

it may be partly explained by the presence of selective pathogenic microflora present in periodontal pockets. An increase in the concentration of glucose in saliva, dental and gingival fluid, as well as a decrease in salivation (up to xerostomy) can adversely affect the nature of bacterial flora, increasing the process of non-enzymatic glycation of proteins: inflammatory mediators, immunoglobulins and other immune defense mediators.

The study of subgingival bacterial morpho types revealed that spirochaetes and motile bacteria (located in the apical part of periodontal pockets) can be directly related to the occurrence and progression of periodontitis. In patients with impaired carbohydrate metabolism in the decompensation stage, as well as with reduced immunity, against the background of a long course of diabetes, the formation of these morphotypes increases, and exponentially. In patients with type 1 diabetes, the most common periodontal pathogen is Porphyromonas gingivalis [5, 6, 24]. Against the background of increased phagocytosis in patients with periodontitis, the bacteroid P. gingivalis and the spirochete Treponema den-ticola (which are residents of the oral cavity) activate the neutrophil matrix of metallo- and serine proteinases, further aggravating the course of the inflammatory process [10, 16, 19]. There were no differences in the proportions of spirochetes and flagellate bacteria in patients with diabetes compared with patients without carbohydrate metabolism disorders comparable in gender and age. However, there are other studies in which no significant changes in the subgingival microflora were found either in the depth or on the surface of the periodontal pockets in patients with type 1 diabetes, even against the background of poor metabolic control [19].

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Histological disorders: Currently, there are various morphometric methods for measuring the cellular and volumetric composition of the subepithelial tissue of the oral mucosa. Despite the assumption that cellular and immune responses are disrupted in patients with impaired carbohydrate metabolism and periodontitis, in none of the numerous studies of periodontal tissues in this category of patients, the qualitative and quantitative compositions of inflamed subepithelial tissue were not measured. As a result of histological studies, it has been proved that in the small blood vessels of the gum in patients with a long course of DM in 80% of cases there are microangiopathic disorders characterized by blockage and increased density of the vascular wall [3, 13]. The study revealed in patients with type 1 diabetes and varying degrees of compensation of carbohydrate metabolism a significantly significant increase in the width of the base layer of endothelial cells, but only in the thinnest vessels.

In patients with type 1 diabetes of reproductive age, a high frequency of microvascular disorders in periodontal tissues is determined, associated with prolonged hyperglycemia, resulting due to the Maylord reaction to a change in proteins (end glycated products (AGEs)), inducing oxidation processes in the gum and, naturally, increasing periodontal destruction [4, 9, 19].

Cellular and molecular changes

In patients with DM, a pathological increase in the activity of collagenases, matrix metalloproteinases, a decrease in collagen and glycosaminglycan synthesis (MMR), as well as metabolic disorders in fibroblasts of the periodontal junction are detected in periodontal tissues.

In studies on rats with DM and undergoing bilateral ovariectomy, it was revealed that it was carbohydrate metabolism disorders that caused an increase in the level of collagenases in the gum. This study confirmed the theory that the interaction of oxidants with potentially periodontopathogenic bacterial proteases activates tissue-destroying neutrophils and matrix proteases, such as MMP-8 and MMP-13. In patients with DM and poor metabolic control, pathologically excessive activity of the metalloproteinase matrix is determined both in the gum itself and in its fluid.

It is assumed that in patients with DM, microangiopathy, disorders of the composition of gingival fluid, disorders of collagen metabolism and deterioration of the wound healing process, as well as changes in the immune response and hereditary predisposition contribute to the development of periodontal diseases with progressive loss of alveolar bone mass. Disorders of neutrophil function (chemotaxis, adhesion, phagocytosis, bactericidal activity) can potentiate the existing predisposition to colonization and proliferation of periodontal pathogens in plaque.

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