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SPECIFIC PATHOMORPHOLOGICAL FEATURES OF THE ORAL CAVITY AND TEETH IN CHILDREN WITH TYPE 1 DIABETES.

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Abstract. It is well known that in patients with diabetes mellitus, periodontitis occurs in almost 100% of cases and is characterized by an aggressive course. This pathological process has its own, unique morphological structure, significantly different from inflammatory periodontal diseases in persons without diabetes mellitus. In this case, the frequency and severity of the above process has a direct correlation dependence on the duration of carbohydrate metabolism disorders

Keywords. nosological form, general somatic, periodontitis, diabetes mellitus.

As a result, the authors concluded that the dynamics of indicators of pathological lesions, harmony of physical development, and health groups among schoolchildren during primary and secondary school education is undulating, with negative trends in the first three years of study, at the end of the fifth grade and at the beginning of sixth grade, eighth grade and the end of ninth grade, which reflects critical periods of the formation of students' health associated with the adaptation of the child to learning conditions and the processes of his growth and development [1].The relationship of dental diseases with general somatic pathology is well known [11-13].

Programs for the prevention of caries and dental anomalies are developed and implemented annually. The issues of children's health improvement are constantly

discussed. However, in most cases, the programs being developed are aimed at preventing a particular nosological form. The low efficiency of such programs is most likely due to the lack of an integrated approach and a system of interdisciplinary interaction of pediatricians in prenosological diagnosis and early elimination of predisposing factors of the disease [14]. Currently, studies are being conducted more and more often, the results of which indicate that the state of the dental system is viewed as an indicator of somatic health. Studies [15, 16] have convincingly shown that the changes that occur in the state of the dental status of children with various health abnormalities are a reflection of the disorders occurring in the body. This point of view corresponds to the ideas about the unity of the structure and function of human body systems [17].

Etiology and pathogenesis of periodontitis in type I diabetes

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, most studies reveal a high prevalence of periodontitis at various age periods in patients with impaired carbohydrate metabolism [14, 28]. Thus, the prevalence of periodontal diseases among 100 patients with diabetes mellitus (DM) aged 18-30 years living in Santiago (Chile, 1999) was: for gingivitis - 22%; periodontitis - 41%. Only 37% of patients with impaired carbohydrate metabolism did not have any periodontal diseases. Nevertheless, most studies reveal a high prevalence of periodontitis at various age periods in patients with impaired carbohydrate metabolism [14, 28]. Thus, the prevalence of periodontal diseases among 100 patients with diabetes mellitus (DM) aged 18-30 years living in Santiago (Chile, 1999) was: for gingivitis - 22%; periodontitis - 41%. Only 37% of patients with impaired carbohydrate metabolism did not have any periodontal diseases.

According to other studies, a significantly significant correlation was clearly revealed only between the severity of periodontitis, the frequency of cardiovascular diseases and the presence and/or absence of diabetic nephropathy [32]. It was also found that in patients with diabetic retinopathy, the loss of periodontal attachment was more pronounced than in patients without complications from retinal vessels [2, 9]. At the same time, in children and adolescents with type I diabetes who are in the stage of decompensation of the underlying disease and/or have concomitant somatic pathology, the increased intensity of gingivitis was determined in a significantly greater number of cases when compared with their peers without diabetes [3, 13]. Therefore, the next step

in the study of the above problem was to analyze the effect of metabolic control disorders on the progression of oral diseases So, Ainamoetal. (1990) in one of the studies proved the progression of destruction of periodontal tissues in patients with poor metabolic control against the background of chronic hyperglycemia. At the same time, in this category of patients, periodontal lesions were often combined with an increased amount of tartar and a pronounced loss of periodontal attachment [1, 31]. Glavindetal. (1968) showed that 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 [5]. Submitted by Bacicetal. (1988), examination of patients aged 40-49 years and with a duration of DM for more than 20 years revealed a more significant loss of periodontal attachment and alveolar bone at a depth of periodontal pockets > 6 mm in comparison with patients without DM [2, 32] 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 drug-induced gum growth.

Vascular disorders

Periodontal angiopathy, along with retinal and renal angiopathy, refers to the earliest and most common lesions detected in 90-93% of diabetic patients of both sexes.

According to various researchers, angiopathy is given one of the dominant roles in the pathogenesis of periodontal syndrome in DM. In turn, dysproteinemia plays a primary role in the genesis of small vascular lesions in this disease (Efimov A. I. et al., 1970). Pathomorphological changes in blood vessels with disorders of carbohydrate metabolism are peculiar and manifest themselves by damage to the vascular wall against the background of an unchanged lumen of the vascular bed (Zerbino D.A., 1977). At the same time, it is well 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. These changes have nothing to do with inflammation, and, consequently, microcirculatory disorders are of a primary nature.

Against the background of already existing disorders 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. And the secondary overload of its tissues further aggravates the above-mentioned pathological process [4, 25, 26].

MICROBIOLOGICAL DISORDERS

The etiology of periodontitis in patients with type I diabetes on the background of decompensation of carbohydrate metabolism may be partly explained by the presence of selective pathogenic microflora present in periodontal pockets.

An increase in the concentration of glucose in saliva, gingival fluid, as well as a decrease in salivation (up to xerostomia) 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, as well as cells involved in the immune defense of the oral cavity. The above process, according to Morinushietal. (1989), naturally, leads to a decrease in immune protection.

According to foreign researchers, the study of subgingival bacterial morphotypes revealed that spirochetes 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 the above morphotypes increases, and exponentially. At the same time, in patients with type I diabetes, the most common periodontal pathogens are *Porphyromonas gingivalis* [7, 32]. Thus, against the background of increased phagocytosis in patients with periodontitis, the bacteroid *P. gingivalis* and spirochete *Treponema denticola* (which are residents of the oral cavity) activate the neutrophil matrix of metallo- and serine proteinases, further aggravating the course of the inflammatory process [35]. At the same time, according to foreign researchers, 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 where, for example, *Sastrowijotoetal.* (1990) 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 I diabetes, even against the background of poor metabolic control [20].

HISTOLOGICAL DISORDERS

Prolonged hyperglycemia causes vascular disorders, leading to changes at the cellular level of the oral mucosa. And most histological studies have proved that

small blood vessels of the gums in patients with long-term DM in 80% of cases have micro-angiopathic disorders characterized by blockage and increased density of the vascular wall. A study conducted by Listgarten et al. (1974), revealed in patients with type I 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.

Moreover, Vlassara and Bucala (1995), Berg et al. (1997), Chappey et al. (1997) proved that in patients with type I diabetes of reproductive age, a high frequency of microvascular disorders in periodontal tissues is determined, associated with prolonged hyperglycemia, which, due to the Maylard reaction, leads to changes in proteins (final glycated products (AGES), inducing oxidation processes in the gum, and, naturally, increasing the destruction of periodontal [22].

.BIOLOGICAL MECHANISMS:

CELLULAR AND MOLECULAR CHANGES

According to foreign studies, patients with DM in periodontal tissues have a pathological increase in the activity of collagenases, matrix metalloproteinases, a decrease in collagen and the synthesis of glycosaminoglycan (MMR), as well as metabolic disorders in fibroblasts of the periodontal junction.

In studies on rats with induced DM and who underwent 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 also confirmed the theory that the interaction of oxidants with potentially pathogenic bacterial proteases activates tissue-destroying neutrophils and matrix proteases, such as MMP-8 and MMP-13. And in patients with DM and poor metabolic control, pathologically excessive activity of matrix metalloproteinases is determined both in the gum itself and in its fluid (Sorsa et al., 1992; 1996).

It is assumed that in patients with DM, microangiopathy, disorders of the gingival fluid composition, 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 [6, 17]. At the same time, disorders of neutrophil function (chemotaxis, adhesion, phagocytosis, bactericidal activity) can potentiate the already existing predisposition to colonization and proliferation of periodontal pathogens in plaque.

Recent studies have provided additional data on potential metabolic and genetic factors contributing to an increased risk of development and severity of destructive processes in periodontal tissues in patients with DM. Thus, in recent studies, receptors for glycation products (RAGE) have been found both on the surface of

mononuclear phagocytes and on the surface of endothelial cells [22, 34]. In this regard, it has been suggested that oxidative stress contributes to an increase in the frequency and progression of the destruction process in periodontal tissues in patients with impaired carbohydrate metabolism. This is due to the fact that the interaction of glycated products with their receptor induces oxidative stress, contributing to the activation of NF- κ B cells with subsequent expression of mRNA, as well as the expression of anti-inflammatory cytokines (TNF α , IL-1 β , IL-6, mononuclear phagocytes), further aggravating the course of the inflammatory process. At the same time, the above mediators are defined as effectors of the processes of inflammation and destruction of periodontal diseases [16, 18]. In addition, the interaction of glycated products with their endothelial cell receptor leads to increased permeability and expression of vascular cell adhesion molecule - 1 (vascular cell adhesion molecule), which promotes the attraction of mononuclear cells to the vascular wall [11, 21]. Thus, the interaction of glycated products with their receptor leads to a more pronounced and prolonged course of the inflammatory process, as well as to a violation of wound healing and an increase in the frequency of periodontal diseases [10]

According to modern data, the effect of hyperlipidemia on the function of monocytes/macrophages in wound signaling during metabolic dysregulation is obvious. And this is due to the fact that the above mediators are important objects of the inflammation / wound healing phase, playing a predominant role in signal transduction and initiation of the transition from the inflammation phase to the granulation phase. So, with one of the supposed effects of hyperlipidemia - the interaction of fatty acids with the membrane of monocytes

There is a violation of the function of membrane receptors, as well as violations of enzyme systems [8]. All of the above leads to an "inadequate" increase in the signal during its transmission to the wound monocytes in DM, there is a non-enzymatic glycation of lipids and triglycerides, which disrupts the normal differentiation and maturation of specific phenotypes of monocytes at different stages of wound healing. The total effect of these processes is an exacerbation of inflammation and destruction of periodontal tissues. At the same time, against the background of impaired monocyte function associated with diabetes, lipid dysregulation (leading to an increase in LDL cholesterol and triglycerides) may be an important factor in increasing the risk of developing and severity of the above oral diseases [8, 15].

The predisposition of patients with type I diabetes to the presence of a hyperreactive monocyte phenotype leads to an increased response to gram-negative bacterial infections. As a result, it was assumed that this hyperreactive phenotype of monocytes is genetically predetermined and possibly regulated by genes in the HLA-DR3/4 and HLA-DQ regions[33].

DISORDERS OF THE IMMUNE SYSTEM

In patients with DM and clinical signs of periodontitis against the background of decompensation of carbohydrate metabolism, 80% of cases reveal violations of the immune response (mainly at the cellular level) resulting from damage and weakening of the function of neutrophil cells, monocytes/macrophages [8]. At the same time, a decrease in all cellular functions of polymorphonuclear neutrophil leukocytes (PMN), including their chemotaxis and adhesion, is determined.

It is interesting to note the fact that in patients with frequent hypoglycemia, violations in the mobilization of specific subpopulations of lymphocytes, which are one of the important manifestations of damage to the immune system, are detected in a larger percentage of cases. On the contrary, hyperglycemia and/or ketoacidosis lead to a decrease in the synthesis of collagen and glycosaminoglycans (GAGS). And under the influence of the process of non-enzymatic glycation, most of the functions of proteins and cells involved in immune defense are significantly weakened.

PERIODONTAL AND CARBOHYDRATE METABOLISM

An attempt to study the effect of DM on an increase in the frequency of periodontal diseases focuses on the level of glycemia, the increase of which is associated by most researchers with the progression of inflammatory processes of the oral cavity [19, 27]. That is, poor metabolic control adversely affects indicators reflecting periodontal health [23, 28, 30].

One of the first studies indirectly devoted to the above relationship is the correlation analysis between insulin resistance and inflammatory diseases of periodontal tissues [6, 12]. In the above-mentioned IC studies, it was shown that the tissues of the inflamed periodontal can serve as a source of tumor necrosis factor - α (TNF α), as well as other inflammatory mediators that have a significant effect on glucose and lipid metabolism. At the same time, the influence of IL-6 and IL-1 as hormone antagonists was not excluded- insulin [12].

Among the studies published before 1990, 6 out of 16 reports also indicated a high frequency and severity of periodontal diseases in patients with poor glycemic control. In the papers published after 1990, 13 out of 18 studies confirmed the above result [28, 30]. However, an analysis of the results of a long-term, 5-year follow-up of 20 patients with type I diabetes and 20 patients of the control group

(for 3 months) did not reveal a correlation between the intensity of bleeding of periodontal pockets, their depth and the values of the level of glycated hemoglobin - an indicator reflecting the degree of compensation of carbohydrate metabolism over the last 3 months.

Nevertheless, Tervonen and Karjalainen (1997), when observing 8 adult patients with poorly controlled type I diabetes for 12 months, revealed an earlier recurrence of the formation of periodontal pockets when compared with 13 patients in the stage of carbohydrate metabolism compensation and 10 patients in the control group.

The above clinical and epidemiological analysis suggests that periodontitis contributes to the deterioration of glycemic control in patients with DM. However, it is necessary to continue conducting controlled studies in various populations in order to confirm the importance of prevention and treatment of periodontal infections, which contribute to improving glycemic control and, possibly, reducing the risk of developing complications of diabetes.

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