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Or Pathogenesis of Periodontal Disease in I and II Levels of Obesity

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Received 2nd Oct 2023, Accepted 19th Nov 2023, Online 14th Dec 2023 **Abstract:** Today, periodontal disease is one of the most common and complex pathologies in modern dentistry (Bezrukov V.M., Grigoryants L.A., Rabukhina N.A., Badalyan V.A. 2003) In different regions of the world, about 50 percent of the population between the ages of 17 and 60 have various forms of periodontal disease. (Grigoryan A.S. etc. 1999). In developed countries, about 90 percent of the population have symptoms of gingivitis, 50 percent have moderate periodontitis, and 3 percent have severe periodontitis. Hetz G. 2009).

Key words: PERIODONT, disease, I AND II LEVEL.

According to the World Health Organization (2005), functional disorders caused by the death of the dental system in periodontitis diseases increase 5 times compared to the complications of caries and take the second place in the period of spread among all dental diseases (Vishnyak G.N. 1999 V.V. 2005. Zazulevskaya L.Ya. 2006.) Information about the spread of local periodontological inflammation and infectious pathologies based on the analysis of accounting and accounting documents of dental institutions, the study of the structure of etiological factors and the frequency of influence on the development of local peripheral diseases took Research has determined that the main causes of local periodontal development are the structure and nature of the inflammatory and destructive process, including pathogenetic factors and infectious-toxic factors, depending on the quality of dental care provided to the population, when the dental pulp is inflamed. (Grechishnikov V.V. 2005y.) Literature analysis shows that current scientific research does not fully disclose and justify the confusion and criteria that determine the relevance of the problem for the diagnosis of periodontal diseases (Puzin M.N., Kiparisova E.S., Wagner V.D. 2007y.) In the scientific literature published in recent years published data on this problem show the concept of chronic inflammatory diseases of the gastrointestinal system, which cause damage to the organs and tissues of the oral cavity and periodontitis. According to the author, conditions are created for the appearance of inflammation in periodontitis in diseases of the digestive system, because a number of regulatory mechanisms are disturbed: immune and endocrine imbalance, endokytosis, microcirculation, neurohumoral regulation, psychosomatic dependence, changes in the metabolism of connective tissues, mineral metabolism, vitamin deficiency. Microorganisms growing in subgingival plaques include more than 30 pathogenic microorganisms. Among them are Porphyromonas gingivalis, Prevotella intermedia, Actinobacillus actinomycetemcomitans, Bacteroides forsutus, Treponema denticola (Bitsermeister S. D. 2003. Kankanyan A. P., Leontev V. K. 1998y .Müller HP2008y).The authors proved the etiological role of

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these microorganisms in the occurrence of periodontitis, their connection with the severity of lesions determined the quantitative levels of various anaerobic microflora (Bitsermeister S. D. 2003.

Nikolaeva E. N., Tsarev V. I. 2004 Gonzalez JR, Michel J., Diete A. 2002y.v.b). The most important etiological factor in gingivitis and periodontitis, most authors consider dental microflora. (Tsepov L.M., Orekhova L.Yu., Nikolaev A.I., Mikheeva E.A. 2005y.v.b). Pathogenic and conditionally pathogenic microorganisms of the oral cavity are recognized as one of the underlying factors in the etiology of inflammatory periodical diseases. (Tsepov L.M., Goleva N.A. 2009y.v.b). The results of bacteriological examination and the study of sublingual plaques show a complex composition of microbes in periodontal diseases. Microorganisms include various streptococci, hemolytic staphylococci, trichominadas, fusobacteria, actinomycetes and others. In addition, specific gramnegative bacteria are identified in periodontal pockets, including: Porphyromonal gingivalis, Prevotella intermedia, Actinobacillus actinomycetemcomitans, Capnocytophages, Bacteriodis forsythus, Treponema dentiicola, Veilonella. (Tsepov L.M., Goleva N.A. 2009). 85 percent of all microbes are anaerobes, and 10 to 15 of them have pathogenic properties. These microbes, which make up a group of periodontopathogenic genes, are highly viscous, with invasive and toxic properties, damages the gingival epithelium moving along the edge of the tooth. It is a lipopolysaccharide that has a negative effect (Ivanov VS, 1998). Exo and endotoxins disrupt cell metabolism, cause changes in periodontal tissues, which contributes to the development of an inflammatory reaction. The development of the inflammatory process in periodontitis, its spread and chronic effects are determined not only by the types and amount of oral microorganisms, but also by the body's protective status and the responses of the immune system.

The body's immune reactivity plays an important, if not an important, role in the development of inflammatory periodontitis diseases (Grudyanov A.I., Dmitrieva N.A., Fomenko E.V. 2002). , associated with chemical activation and recruitment of anti-inflammatory cells, local and systemic metabolism, hemodynamic immunological and neuroregulatory irregularities and changes in microbiocenosis. (Yonemura T. 1989y. Watanabe K.v.b. 1991y. Firatii E.v.b. 1996. Siqueira JF et al. 2001).

Inflammation and inflammation in the periodontal period play an important role in the triggering mechanisms of destructive processes, and there are local and general systems of homeostasis protection and maintenance. Changes in nonspecific reactivity in periodontitis have been identified by many researchers. Depending on the severity of the pathological process in patients, complement titer and serum parezin amount, the level of serum lysozyme and the phagocytic activity of blood leukocytes decrease.

Thus, there is a change in the immunoglobulin indices A and sgA at the moderate level of periodontitis. (Hagewald S, Bernimoum JP, Kottgen E, Rage A. 2000).

In the process of phagocytosis, the degeneration process is very important, during which neutrophil particles are released. At the phagolysosome stage, they take an active part in the process of burning and washing periodontal microorganisms, which greatly contribute to reducing and reducing the rate of spread of microbial infection in periodontal tissues. Therefore, phagocytosis is the main defense against periodontal infections. mechanism. (Straka MC2002y).

Complement is a group of plasma proteins that circulate in the blood, they are actively activated with each other, and after activation, they combine with membrane proteins in cells, and at the same time, they are provided with a strong antibacterial defense against periodontitis. The role of components in the etiopathogenesis of periodontitis diseases. not defined. At the same time, the reciprocal effect of phagocytosis, the formation and attachment of antelocytes and complement provides the main protection against periodontal tissues. Although individual components of complement are found in

high quantities in serum, gingival fluid and tissue samples in patients with periodontal disease, the concentration of individual components of the complement often differs. (Bulgakova A.I. 2001)

It is explained by the fact that many types of periodontitis bacteria, which play an important role in the protection of periodontitis diseases against pathogens, can turn off some components of the accessory system. Porphyromon gingivalis, a component of complement in gingival fluids or a known pathogen that can cause accumulation on their bacterial surfaces (Bitzermeister S. D. 2003y. Grudyanov A.I. 1997y. Conde M. C, Yan S. 2000y.)

In the same way, it reduces complement activation by producing Fc-binding proteins released during the growth of A. actinomycetecomitans microbe.

The formation of specific antibodies against periodontitis bacteria supports the specific and subsequent type of immunotitis.

Antibodies released from periodontitis pathogens inactivate various harmful factors of these bacteria, resulting in preparation for phagocytosis by various immune reactions.

Today, periodontitis is evaluated as a manifestation of increased serous antibody activity against bacteria. In the late stage of periodontopathic disease, the spread of bacterial antigens in the human body occurs. This causes the penetration of specific bacteria into the periodontal tissue, which leads to inflammation and increased immunity. .

T- and B-Lymphocytes are two types of cells that must be given an immune response by a certain person. As it is known, the bacteria that live in the periodontal pockets are considered to be a source of toxins and antigens, activates. Macrophage activity in destructive periodontitis is one of the possible pathogenetic ways for the development of the disease, because macrophages can damage their periodontal tissues and produce many anti-inflammatory toxins. An important part of local toxins and the emergence of new B-lymphocytes as a possible exacerbation coefficient of the inflammatory process Th2 cytokine profiles (IL-1 alpha, IL-5, IL-6). In another group of patients with periodontal disease, IFN-alpha and IL-6 significantly increased (Sokolov D. I, Kuznetsov S.A, Kotov A. Yu. 2000y. Hayashi J, Saito I, Ishikawa I, Miyasaka N. 1997y.). Significant release of cytokines and immunoglobulins in inflamed areas leads to significant destruction changes. Periodontitis produces antibodies directly with inflammatory plasma cells, which account for about 50 percent of total B cells. These inflammatory and immunological reactions caused by a lack of IgG contribute to the inflammation of periodontal tissues. Increased levels of L-1, IL-6, and TNF-alpha cause periodic damage and chemical reactions. Hayashi J, Saito I, Ishikawa I, Miyasaka N. 1994y)

In recent years, according to clinical and experimental data, etiological factors in the oral cavity have been recognized by most researchers. Vishnyak G.N. 1999y. Grudyanov A.I., v.b. 2002y. Dmitrieva N.A.v.b 2002y

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