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## Modern Aspects of Etiopatogenez of the Generalized Periodontal Disease

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**Abstract** The review of literature on features of a current of parodontit at a metabolic disorder is provided in article. Thus, inflammation as one of the most ancient problems of medicine and also the related questions of reactivity and change of various components of a homeostasis including and a complex of metabolic changes, not only did not lose the value, but as the changed structure of incidence shows, acquires today still big relevance. Immune and inflammatory factors modulate a lipidic range. Lipoproteida render regulatory effects on the immune answer, metabolism of cages of a system of immunity and nonspecific resistance to pathogens. In relation to a parodontopatologiya issues of interaction of inflammatory process and violations of fatty exchange and their pathophysiological analysis were practically not developed.

**Keywords** Periodontal disease, metabolic syndrome, obesity, inflammation and metabolic syndrome

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### Introduction

The prevalence of periodontitis in the population, the difficulties in the prevention and treatment of the disease, the ambiguity in the interpretations of the main pathogenetic mechanisms (interdependent inflammatory, immune and metabolic) makes this problem extremely relevant in medicine. It is known that among patients with metabolic disorders (metabolic syndrome, diabetes, gout, systemic lupus erythematosus) inflammatory diseases of the periodontal complex are widespread. Periodontium, its structures are sensitive to the pathogenic effect of factors forming the proatherogenic spectrum of metabolic disorders. This aspect reflects the interdependent effect of two major pathogenetic mechanisms - inflammatory and metabolic [2, 5].

The author noted that periodontal infection can adversely affect the glucose level in diabetes. Treatment of periodontitis, which reduces the bacterial effect and, consequently, the inflammatory destruction of periodontal disease, helps to reduce the amount of glucose in the blood in patients with diabetes. According to Zvigintsev M.A. [20] there are several mechanisms by which diabetes adversely affects periodontal tissues: vascular changes; connection of glucose with tissue proteins; a change in collagen metabolism; increased activity of matrix metalloproteinases (collagenases); an increase in glucose in the gingival fluid, which leads to impaired function of periodontal cells, tooth decalcification, and carious tooth decay; violation of the immune response, as a result of which the function of neutrophils is weakened and a hyper reactive monocytic response arises, due to which periodontal tissues are destroyed [2]

According to WHO, obesity is recognized as an epidemic of the 21st century. At the end of the 20th century, 30% (approximately 1.7 billion) of the world's population showed signs of obesity. A special epidemiological situation is recorded in highly developed countries. For example, in the US 60% of the population is overweight [3].



Factors contributing to the development of obesity include genetic factors and environmental factors (excessive consumption of high-calorie foods, low physical activity, socio-cultural characteristics of society, etc. A particularly rapid spread of obesity is associated with its nutritional-constitutional or diet-induced (DI) form.

Analyzing the results obtained and based on literature data, the author expresses a number of points explaining the relationship between the development of the inflammatory reaction and changes in lipid metabolism, and also tries to give a pathogenetic assessment of these connections [18].

The nature of changes in immune reactivity at various stages of periodontitis has been studied by many authors [7-8]. This was a violation of immune defense factors and a change in immune regulation during advanced periodontitis, which is characterized not so much by an inflammatory reaction as by changes in osteoblasts and osteoclasts and bone resorption [10, 13].

When considering the pathogenesis of periodontitis, there is a significant lack of knowledge about the genesis and mechanisms of development of tissue lesions. Treatment and rehabilitation of patients with periodontitis are a significant challenge. When gingivitis occurs, which is the initial stage of the development of periodontitis, a number of authors found a pronounced weakening of specific factors of local protective reactions of the oral cavity [5, 11, 14].

Local therapeutic effects on the affected periodontium in such patients are often ineffective [12,14,17]. Lymphocytes are stimulated by bacteria of *V. alcalescence* type, obtained from patients with gingivitis or periodontitis, a factor inhibiting macrophage migration (MIF) is released. Dental plaque and certain types of bacteria - *V. alcalescence*, *A. viscosus*, *F. nucleatum*, *B. melaninogenicus* - are able to induce the production of MIF by lymphocytes of individuals with experimentally caused gingivitis and thus inhibit or completely suppress phagocytosis [12, 16].

Today, scientists in many countries are developing a concept according to which inflammation in general, and subclinical inflammation in particular, are considered as the general pathophysiological basis of modern pathology, which closes the pathogenetic circles of nosological forms of diseases of civilization. The commonality of inflammation and proatherogenic metabolic disorders from a pathophysiological point of view is quite natural, since both syndromes form the same cells: endothelial and smooth muscle, fibroblasts, monocytes and macrophages, neutrophils, platelets and, to a lesser extent, T and B lymphocytes .

In inflammation and atherosclerosis, the adhesion (fixation) of monocytes and neutrophils on the surface of the endothelium is activated by the same proteins of cellular interactions: integrins on the membrane of neutrophils and monocytes, E-selectin on the membrane of the endothelium and P-platelet platelet. In both pathological processes, active infiltration (chemotaxis) of tissues by monocytes and neutrophils circulating in the blood occurs. In both situations, activated neutrophils and tissue macrophages in a respiratory explosion reaction enhance the formation of superoxide radicals and activate peroxidation of proteins and lipids, causing alteration of normal tissues - [6, 9, 10]. Since 1999, after the publication, which has already become the textbook work of R. Ross, the inflammatory nature of atherosclerosis is recognized by most scientists. Since then in all studies and published articles, the idea of inflammation as the essence of the atherosclerotic process is dominant, and it pushed into the background the importance of hypercholesterolemia as a factor of atherogenesis. However, in most recently published works, the thesis on the importance of inflammation in the pathogenesis of atherogenic metabolic disorders has acquired a declarative character, since each author puts his own meaning into this concept. Over two decades of research, reports periodically appear that an association between inflammation in periodontal tissues and cardiovascular disease is detected.

Still debatable is the question of the etiological dependence of periodontal inflammation and lipid metabolism. . In a number of modern epidemiological, clinical and experimental studies, conflicting results have been obtained regarding periodontitis and coronary heart disease (CHD), which is associated with general additional risk factors characteristic of these nosological forms (aging, male gender, socioeconomic status, smoking). Nevertheless, studies of the relationship between serum antibody levels against periodontopathogens (*Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*) and risk factors for coronary heart disease (proatherogenic lipid profile) confirm this dependence [1, 4, 11].



Chronic infections with a subclinical course (for example, periodontitis), can play a greater role in the development of atherosclerosis than is currently considered. The decrease in the frequency of cardiovascular diseases with statins is due not only to lipid-lowering, but also anti-inflammatory effect of these drugs - [19, 20, 21].

Lipoproteins have regulatory effects on the immune response, the metabolism of cells of the immune system and non-specific resistance to pathogens. The relationship of the immune and lipid systems is usually analyzed when discussing several issues.

### **Conclusion**

In relation to periodontopathology, the issues of the interaction of the inflammatory process and disorders of fat metabolism and their pathophysiological analysis have not been practically developed. This aspect seems to be completely pathogenetically justified (the interdependent effects of inflammatory and metabolic components) from the standpoint of both pathophysiology and dentistry, which is important for the development of new (pathogenetically significant) therapeutic approaches.

### **References**

- [1]. АлявияЗ. А The influence of ozone and ultrasound on the factors of local immunity of the oral cavity with periodontitis: Author's abstract. ... Candidate of Medical Science. - Tashkent, 2009. – 21 с.
- [2]. Bitsermaster S.D. Microbiological diagnosis of periodontal tissue diseases // *New in dentistry*. - 2003. -№7. - С. 27-30.
- [3]. Bubnova M.G. Obesity: causes and mechanisms of mass gain, approaches to correction // *Consilium-medicum*. - 2005. - Том 7. - N5.
- [4]. Bulgakova A.I. The influence of the state of local immunity of the gums and oral cavity on the course of chronic periodontitis // *New in dentistry*. -2001(100). - №10. - С. 90-93.
- [5]. Butrova S.A. Metabolic syndrome: pathogenesis, clinical features, diagnosis, treatment approaches // *Russian Medical Journal*2001. - Т. 9, №2. - С. 56-60.
- [6]. Vishnyak G.N. Generalized periodontal disease. - Kiev, 1999. -
- [7]. Volozhin A.I., Poryadin G.V., Kazimirsky A.N. Immunological disorders in the pathogenesis of chronic generalized periodontitis // *Dentistry*. - 2005. - No. 3. - P.4-7.
- [8]. Volozhin A.I. Dysregulation of immunological mechanisms and phagocytic activity of leukocytes is the leading cause of acute inflammation // *Pathological Physiology and Experimental. therapy*. - 1997. - No. 2. - S. 29-31.
- [9]. Garazha N.N., GarusYa.N., Ivashov A.V., Sakurov A.A. The effectiveness of the antioxidant drug Mexidol in the complex treatment of inflammatory periodontal diseases // *Dentistry*. - 2006. - No. 6. - S.19-21.
- [10]. Grachev S.V. NO-therapy is a new direction in medicine. A look into the future. - Moscow: Publishing House "Russian Doctor", 2001. - P.19-21.
- [11]. Grigorian A.S. The role and place of the phenomenon of damage in the pathogenesis of periodontal disease // *Dentistry*. - 1999. - No. 1. - S. 16-20.
- [12]. Grigoryan A.S., Grudyanov A.I. Key links in the pathogenesis of periodontal diseases in the light of the data of the cytomorphological research method // *Dentistry*. - 2001. - No. 1. - S. 5-8.
- [13]. Grudyanov A.I. Periodontics (etiology, pathogenesis, treatment, prevention) selected lectures. - M.: JSC Stomatology, 1997, 32 p.
- [14]. Grudyanov A.I., Dmitrieva L.A., Maksimovsky Yu. M. Periodontics. The current state of the issue and directions of scientific developments // *Dentistry*. - 1999. - No. 1. - S. 31-33.
- [15]. Grudyanov A.I., Dmitrieva N.A., Ovchinnikova V.V. Substantiation of the optimal concentration of the drug metrogil-dent in the treatment of inflammatory periodontal diseases // *Dentistry*. - 2002. - No. 1. - S. 44-47.



- [16]. Grudyanov A.I., Dmitrieva N.A., Fomenko E.V. The use of tablet forms of probiotics Bifidumbacterin and Acylact in the complex treatment of inflammatory periodontal diseases // *Dentistry*. - 2002. - No. 1. - S. 39-43.
- [17]. Grudyanov A.I., Barsegyan T.A., Chkonia G.D. Combined treatment of inflammatory periodontal diseases with cycloferon and metranidazole // *Russian dental journal*. - 2006. - No. 6. - S.20-23.
- [18]. Demurov E.A., Moiseeva E.G., Illarionova T.S. Pathogenetic mechanisms of chronic inflammation // *Development of scientific research at medical faculties of Universities of Russia: Materials 1 All-Russian. Scientific conference*. - Moscow, 2001. S. 206-207.
- [19]. Elovikova T.M. Treatment of lesions of the periodontal and teeth in patients with type 1 diabetes mellitus // *Dentistry*. 1989. No. 4. P. 18-20.
- [20]. Zvigintsev M.A. Dental rehabilitation of patients with diabetes mellitus: author. dis. ... Dr. med. sciences. - Omsk, 1998, 43 p.
- [21]. Abduraxmanov M.M., Abdullaeva M.A. Congenital risk factors in uzbek population with nonspecific aortoarteriitis // *European science review*.—Austria, 2018. - N11-12. - P.51-53.

