The Pharmaceutical and Chemical Journal, 2019, 6(6):108-113

Available online www.tpcj.org

Review Article

ISSN: 2349-7092
CODEN(USA): PCJHBA

Features of Periodontitis in Metabolic Disorders

Navruzova U.O., Khamidova N.K, Yusupov Sh.A.

Bukhara State Medical Institute, Department of Pathological Physiology

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.

Keywords periodontal disease, metabolic syndrome, obesity, inflammation and metabolic syndrome

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 [1-2].

Periodontal pathology is mainly inflammatory in nature and can develop under the influence of both local causes and the combined action of common (endogenous) and local factors against the background of a change in the reactivity of the body, the organization of the immune response. The immune system, designed to ensure the genetic constancy of the internal environment of the body, the protection of the macroorganism from all kinds of exogenous and endogenous pathogens, due to various reasons, can be the basis for the formation of a chronic inflammatory process and metabolic disorders. Such disorders underlie the development mechanism of many pathological processes, such as hypertension, coronary heart disease, obesity, gout, etc. The organs and tissues of the oral cavity, in particular periodontium, are also involved in the pathological process. At the same time, inflammatory and dystrophic changes in periodontal disease are directly dependent on factors such as the age of the patients, the severity of the disease, and the therapy. Recent studies in the field of metabolic syndrome have established that a number of forms of insulin, proinsulin, insulin growth factors have the same immune characteristics and are defined as immunoreactive insulin. Immunoreactive insulin contains several forms, such as proinsulin, insulin growth factors, C-peptide, free, as well as protein associated with insulin, etc. The ratio of these forms can be different, which is very important, since they have different biological and proliferative activity. The predominance of one form or another of insulin and proinsulin will determine the direction of the pathological process, including in periodontal disease [3].

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. [4] 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 hyperreactivemonocytic response arises, due to which periodontal tissues are destroyed [2].

According to the indicators of carbohydrate and lipid metabolism, it is possible to form a clear idea of the degree of compensation for diabetes mellitus, which is very important when choosing treatment tactics and predicting the timing of dental rehabilitation. In the past, some literary sources cited data on the effect of diabetes on the occurrence and course of the destructive process in periodontal disease.However, modern observations confirm the fact that periodontitis can significantly worsen the prognosis of diabetes [5-6]. Various epidemiological studies state that diabetes increases the risk of periodontitis by a factor of 2–3 and also significantly affects the intensity and degree of destruction of periodontitis [7-8].

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 [9].

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 f society, etc. A particularly rapid spread of obesity is associated with its nutritional-constitutional or diet-induced (DI) form.

It is known that adipose tissue is not only an energy depot, but also produces many factors (endocrine, metabolic, immunological) that are involved in the regulation of various physiological processes. Dysregulation of adipokine production in adipose tissue and violation of the mechanisms of signal regulation mediated by them play an important role in the development of obesity and associated metabolic and pathological disorders. As you know, with obesity, the death of adipocytes along the path of necrosis / apoptosis is intensifil. The mechanism of cell death (apoptosis / necrosis) largely depends on the level of ATP, in particular, the intensity of oxidative phosphorylation of mitochondria. Impaired mitochondria of adipose tissue in obesity is probably one of the reasons for the prevalence of adipocyte necrosis. A selective increase in the production of reactive oxygen species in stored fat contributes to the development of systemic oxidative stress in the body and the manifestation of obesity-related complications, such as insulin and leptin resistance. Studies show that increasing oxidative stress in accumulated fat is an early marker of metabolic syndrome [10].

An exception is the processes of lipoproteins, the amount of which in the blood increases with an inflammatory reaction in periodontal disease [11].

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, 29, 33].

The nature of changes in immune reactivity at various stages of periodontitis has been studied by many authors [15-16].

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 [17-18].

There is practically no data in the literature on the nature of the inflammatory reaction and immune regulation of metabolic changes at the initial stage of periodontitis, when the process is limited by the inflammatory reaction of the gums and is local, local in nature. The role of the defense system is associated with the induction of pro-inflammatory expression of tissue cytokines, activation of chemoattractants and involvement of pro-inflammatory

cells, with metabolic, hemodynamic, immunological and neuroregulatory disorders and microbiocenosis shifts [19-24]. The authors studied IL-1β, IL-6, and TNFα, which are involved in the regulation of inflammation, immune response, and hepatopoiesis in patients with periodontitis. The nature of changes in the activity of IL-6 in blood serum corresponded to the dynamics of TNF-α and IL-1β [25].

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 [1, 26-27]. Local therapeutic effects on the affected periodontium in such patients are often ineffective [28-29].

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. nucleatumn, 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 [28, 30-31].

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 [17, 32-33].

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 [26, 34-35].

Chronic infections with a subpclinical course (for example, periodontitis), can play a greater role in the develoment 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 [4, 36].

The intestinal microflora, as well as the discharge of lipids into the bloodstream of the liver during abdominal obesity and metabolic disorders, are considered as possible factors for the initiation and progression of the pro-inflammatory status of the body. But there is one more system. The microflora of the oral cavity is an integral part of our body. The number of bacteria in the oral cavity in terms of the number of species and the content in a unit of

material competes with the gastrointestinal tract, and the pro-inflammatory cytokine spectrum in any type of inflammation can initiate systemic disorders. There is no doubt that with periodontal inflammation, the immune and metabolic processes proceed in parallel, and they are based on common mechanisms associated with the imbalance (congenital and / or acquired) between the production of pro-inflammatory (pro-osteoporetic) and anti-inflammatory (anti-osteoporetic) cytokines, as well as with the corresponding changes in the activity of neutrophils and macrophages as effector cells. Thus, inflammation as one of the oldest problems of medicine, as well as related issues of reactivity and changes in various components of homeostasis, including a complex of metabolic changes, not only has not lost its significance, but, as the changed structure of the incidence shows, it is gaining even more urgency today .Violation of the interaction of inflammation, the immune system and lipid metabolism is an important mechanism for the development of modern pathology. Immune and inflammatory factors modulate the lipid spectrum [37].

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.

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]. Butrova S.A. Metabolic syndrome: pathogenesis, clinical features, diagnosis, treatment approaches // Russian Medical Journal 2001. - T. 9, №2. - C. 56-60.

[2]. Bitsermaster S.D. Microbiological diagnosis of periodontal tissue diseases // New in dentistry. - 2003. -№7. - C. 27-30.

[3]. Pozharitskaya M.M., Rudneva E.V., Simakova T.G., Chilizubova I.V. The effectiveness of the drug Coenzyme-Q10 in the complex treatment of inflammatory periodontal diseases // Periodontology. - 2004. - № 2(31) –C. 33-36.

[4]. Zvigintsev M.A. Dental rehabilitation of patients with diabetes: Abstract. dis. ... Dr. honey. sciences. - Omsk, 1998 43 p.

[5]. Orekhova L.Yu., Oganyan E.S., Levin M.Ya., Kalinin V.M.// Periodontology.– 1999. - № 3(13). – C. 24-26.

[6]. OrekhovaL.Yu.,Oganyan E.S., Levin M.Ya. // Dentistry of the new millennium: Abstracts. -M., 2002. – C. 183-184.

[7]. Finger M.A., Anichkov N.M. Pathological Anatomy: Textbook. - M., 2001. –T.2, Ч. 1.– C. 709-729.

[8]. Rabinovich S.A., Moskovets O.N., Efremov O.S. // Dentistry of the new millennium: Abstracts – M., 2002. – C.76-77.

[9]. Bubnova M.G. Obesity: causes and mechanisms of mass gain, approaches to correction // Consilium-medicum. - 2005. - Том 7. - N5.

[10]. Kamigaki M., Sakaue S., Tsujino I., Ohira H., Okeda D. Oxidative stress provokes atherogenic changes in adipokine gene expression in 3T3-L1 adipocytes // Biochemical and Biophysical Research Communications. - 2006. - N339. - P. 624-632.

[11]. Moiseeva G.G. Metabolic homeostasis and the body's immune reactivity in the dynamics of inflammation in periodontal tissues: Abstract. diss. ... Dr. med. - Moscow, 2008 .-- 36 p.

[12]. Demurov EA, Moiseeva EG, Illarionova TS Pathogenetic mechanisms of chronicity of the inflammatory process // Development of scientific research at the medical faculties of the Universities of Russia: Materials 1 Vseros. scientific conference. - Moscow, 2001.- P.206-207.

[13]. Mustaetsa V.F., Kuzovnikov A.E., Pasechnik A.V., Moiseeva E.G. Functional parameters of leukocytes in conditions of altered carbohydrate metabolism // Health and education in the 21st century; Concepts of the Diseases of Civilization: Scientific proceedings of the 8th International Congress. - Moscow, 2007 -- P. 444.

[14]. Pasechnik A.V., Moiseeva E.G., Frolov V.A., Drozdova G.A. Pathophysiological aspects of the regulation of the inflammatory process: additive effects of agonists and corticosteroids // International Journal on Immunoreabilitation. - 2001. - №1. - C.114.

[15]. Volozhin A.I., Poryadin G.V., Kazimirsky A.N. Immunological disorders in the pathogenesis of chronic generalized periodontitis // Dentistry. - 2005. - No. 3. - S. 4-7.

[16]. Volozhin A.I. Dysregulation of immunological mechanisms and phagocytic activity of leukocytes is the leading cause of acute inflammation // Pathological physiology and experiment. therapy. - 1997. - No. 2. - S. 29-31.

[17]. Grachev S.V. NO-therapy is a new direction in medicine. A look into the future. - Moscow: Russian Doctor Publishing House, 2001. - P.19-21.

[18]. Grudyanov A.I. Periodontology (etiology, pathogenesis, treatment, prevention) selected lectures. - M.: OJSC Dentistry, 1997. - 32 p.

[19]. Krechina E.K., Maslova V.V. ,Frolova S.A. Evaluation of the state of microcirculation in periodontal tissues according to laser and ultrasound Dopplerometry // Dentistry. - 2007. - Special issue. - S. 45.

[20]. Krechina E.K., Maslova V.S., Rakhimova E.N. Determination of hemomicrocirculation in periodontal tissues using laser and ultrasound dopplerometry. - M.: Medicine, 2008 .--18 p.

[21]. Lemetskaya T.I., Motna M.N. The condition of periodontal tissues in patients with obesity // Basic dental diseases. 1997. No. 4.- P.41-43.

[22]. Lemetskaya T.I. The effect of concomitant somatic pathology on the severity of destructive changes in periodontal disease // Problems of Neurostomatology and Dentistry. - 1997. - No. 2. - S. 2628.

[23]. Lemetskaya T.I. Clinical and experimental justification for the classification of periodontal diseases and pathogenetic principles of therapeutic and preventive care for patients with periodontal pathology: Author. dis. ... Dr. honey. sciences. - Moscow, 1999 .-- 62 p.

[24]. Sigusch B.T., Eick S., Pfister W. Altered chemotactic behavior of crevicular PMNs in different forms.

[25]. Lyanova D.K., Kosyreva T.F., Drozdova G.A. Cytokine status in the development of inflammatory periodontal diseases in patients with diabetes mellitus // Russian Dental Journal. - 2009. - No. 3. - S. 42-45.

[26]. Grigoryan A.S. The role and place of the damage phenomenon in the pathogenesis of periodontal disease // Dentistry. - 1999. - No. 1. - S. 16-20.

[27]. Grudyanov A.I., Dmitrieva L.A., MaksimovskyYu.M. Periodontology. The current state of the issue and the direction of scientific developments // Dentistry. - 1999. - No. 1. - S. 31-33.

[28]. 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.

[29]. Grudyanov A.I., Barseghyan T.A., Chkonia G.D. Combined treatment of inflammatory periodontal diseases with cycloferon and metranidozole // Russian Dental Journal. - 2006. - No. 6. - S.20-23.

[30]. Grudyanov A.I., Dmitrieva N.A., Fomenko E.V. The use of tablet forms of probiotic Bifidumbacterin and Acylact in the complex treatment of inflammatory periodontal diseases // Dentistry. - 2002. - No. 1. - S. 39-43.

[31]. Kolobkova L.N. Nikolaev I.V., Stepanova E.V., Landesman E.O. The use of Xidophone in the complex of measures for the prevention of inflammatory periodontal diseases // Dentistry. - 2007. - No. 2. - S.24-29.

[32]. Vishnyak G.N. Generalized periodontal disease. - Kiev, 1999.

[33]. Garage N.N., Garus Y.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.

[34]. Alavia Z.A. 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 c.

[35]. 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. - C. 90-93.
[36]. Elovikova T.M. Treatment of periodontal and dental lesions in patients with type 1 diabetes mellitus // Dentistry. -1989. - No. 4. - S. 18-20.
[37]. Grudyanov A.I., Dmitrieva N.A., Ovchinnikova V.V. Justification of the optimal concentration of the drug metrogildent in the treatment of inflammatory periodontal diseases // Dentistry. - 2002. - No. 1. - S. 44-47.