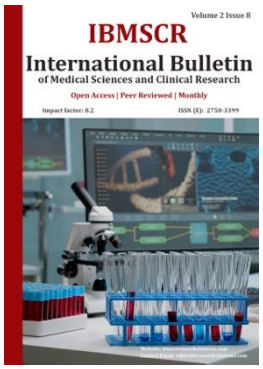

CHANGES IN PERIODONTAL TISSUES IN CHRONIC DISEASES

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https://doi.org/10.5281/zenodo.14715926

Abstract. The article presents modern information reflecting the main risk factors for the development of inflammatory periodontal diseases. Along with immune, genetic and general somatic mechanisms, the role of microorganisms and free-radical oxidation reactions as the main links in the pathogenesis of chronic periodontitis is described in detail.

Key words: chronic periodontitis, dental plaque, lipid peroxidation.

Izoh. Maqolada yallig'lanishli periodontal kasalliklar rivojlanishining asosiy xavf omillarini aks ettiruvchi zamonaviy ma'lumotlar keltirilgan. Surunkali periodontit patogenezida immun, genetik va umumiy somatik mexanizmlar bilan bir qatorda mikroorganizmlar va erkin radikal oksidlanish reaksiyalarining asosiy bo'g'inlari sifatidagi roli batafsil yoritilgan.

Kalit so'zlar: surunkali periodontit, tish plastinkasi, lipid peroksidatsiyasi.

Аннотация. В статье представлены современные данные, отражающие основные факторы риска развития воспалительных заболеваний пародонта. Подробно обсуждается роль микроорганизмов и реакций свободнорадикального окисления как ключевых звеньев в патогенезе хронического пародонтита, наряду с иммунными, генетическими и общесоматическими механизмами.

Ключевые слова: хронический пародонтит, зубной налет, перекисное окисление липидов.

Introduction. Despite certain successes in the treatment of periodontal diseases, the search for new treatment methods remains relevant [1,2]. In recent years, the use of photodynamic therapy (PDT) for the treatment of inflammatory periodontal diseases has been proposed [3-8]. High antimicrobial efficiency of PDT has been proven [9,10]. In dentistry, photosensitizers of various groups are used for PDT, mainly in the form of gels for topical application. Derivatives of chlorin E6 are most often used as photosensitizers: photoditazine, radachlorin, etc. [5,6]. Inflammatory diseases of periodontal tissues, which include chronic generalized periodontitis, are widespread in dental practice and are not only a medical but also a social problem [1,4,8,10]. This is due to the fact that periodontitis leads to tooth loss, and foci of infection in periodontal pockets negatively affect the body as a whole. Considering the prerequisites for the development of periodontitis, it is important to note that the cause of the pathological process in periodontal tissues can be various factors of both exogenous and endogenous origin. In this case, the action of pathogenic factors is manifested in the case of suppression of the protective and adaptive capabilities of periodontal tissues with a decrease in the general reactivity of the body [6,7,10].

Currently, by assessing the frequency of occurrence and development of chronic generalized periodontitis, the dominant influence of endogenous factors has been revealed.

Among the common factors that reduce the body's resistance and create a predisposition to the development of periodontal diseases, endocrine diseases (diabetes mellitus, hormonal dysfunction of the reproductive system, diffuse toxic goiter), pregnancy, neurosomatic diseases (rheumatism, metabolic disorders), blood diseases, hypovitaminosis C, B, A, E, infectious diseases, obesity, systemic osteoporosis, diseases of the gastrointestinal tract, cardiovascular and respiratory systems are noted. Many scientific papers are devoted to the role of the central nervous system in the development of periodontal inflammation.

Most authors tend to consider the neurogenic effect on the periodontium as a neurovegetative or neurohormonal mechanism for regulating regional blood circulation. According to the embryogenetic theory, the form of periodontal pathology (inflammatory or dystrophic) and the place of onset of the disease (bone tissue or gingival margin) depend on which embryogenetically dependent link or system of the body the changes occur in.

Scientists have identified genetic factors that predispose to the development of periodontal diseases, such as the presence of HLA-A24 antigens; the DR4 factor on chromosome 6; blood group III due to a reduced amount of lysozyme and secretory immunoglobulin A; acetyltransferase polymorphism; anatomical features of the jaw structure in people of Arab, Jewish, Tatar, and some Caucasian nationalities. Local risk factors for the development of inflammatory periodontal diseases include dental plaque, anomalies and deformations of the jaws, dystrophy and crowding of teeth, bite anomalies, a shallow vestibule of the oral cavity, pronounced strands of the mucous membrane, anomalies of the attachment of the frenulum of the lips and tongue, bruxism, iatrogenic retention areas, bad habits, etc.

Currently, most researchers have come to a consensus that dental plaque underlies the development of inflammatory changes in the periodontium. A microbiological study of its composition in individuals with the initial form of inflammatory disease of the marginal periodontium showed a wide variety of detected strains, a high frequency of obligate anaerobic bacteria mainly due to an increase in the proportion of periodontopathogenic rods and cocci. The role of such pathogens as A. Actinomycetemcomitans, Bacteroides forsythus, Porphyromonas gingivalis, Prevotella intermedia, T. denticola, S. noxia in the development of periodontitis with loss of gingival attachment and destruction of bone tissue has now been proven.

Immunological studies are promising for studying the etiology and pathogenesis of chronic inflammatory periodontal diseases, since the waste products of microorganisms and lipopolysaccharides of their cell wall activate the synthesis and secretion of a wide range of molecules by macrophages and leukocytes: cytokines (interleukin-1, tumor necrosis factor-α), prostaglandins (PG-E2) and hydrolytic enzymes, which cause degradation of the extracellular matrix and lead to further destruction of the alveolar bone tissue.

Phagocytosis is the main mechanism of protection against periodontopathic infections. The inflammatory infiltrates in the gums that arise are mainly represented by immunocompetent cells, which also indicates the interest of the immune system in this process. Often the inflammatory process in the periodontium occurs against the background of an altered state of the cellular and humoral links of immunity, supported by the system of T- and B-lymphocytes, respectively. Antibodies from periodontopathic bacteria are immunoglobulins, which in the human body are responsible for the humoral specific immune response. Non-specific protective factors of saliva, such as lysozyme, mucin, interferon,

nucleases, acidic glycoproteins, beta-lysins and secretory immunoglobulin A, are included in the functional concept of the "colonization resistance barrier" of the mucous membranes, in the formation of which, on the one hand, normal microflora takes part, on the other - epithelial cells and their receptors, complementary to bacterial adhesins, which make up the microbiocenosis of a specific biotope.

Smoking is one of the risk factors for periodontitis, it suppresses the vascular reaction usually associated with gingivitis and periodontitis, and reduces the body's immune response. Nicotine causes destruction of periodontal tissues by regulating the release of cytokinin, which probably explains the less pronounced inflammation and bleeding of the gums in smokers.

A large number of scientific studies in recent years have been aimed at studying energy metabolism in tissues with periodontitis. Under conditions of hypoxia in periodontal tissues, oxygen delivery is primarily disrupted, then a cascade of biochemical reactions is launched, including, first of all, a disruption of energy metabolism. This is facilitated by a decrease in the rate of tissue respiration, uncoupling of oxidative phosphorylation, accumulation of under-oxidized metabolites and changes in the redox systems of the cell. This pathochemical complex, together with a deficiency of macroergs and a disruption of active transport mechanisms, can lead to irreversible changes. It has been established that of all the consequences and complications of hypoxia, the most serious is the intensification of free-radical (peroxide) oxidation and the suppression of antioxidant protection of biological tissues and environments.

Peroxidation normally occurs continuously in all living tissues of the body, and free radical processes at low intensity are one of the types of normal metabolic processes. Violation of the balance of the rate of formation of active oxygen forms contributes to the self-accelerating process of peroxidation, which leads to the complete destruction of unsaturated lipids, disruption of the structure and function of proteins, nucleic acids and other molecules, and ultimately causes cell death. At the organ level, biochemical effects are realized in the form of exudation and proliferation reactions in the inflammation focus. At the systemic level, intoxication, microcirculation disorder, autosensitization, and immune response disorder occur, which, in turn, at the organismal level forms a pathological regulatory system and inadequate adaptive reactions. Experimental studies have clinically proven the key role of the antioxidant system in the metabolic correction of the processes of redistribution of energy substrates, its effect on the structural and functional state of membranes and receptor sensitivity of cells.

The physiological level of lipid peroxidation is controlled by various regulatory systems. One group includes prooxidants that accelerate lipid peroxidation. In the normal course of biochemical processes, prooxidant activity is determined by the concentration of metals of variable valence. The second group includes antioxidants that can inhibit non-enzymatic free-radical oxidation of lipids in low concentrations. Antioxidants include vitamins containing amino acids and peptides, and antioxidant enzymes include catalase, superoxide dismutase, glutathione peroxidase and other peroxidases.

In patients with varying severity of periodontitis, disturbances in the antioxidant system were found, namely, changes in the activity of superoxide dismutase, catalase, malondialdehyde, conjugates, Schiff bases and a decrease in the antioxidant activity of saliva.

It was found that aging cells are more susceptible to peroxidation. Accumulating intermediate products of lipid peroxidation with a simultaneous decrease in the antioxidant activity of saliva confirm the connection of generalized periodontitis with the patient's age. It is assumed that changes in antioxidant deficiency and aging accelerate the age-related involution of periodontitis.

Thus, chronic generalized periodontitis is a microbial-induced immune damage to the periodontal complex with a high probability of genetic and general somatic predisposition, occurring with a violation of free-radical mechanisms in tissues, characterized by a progressive course with an outcome in the resorption of bone tissue of the alveolar process.

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