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Drug Therapy in the Treatment of In flammatory Periodontal Diseases

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Relevance of the study. One of the most common inflammatory pathologies in adults is periodontal disease, which affects 70-90% of the world's population over the age of 40. To date, the multifactorial causes of pathology are clear, as well as the mechanisms of pathogenesis are almost revealed. Among them, the leading role is played by the microbial factor, a violation of microcirculatory mechanisms, as well as the severity of local immunity. Pathogenic microorganisms that cause periodontal diseases produce endotoxins that contribute to the progression of systemic diseases, including cardiovascular diseases, type 2 diabetes, respiratory tract infections, speech disorders, low self-esteem, and decreased quality of life. However, no highly effective therapy method has been proposed at the moment, and therefore the search for a combination of therapeutic factors contributing to the long-term remission of patients with inflammatory periodontal diseases continues. The current scientifically proven trend in the treatment of chronic inflammatory periodontal pathologies is to minimize surgical interventions, as well as to maximize the use of advanced conservative methods, which is achieved by using anti-inflammatory drugs and physical therapeutic effects. For local therapeutic effects on inflamed periodontal tissues , the drug celecoxib was chosen, which is a nonsteroidal anti-inflammatory drug by chemical nature that reduces swelling, redness of the mucous membrane, and reduces inflammatory phenomena. To prolong the effect of the drug, as well as as a detoxifying measure, polysorb was used together, which is proven capable of in vitro sorption of various drugs, their desorption in the inflammatory infiltrate, as well as persistent immobilization of microbes and their waste products. The aim of the study is to increase the effectiveness of drug and laser therapy in the treatment of inflammatory periodontal diseases. Gingivitis and periodontitis are inflammatory diseases caused by the penetration of infection into the soft tissues and bone structures supporting the teeth (into the periodontium). Genetic, environmental, and behavioral stereotypes, including bad habits and dietary patterns, are considered to be potentiating factors in the development of diseases. Periodontal diseases, especially mild and moderate-stage diseases, are widespread among the elderly worldwide, while the severe form is more common between the third and fourth decades of life, with a global prevalence of CVD of about 80-100%.

In addition to the above-mentioned microorganism, Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola, which make up the so-called red complex, showed the strongest association with periodontal diseases. It has been proven that P. gingivalis and T. denticola, as well as Filifactor alocis, a gram-positive anaerobe, also belong to the causative agents of CVD. Of these, Porphyromonas gingivalis is the main pathogen in this process, causing an allogeneic interaction between the subgingival biofilm and the reaction of the macroorganism. Even as an optional component of the subgingival microbiota, it can seriously affect the ecosystem, the number and localization of commensal bacteria, impairing immunity. P. gingivalis, a highly proteolytic gram-negative anaerobe, is quite rare in children and adolescents, but its content increases significantly with age, the largest number is found in the majority of the population after 55 years. Unlike P. gingivalis, there is no correlation between the number of Aggregatibacter actinomycetemcomitans colonies and age characteristics. This gram-negative periodontal pathogen manifests itself in aggressive forms of periodontal diseases. Many CVP-related microbial species, including A. actinomycetemcomitans, P. gingivalis, and F. nucleatum, or even polymicrobial aggregates, are able to penetrate periodontal tissues, avoiding many of the defense mechanisms of the macroorganism, which in turn affects the resistance of inflammation and the progression of periodontal tissue destruction. Keratinocytes, which make up the majority of the gingival epithelium, are capable of producing and secreting various mediators of the immune response, including human P-defensins, inflammatory cytokines,

chemokines, and angiogenetic proteins. In healthy gums, the genetically determined response is mainly regulated by keratinocytes and neutrophils.

The connective tissue of the gum, the periodontal ligament and the organic component of the bone are formed mainly from collagen. Fibroblasts are responsible for the synthesis of new collagen bundles, and they remove old collagen by secreting matrix metalloproteinases. Overexpression of metalloproteinases by gingival fibroblasts can induce the release of cytokines and chemokines from the extracellular matrix or cleave cytokines and interrupt the cascades of the immune response. The interaction between neutrophils and gingival fibroblasts is a good example of bidirectional interactions between resident and immune cells. Neutrophils form the primary defense system in periodontal tissues. It is noteworthy that their migration through the connective epithelium into the gingival groove is a continuous process that may differ from other organs where transmigration is a distinctive feature of the infectious process. Inflammatory phenotypes of neutrophils are present in periodontal diseases. Severe forms of CVD may be associated with diseases and defects in neutrophil function, such as leukocyte adhesion deficiency. The lack of neutrophil surveillance of bacterial infection is considered to be the cause of periodontal deterioration with a deficiency of neutrophilic functions. Peripheral blood neutrophils in patients with CVD secrete higher levels of inflammatory cytokines and reactive oxygen species compared to people with healthy periodontitis, and this hyperinflammatory response persists even after successful treatment with CVD. Inflammatory cytokines, chemokines, and antimicrobial peptides produced by keratinocytes, fibroblasts, and dendritic cells are chemoattractant gradients for neutrophils that migrate into inflamed tissues and stimulate chemotaxis of non-resident cells (macrophages, lymphocytes, plasma, and mast cells) to the site infections.

Conclusion. Phagocytic cells are mainly aimed at eliminating the invasion of pathogenic microorganisms by producing and secreting antimicrobial agents, reactive oxygen species, and enzymes. Nevertheless, high concentrations of collagenolytic enzymes and elastase in tissues activate the degradation of type I collagen in connective tissue and periodontal ligament. Irreversible periodontal destruction occurs when an infiltrate of inflammatory cells, predominantly containing plasma cells, spreads deeper into the connective tissue, leading to tissue damage in the periodontal ligament and alveolar bone. Alveolar wall resorption is the main pathological characteristic of periodontitis. Activation of osteoclasts, multinucleated bone-resorbing cells, is regulated by a cascade of inflammatory proteins (cytokines) and enzymes. Interleukins are the main inflammatory cytokines in the activation of osteoclastogenesis, which is achieved by increasing the expression of the nuclear factor receptor, kappa ligand, and inhibiting osteoblast differentiation, as well as reducing osteocalcin production and bone formation.

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