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Laser Therapy in the Treatment of Inflammatory Periodontal Diseases

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Relevance of the study. 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. Certain demographic characteristics, such as age, gender, ethnicity, and socioeconomic status, affect the prevalence of CVD. Predisposing factors include smoking, diabetes mellitus, metabolic syndrome, and obesity. It is noteworthy that smoking and diabetes can provoke a progressive form of periodontal disease in people as early as adolescence and early adulthood before the age of 40. In addition, periodontal diseases, as well as tooth loss, are believed to be associated with various chronic diseases and conditions that affect overall health. Protection against inflammation is the cellular-humoral system, which is constantly present in the periodontium, maintaining a balance between the microbial biofilms of the oral cavity and the macroorganism. This constant communication regulates the active immune response, which is a mutual, synergistic and dynamic interaction. In periodontal disease, the immune response has the following characteristics: the first interaction with microbes is associated with a non-specific innate response, while prolonged pathogenic infection activates specific adaptive responses. Plaque accumulation leads to inflammation with varying proportions of aerobic and often anaerobic microbial species. The presence of microbes with pathogenic potential in the gingival sulcus initiates an inflammatory response. Further, this inflammation becomes chronic, which can have serious consequences in periodontal tissues. Interactions between the components and metabolic activity of the oral microbiota and the macroorganism either maintain balance (homeostasis) or lead to disruption (dysbiosis) within the microbiota. Commensal microorganisms that ensure periodontal health are important for protecting the balance, ensuring it, for example, by inhibiting the growth of CVD-related pathogens. However, qualitative and quantitative changes in subgingival biofilms can lead to a violation of homeostasis, which contributes to the occurrence of diseases with varying degrees of destruction of periodontal tissues. This gradual maturation and change in microbial composition affects the pathogenicity of subgingival biofilms. F. nucleatum is involved in both the initiation and progression of periodontal diseases. In a biofilm, this anaerobe can survive and increase the number of colonies under anaerobic conditions. For the occurrence of periodontal diseases, not only the presence of a periodontal pathogen plays an essential role, but also the interaction between the composition of the subgingival biofilm and the reaction of the macroorganism, where immune factors play an important role. Dysbiotic biofilms contain a large number of immunostimulating pathobionts and their virulence factors, and the inhibitory effect of commensal bacteria decreases, which leads to an increased inflammatory response. In the gingival epithelium, cellular reactions are particularly manifested against polymicrobial biofilms due to their inter-bacterial synergism and virulence, leading to the initial loss of the gingival ligament and the formation of a pocket. The

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growth of colonies of inflammatory periodontal pathogens and pathobionts contributes to the aggravation of the condition of periodontal pockets with an anaerobic environment, inflammatory conditions and a large number of substrates resulting from tissue destruction. It is noteworthy that daily smoking contributes to further disorders in the microbiota, contributing to an abundance of periodontal pathogens and a decrease in beneficial commensals, thereby exposing smokers to the risk of CVD.

Due to the constant interaction with bacteria, immune cells (neutrophils, macrophages and lymphocytes) are present in periodontitis, which are involved in maintaining a healthy balance of the oral cavity. Neutrophils continuously migrate through the epithelium of the junction into the gingival sulcus and secrete antimicrobial peptides (a-defensins) against invasive bacteria, at the same time they stimulate the adhesion and spread of keratinocytes to the tooth surface [22]. Periodontal resident cells (keratinocytes, fibroblasts, dendritic cells, and osteoblasts) are passive barriers against bacterial invasion, initiate an innate immune response, and regulate the adaptive immune response. An important component is the migration of components of the complement system, which activates, enhances and synchronizes the innate immune response by opsonizing and destroying bacteria, as well as activating mast cells, neutrophils and periodontal macrophages. Neutrophils have a relatively short lifespan and are programmed to die through apoptosis. Apoptotic neutrophils are phagocytized from tissues by macrophages and excreted through the lymphatic system. Since neutrophils produce and secrete significant amounts of inflammatory molecules, their removal is a hallmark of healing. In inflamed periodontal tissues, partly due to pathogenic biofilm, increased neutrophil recruitment and delayed apoptotic cell death occur. However, instead of enhanced elimination of pathogens, neutrophils exhibit impaired antibacterial function with uncontrolled and prolonged activation of the immune response. Tissue macrophages originate either from circulating monocytes or from progenitors. Their phenotyping as inflammatory and absorbable macrophages determines their role in diseases and health. Inflammatory macrophages produce and secrete a large group of cytokines (1B-1p, GB-23, GB-6, tumor necrosis factor, and enzymes) that are involved in osteoclastogenesis and collagen degradation in CVD. The transformation from a destructive inflammatory phenotype to an absorbable and bone-forming phenotype requires the presence of apoptotic neutrophils. gingivalis can reverse the conversion of inflammatory macrophages into absorbable macrophages by inducing inflammatory cytokines. Impaired neutrophil elimination by macrophages and defects in the activation of absorbable macrophages lead to the onset, progression and recurrence of periodontitis.

Thus, periodontitis is a complex disease with a nonlinear course, and its effect on the immune response is variable and often disproportionate. Although knowledge about the functions of immune cells has increased significantly, it is still difficult to fully understand the cellular interactions in the pathogenesis of periodontal diseases due to the multifactorial etiology of CVD. The connective epithelium forms a unique structured cellular seal between the surface of the root and the gum, and its main function is to protect the underlying tissues from the constant effects of oral microbes and their metabolic products. Various molecular factors involved in adhesion, intercellular interactions, chemotaxis, inflammatory cytokines, epithelial growth, and the production of antimicrobial peptides determine the functioning of the epithelial junction. If this adapted protection system is overloaded with bacterial virulence factors (for example, P. gingivalis) and clinically, prolonged inflammation is observed (bleeding gums and changes in the contour and color of soft tissues), the destruction of collagen is activated, which ultimately leads to the resorption of the gingival ligament and the formation of a periodontal pocket.

Conclusion. It should be noted that although gum inflammation is a precursor to periodontitis and a clinically significant risk factor for disease progression, gingivitis does not always transform into periodontitis. During the formation of the periodontal pocket, the formation of new tissues by resident cells (keratinocytes, fibroblasts, osteoblasts) is suppressed, while tissue degradation by neutrophils, macrophages, and osteoclasts is stimulated; thus, the balance between tissue elimination and regeneration is disrupted.

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