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Reducing the Risk of Inflammation Associated with Cardiovascular Diseases by Treating Aggressive Periodontitis

Nurmatov Ortiq Sattor o'g'li

independent applicant

Samarkand State Medical University

Abstract: In this article, the author pays special attention to reducing the risk of inflammation associated with cardiovascular diseases by treating aggressive periodontitis. It should be noted that one of the pressing problems of modern dentistry is the study of the negative systemic effect of long-term inflammatory diseases of the oral cavity, especially with regard to periodontal disease, since, according to WHO statistics, the world population over 45 years of age has this type disease and its consequences.
Key words: Periodontitis, inflammation, cardiovascular diseases.

Periodontitis is a chronic microbial infection affecting the soft and hard tissues supporting the teeth. Its prevalence ranges from 30% to 55%, increasing with age and peaking in the fifth and sixth decades of life. The chronic inflammatory reaction in periodontitis is caused by persistent subgingival bacterial deposits, which gradually accumulate and, in most cases, do not cause painful sensations. Without treatment, periodontitis can lead to loss of supporting bone and connective tissue around the teeth, resulting in loosening and ultimately tooth loss. The consequences of periodontitis can deteriorate the quality of life and induce psychological disorders due to aesthetic impairment and reduced chewing function.

The pathophysiology of atherosclerosis has traditionally been explained by the accumulation of lipid deposits on the surfaces of arterial walls, leading to reduced or complete blockage of blood flow, resulting in cardiovascular diseases such as myocardial infarction or stroke. There is increasing evidence that inflammation plays a key role at all stages of atherosclerosis, from the formation of early lesions to thromboembolism. Elevated levels of inflammatory markers, particularly C-reactive protein (CRP), are associated with increased risk of atherosclerosis, and in survivors of myocardial infarction (MI), there is an increased risk of recurrent infarction and death from ischemic heart disease (IHD). Thus, reducing this inflammatory risk is an integral part of improving cardiovascular disease (CVD) outcomes.

Research on the outcomes of anti-inflammatory treatment of thrombosis with canakinumab has demonstrated that its impact on inflammation through direct inhibition of interleukin-1β in survivors of acute MI with elevated CRP levels ≥2 mg/L led to a moderate reduction in major adverse cardiovascular events. However, canakinumab is not economically efficient, and serious adverse events (SAEs) such as sepsis and fatal injection were reported in this study. Another anti-

inflammatory drug for reducing CVD events is colchicine, an antitubulin drug commonly used in treating gout. Colchicine has a broader mechanism of action compared to canakinumab and includes inhibition of tubulin polymerization, leading to suppression of inflammatory pathways and alteration of leukocyte reactivity. It has been specifically shown that colchicine reduces high-sensitivity CRP (hsCRP) levels, which is an established marker of future CVD development. Furthermore, randomized controlled trials (RCTs) of low-dose colchicine (0.5 mg) in patients with chronic coronary artery disease in the LoDoCo and COLCOT studies have shown that while colchicine reduces the overall risk of CVD in survivors of MI, there was no significant impact on cardiovascular mortality or MI. Similar studies of nonspecific anti-inflammatory treatment with methotrexate found that it had no effect on cardiovascular outcomes and did not reduce inflammatory markers. Therefore, there is currently no evidence to support the routine use of anti-inflammatory drugs for secondary prevention of CVD, raising questions about the justification of their future application.

While the impact of colchicine on hsCRP is established, its influence on lipids is inconclusive. A short-term study (30 days) of colchicine did not demonstrate a significant effect on lipid levels in patients with chronic coronary artery disease. However, in a diet-induced hyperlipidemia model in rodents, 5 weeks of colchicine treatment reduced plasma lipid content. Thus, further clinical studies are warranted.

Periodontal therapy aims to reduce inflammation, and there is moderate evidence supporting the reduction of serum inflammatory markers, such as CRP, following specialized periodontitis treatment. Although current data do not suggest that periodontal therapy lowers plasma lipid levels, periodontitis has been associated with dyslipidemia, thus warranting further clinical research into the effects of periodontal tissue treatment on plasma lipids.

Cardiovascular diseases (CVD) encompass disorders of the heart and blood vessels and stand as a leading cause of mortality and morbidity today. By the time CVD is detected, its primary cause (atherosclerosis) typically resides in advanced stages. Thus, prevention through risk factor modification, including proper nutrition, physical activity, and smoking cessation, is paramount. Contemporary medical research suggests a potential link between the development of periodontitis and CVDs. According to one theory, bacteria and acute-phase inflammatory proteins present in gingival tissue may enter the bloodstream, triggering adverse effects on the cardiovascular system.

Diagnosing periodontitis and devising a treatment plan necessitates a comprehensive assessment of the patient's health. For patients at risk or with a history of cardiovascular diseases, evaluating the stage and duration of the disease, presence of comorbidities such as diabetes mellitus that may influence the course of CVD, and assessing concurrent risk factors for periodontal diseases are essential. Additionally, dentists may consult with attending physicians to determine the severity of the case, plan the treatment course, and evaluate the patient's overall health status.

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