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Interrelation of paradont tissue diseases with cardiovascular pathology (Literature review)

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Abstract

The relationship between periodontal diseases and cardiovascular disease is addressed in this article. Both these diseases have an inflammatory basis. Because periodontal disease is a risk factor for developing cardiovascular disease, diagnosis of the former is important. Particular attention must be paid to patients who have periodontal disease with other risk factors for atherosclerotic vascular disease. Recommendations managing these patients have been made included.

Keywords: periodontal disease, atherosclerosis, arterial hypertension, epidemiological data

1. Introduction

For many years, cardiovascular disease (CVD) is the leading cause of death in many economically developed countries. In the world, the mortality caused by cardiovascular pathology was about 55% of the total number of deaths (over half a million people) [8].

CVD is often accompanied by various changes in the organs and tissues of the oral cavity. Chronic inflammatory periodontal diseases (gingivitis, periodontitis) occupy the second place after caries after the prevalence among dental pathology. According to the World Health Organization, over 60% of the population of European countries and up to 50% of the US population have signs of chronic inflammation of the gums. Parodontitis is a disease of the dentoalveolar system, characterized by the development of acute or chronic inflammation, destruction of periodontal tissues and atrophy of bone tissue of the alveoli. The American Academy of Periodontology parodontitis is considered as an inflammatory disease of bacterial origin [3].

2. The purpose of the paper is aimed at familiarization of broad medical public with the presence of the relationship between diseases of periodontal tissues and cardiovascular disease. A thorough comprehensive analysis and generalization of scientific achievements elucidated in the fundamental and periodical publications, relating to diseases of the periodontal tissues and cardiovascular disease, has been carried out.

3. Results and their discussion

A significant contribution to the etiopathogenesis of periodontal diseases is made by somatic diseases: CVD, diabetes mellitus (DM), diseases of the digestive system, systemic osteoporosis, respiratory tract diseases. The most active in the literature in recent decades is the relationship between periodontal diseases and CVD [7].

Periodontal and cardiovascular diseases have many common risk factors: metabolic syndrome, diabetes, dyslipidemia, arterial hypertension (AH) [1, 5].

Of the diagnostic criteria of the metabolic syndrome, the clearest connection of the pathology of the periodontal disease is noted with abdominal obesity. The close relationship between diabetes and periodontal disease is well known and is based on numerous studies in the 1990s that allow treating periodontitis as one of the major complications of diabetes [2].

The results of numerous studies suggest that dyslipidemia, especially the increase in triglycerides, total cholesterol and low-density lipoprotein cholesterol, may be associated with periodontitis in somatically healthy people [11].

Epidemiological data indicate a potential association of periodontitis with an increase in blood pressure and prevalence of hypertension.

Data from cross-sectional studies suggest that with AH, the presence of periodontal disease in patients may increase the risk and extent of damage to target organs. According to the results Korean study, the authors suggest to consider the hygienic state of the oral cavity as an independent indicator of the risk of hypertension [9].

In a large number of studies, there is a definite positive relationship between clinical manifestations and inflammatory changes in periodontal diseases with atherosclerosis and CVD.

The results of a study in Spain indicate a positive relationship between the degree and severity of periodontitis with acute myocardial infarction and its size. The INVEST study (Oral Infections and Vascular Disease Epidemiology Study) has added new data to the already significant volume of epidemiological evidence linking CVD and periodontal disease. Observation for 3 years for 420 participants showed that the progression of intima-media thickness of the carotid artery was weakened with the improvement of the clinical or microbiological state of periodontal disease [3].

A number of in vitro and in vivo studies suggest a possible association between oral bacteria and atherosclerosis. In serological studies, high titres of antibodies to periodontal bacteria in atherosclerosis and CVD were noted. Viable periodontal bacteria in a number of studies are isolated directly from an atherosclerotic plaque [2].

Periodontitis is considered as a factor contributing to the development of systemic inflammation as a result of ingestion of bacteria and inflammatory / proinflammatory cytokines into the blood, which, accordingly, can affect other organs and systems of the body and, in the first place, the vascular endothelium. Elevated levels of C-reactive protein, which accompanies periodontal inflammation, in turn, 4 times increases the risk of patients developing cardiovascular events [10].

To date, the available data do not allow one to unequivocally assess the nature of the relationship between the periodontal pathology and CVD and answer the question posed in the title of the article. However, the presence of these relationships must be remembered and the dentist and internists. For example, the consensus on periodontitis and atherosclerotic cardiovascular diseases, published in the American Journal of Cardiology and the Journal of Periodontology, recommends that patients with moderate and severe periodontitis be informed of a possible increased risk of CVD and the need for a cardiac examination [6].

It is necessary to single out one more aspect of the relationship between CVD and periodontitis - effective therapy of periodontal diseases reduces the risk of developing new cardiovascular events.

Inflammatory periodontal diseases refer to infectious chronic inflammatory diseases, so the normalization of microflora is an indispensable condition for their rational treatment. It is known that with chronic gingivitis and periodontitis there is a distinct shift towards the predominance of anaerobic flora - as the number of strains of anaerobic bacteria increases in inflammation in periodontal pockets up to 70-80%, whereas in normal anaerobes the amount does not exceed 20-30% [4].

With the advent of local anti-inflammatory drugs, as A.I. Grudyanov notes, the therapy of inflammatory periodontal diseases has become much more effective. The main directions in the treatment are regularly conducted hygienic treatment and the appointment of drugs that effectively inhibit the activity of microorganisms that slow down the formation

of microbial clusters and have an anti-inflammatory effect.

A certain influence on the condition of periodontium can also be provided by drugs used to treat CVD diseases. The most significant side effects of selective calcium channel blockers (nifedipine, amlodipine, lacidipine, nimodipine, verapamil, diltiazem, etc.) are the most significant side effects for gum disease. Hyperplasia of the gums (bleeding, soreness, swelling) and hypertrophic gingivitis become [2, 5].

With the use of acetylsalicylic acid, clopidogrel, ticlopidine, warfarin, unfractionated heparin, low-molecular heparins (supraparin, dalteparin, enoxaparin, bemiparin, repyvarin), fondaparinux sodium, rivaroxaban, dabigatran etexilate, abciximab, eptifibatide, there may be increased bleeding gums. Thrombolytic therapy (streptokinase, alteplase, tenecteplase, prourokinase) may also be the cause of gingival hemorrhage.

The positive effects of drug therapy for CVD on periodontal conditions are associated with drugs of the statin group. Statins cause the following systemic (pleiotropic) effects: improvement of the functional state of the endothelium (restoration or improvement of endothelium-dependent dilatation); normalization (improvement) of rheological and reduction of thrombogen-forming properties of blood [9, 11].

Anti-inflammatory effect of statin therapy is provided by such mechanisms as improvement of endothelial function due to increase in NO level, stabilization of atherosclerotic plaque, difficulty of thrombus formation (due to reduction of platelet aggregation and reduction of fibrinogen level, inhibitor of tissue activator of type 1 plasminogen). A number of studies have shown that statins reduce the concentration of C-reactive protein and can reduce the secretion of certain cytokines: interleukin-6, tumor necrosis factor α . A systematic review using the PUBMED and BIREME databases noted that statins reduce bone resorption by inhibiting the formation of osteoclasts and may lead to an increase in the apoptosis of these cells.

4. Conclusion

In conclusion, it should be noted that a number of recent studies have clearly demonstrated the following: timely treatment of periodontal diseases (gingivitis, periodontitis), aimed primarily at controlling the local inflammatory response, reduces the risk of development and progression of pathological changes such as atherosclerosis and ischemic disease heart, and subsequently acute myocardial infarction and stroke.

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