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CARDIOVASCULAR DISEASES IN THE PRACTICE OF A DENTIST

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ABOUT ARTICLE

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Abstract: More than a century ago, a link was discovered between oral infections, tooth extraction and cardiovascular diseases (infectious endocarditis), and by the middle of the 20th century, the practice of routine prescribing antibiotics for the prevention of endocarditis before invasive dental procedures appeared Since 1950, the theory of infection development associated with infection of the alveolar bone system, The etiological role was also expanded to several other anatomical areas not related to the oral cavity.

INTRODUCTION

More than a century ago, a link was discovered between oral infections, tooth extraction and cardiovascular diseases (infectious endocarditis) [1], and by the middle of the 20th century, the practice of routine prescribing antibiotics for the prevention of endocarditis before invasive dental procedures appeared [2] Since 1950, the theory of infection development associated with infection of the alveolar bone system, The etiological role was also expanded to several other anatomical areas not related to the oral cavity. In the last two decades, oral infections have again been considered as a cause of systemic diseases, with special attention being paid to their possible connection with periodontal diseases (PO) and atherosclerotic vascular diseases (AZS) [3, 4]. The purpose of this review is to inform dentists about the factors influencing the development of atherosclerotic heart disease in periodontal diseases, without taking into account their association with other diseases such as spontaneous

abortion, low birth weight, chronic obstructive pulmonary disease (COPD), complications of diabetes, osteoporosis, glomerulonephritis, rheumatoid arthritis, keratitis and retinitis. in the past [5, 6]. Periodontal diseases Periodontal diseases are a serious public health problem [7, 8]. Periodontal diseases have a high prevalence, reduce the quality of life and chewing function, negatively affect aesthetics, lead to disability and tooth loss, are the cause of most cases of complete tooth loss, have economic consequences and can have a negative impact on overall health, including contributing to the development of atherosclerotic vascular disease. This is a chronic disease [9]. Periodontal diseases can be prevented by observing oral hygiene and performing preventive measures such as brushing teeth, flossing and regular professional oral hygiene (ultrasonic cleaning of roots and teeth followed by cleaning and polishing). Periodontal disease is a group of diseases resulting from the accumulation of plaque, with or without the development of destruction and inflammation of the periodontal ligamentous apparatus, including the gum, periodontal ligament and alveolar bone. Clinically, the gingival sulcus deepens, forming gingival pockets, gum attachment to the root surface is disrupted, and the biofilm on the tooth surface migrates to the apical part of the root, which leads to the destruction of connective tissue, loss of alveolar bone and loss of gums [10]. Various microorganisms colonize the glycoprotein-containing layer above and below the gum (plaque), forming a subgingival plaque layer. Supradingival plaque is mainly colonized by Streptococcus sanguis, Streptococcus oralis, Streptococcus mutans, Actinomyces naeslundii and Actinomyces odontolyticus. Subsequently, secondary colonizers such as Fusobacterium nucleatum are added to them, and as a result, a collection of millions of gram-positive and gram-negative bacteria and cocci forming a biofilm is formed. Over time, the microflora at the gum attachment site changes from predominantly gram-positive to predominantly Gram-negative bacteria, including more paraphyletic bacteria, Porphyromonas gingivalis, Tannerella forsythia forsythia), Treponema denticola, Selenomonas noxia, Campylobacter rectus, Agrigatibacter (formerly Actinobacillus Aggregatibacter (Actinobacillus) Actinomycetemcomitans microaerophiles), Prevotella intermedia and Spirochetes. Spirochetes) and other anaerobic gram-negative microorganisms. In addition, Chlamydophila pneumoniae, Mycoplasma, Helicobacter pylori, Candida species, Epstein-Barr virus, cytomegalovirus, herpesviruses, amoebas, methane-producing protocaryotic microorganisms (methanogenic bacteria related to archaea) and sulfate-reducing bacteria (SRB) are found in the periodontal pocket. The following microorganisms are found in periodontal pockets. In periodontal pockets, there is an interaction between bacteria and endothelial cells, signals are generated and an exchange takes place between microorganisms and neighboring cells of the immune system. Inflammatory cytokines and chemokines are isolated, attracting dendritic cells, T and B lymphocytes, macrophages and neutrophils, which are involved in innate and acquired immune reactions and

inflammatory processes [10]. Microorganisms that are considered to be involved in PD, such as Porphyromonas gingivalis, Aggregatibacter (formerly Actinobacillus) actinomycetemcomitans and Prevotella intermedia, enter the epithelial cells lining the gingival sulcus by receptor-mediated endocytosis. They are attracted and absorbed. Thus, microorganisms producing endotoxins (for example, lipopolysaccharides), such as Porphyromonas gingivalis, are protected from the immune system, multiply intracellularly and in some cases spread systemically through the blood circulation, potentially causing a generalized immune response [11]. Cardiovascular diseases Cardiovascular diseases, which are based on atherosclerosis, are the most common cause of morbidity and mortality among the adult population worldwide [12]. Atherosclerotic vascular diseases cause ischemic heart disease (stable angina pectoris and acute coronary syndrome (ACS)), ischemic cerebrovascular disease (cerebrovascular disease (CVD) or stroke and transient ischemic attack (TIA)) and peripheral vascular disease (SDS). The process of atherosclerosis begins with the accumulation of lipids in the subendothelial layer of the arterial wall, followed by oxidation and/or formation of biologically active substances, including malondialdehyde (MDA) - modified low-density lipoproteins (MDA-LDL). Circulating monocytes also adhere to the vascular endothelium and differentiate into macrophages that absorb lipoproteins (xanthoma (foam) cells). They accumulate in the form of fat bands and grow into more complex fibrous-fatty plaques. Monocytes and T cells indirectly adhere to the surface of endothelial cells through chemical intermediaries such as type 1 intercellular adhesion molecule (ICAM-1), type 1 endothelial leukemic cell adhesion molecule (ELAM-1) and vascular endothelial adhesion molecule (VCAM-1). The foam cells eventually die, leaving necrotic nuclei rich in lipoprotein in the artery wall, which temporarily migrate and proliferate in the smooth muscle cells of the artery wall in the intimate layer (Tunica intima). Atherosclerotic plaques are foci of chronic inflammation. The main adverse cardiovascular events, NMC and coronary heart disease, occur in people who develop atherosclerotic plaques in the carotid and coronary arteries. They occur when the fibrous membrane of an unstable atherosclerotic plaque ruptures, exposing the tissue under the intima of the vessel and causing partial or complete occlusion of the vessel as a result of platelet aggregation and in situ thrombus formation. Clinically, SLE reflects the severity of established angina pectoris, primary angina pectoris, or electrocardiographically/biochemically diagnosed extensive myocardial ischemia or myocardial necrosis. Random data Many observational (case-control, cohort and cross-sectional), epidemiological and invasive studies confirm the link between clinically diagnosed periodontal diseases and coronary heart disease. A recent review (published in 2012) was conducted by a working group of the American Heart Association (AHA). They conducted an electronic literature search on the relationship of periodontal diseases with cardiovascular diseases (with the exception of infectious

endocarditis), Behcet syndrome, Stevens-Johnson syndrome and Sjogren syndrome. A total of 537 articles published in dental/periodontological (61%) and medical journals (39%) were analyzed [10]. An overview of data on the definition and prevalence of cardiovascular diseases and periodontal diseases, anatomy, pathophysiology and microbiology of periodontal diseases, risk factors and pathogenesis of cardiovascular diseases and periodontal diseases, indirect and direct pathogenetic mechanisms, as well as the relationship between cardiovascular diseases and periodontal diseases was presented. It was concluded that "periodontal diseases are associated with atherosclerosis regardless of somatic pathology," but there is no evidence of a causal relationship, therefore, "statements suggesting a causal relationship between periodontal diseases and specific cases of atherosclerotic vascular damage are unfounded" [10]. In 2013, a consensus statement was published by the working group of the European Federation of Periodontology and the American Academy of Periodontology (EFP/AAP) [9]. It presented the results of biological, epidemiological and first post-invasive studies. A review of longitudinal studies evaluating the occurrence of cardiovascular events was conducted, in which a statistically significant excess of the risk of developing ACCP in patients with periodontal diseases was revealed, independent of established cardiovascular risk factors. Given the high prevalence of periodontal disease, this risk factor has been recognized as important from a public health perspective. Only moderate evidence has been found that treatment of periodontal disease reduces CRP levels and improves endothelial function. There was no evidence of an effect on the lipid profile, but biomarkers of blood clotting and endothelial cell activity, blood pressure and subclinical atherosclerosis are improved in the treatment of periodontal diseases. The Working Group came to the following conclusion: "2. In vitro animal studies and clinical studies confirm the presence of interactions and biological mechanisms, however, invasive studies conducted to date are insufficient to draw any further conclusions; 3. There is no clear evidence of the effect of periodontal treatment on the prevention of ASP, while there is no clear evidence. Well-planned invasive studies with clinical results are needed. [9]. Other authors conducted a systematic review of epidemiological data on the association between PD and ACCP [16]. All types of longitudinal (cohort or case-control) clinical trials conducted in English- and German-speaking countries were considered. The analysis included only those studies in which periodontal probing, clinical loss of communication and/or X-ray assessment of alveolar bone loss were used as endpoints. To compare the risk of developing ACCP in patients with AF with patients without AF, the risk of developing coronary heart disease (CHD, stable angina pectoris, CHD and death) was first assessed. To assess the association between ST and ACCP, only studies were included that assessed relative risk (risk, frequency, hazard ratio, odds) and controlled for the mixed effect of age and gender. A total of 12 studies were selected for the review (six for coronary heart disease, three for

cardiovascular exacerbations, two for coronary heart disease and cardiovascular exacerbations, and one for peripheral vascular diseases). It was concluded that, despite the evidence of an increased risk of ACC in patients with AF compared to patients without AF, these data cannot be applied to all populations.The relationship between CT and ACC Inflammation is a component of the pathobiology of atherogenesis, as well as a trigger for plaque rupture. Inflammation is also a trigger for plaque rupture. In addition, inflammation is an integral part of the etiology of periodontal diseases. This is due to the fact that inflammation anywhere in the body causes an influx of inflammatory mediators into the systemic environment. Thus, if inflammatory processes are present simultaneously, they can affect each other. Several markers can be used to assess systemic inflammation: the inflammatory response characteristic of the acute phase, indicated by an increase in the level of CRP and highly sensitive CRP (hsCRP), is associated with many chronic diseases and is used to predict mortality from cardiovascular diseases and the risk of myocardial infarction, the Framingham Risk Scale, which is part of the Framingham Risk Scale [15]. However, it is unknown whether this acute phase protein is simply a marker of the inflammatory process or whether it is involved in the pathophysiology of atherosclerosis [13]. Acute phase proteins are present in newly formed atheromatous plaques and are localized along with activated complement; the U.S. Preventive Services Task Force concluded in its systematic review that there is strong evidence that CRP is associated with the development of coronary heart disease, but that CRP should be directed to treatment. As a result of this review, the U.S. Disease Prevention Task Force concluded that there is insufficient evidence that C-reactive protein should be targeted for treatment [15]. Several studies have shown that in patients without a history of SLE, a single routine measurement of CRP can predict future vascular events such as acute coronary syndromes, cardiovascular attacks, peripheral vascular disease and sudden cardiac death. Thus, CRP is an independent marker of future cardiovascular diseases and adds predictive information. Another biomarker of inflammation that is constantly associated with cardiovascular risk is myeloperoxidase (MPO), an enzyme expressed on leukocytes and associated with both inflammation and oxidative stress. Both processes play an important role in the pathobiology of coronary heart disease. Metalloproteinases, such as lipoprotein-associated phospholipase A2, tissue inhibitor of matrix metalloproteinases, fibrinogen, interleukin 6 [IL-6], soluble intercellular adhesion molecule type 1, macrophage cytokine-1 inhibitor and soluble ligand forms, are involved in the lysis of the fibrous membrane of atheromatous plaques, leading to the development of the most common cardiovascular diseases CD40 [10]. Levels of systemic markers of inflammation, including CRP, tumor necrosis factor (TNF-a), IL-1, IL-6 and IL-8, are increased in periodontal diseases. As a result, periodontal diseases, as well as chronic infection, contribute to an increase in the level of chronic inflammatory factors due to

local tissue destruction, an immuno-inflammatory response to periodontal pathogens and the systemic spread of inflammatory mediators secreted by these pathogens and their products. This can contribute to both the development of atherosclerosis and the rupture of plaque in patients with periodontal diseases. Discussion Epidemiological studies on the role of periodontal diseases as an independent risk factor for SLE have been reviewed by reputable organizations such as the American Heart Association (AHA) and the European Federation of Periodontology (EFP)/The American Academy of Periodontics (AAP), as well as other authors [9, 10, 16]. The authors who conducted these studies concluded that there is a clear relationship between these two diseases, but this statement is not reliable for all population groups.

RESULT

The results of the available studies show a tendency for periodontal interventions to affect systemic inflammation and some markers of ACCP and endothelial function, but this is not consistent with all the studies that we analyzed. In addition, transient inflammation and decreased endothelial function were observed after exacerbation of periodontal diseases. Since there are significant gaps in our knowledge of the relationship between periodontal disease and ACCP, more fundamental, epidemiological and invasive studies are needed. Due to public health concerns about this problem, all medical professionals should be aware that periodontal diseases are a risk factor for SLE, and the prevention, diagnosis and treatment of periodontal diseases are of paramount importance. In addition, patients with periodontal disease who have other risk factors for developing ACCC, such as hypertension, obesity, smoking, dyslipidemia, diabetes and a burdened family history, and who have not visited a therapist in the last 12 months, should be advised to do so. Patients with periodontal disease or modifiable lifestyle-related risk factors for ACCC should be consulted at their dental clinic about these factors as part of a comprehensive periodontal treatment plan. If possible, they should also be advised on smoking and obesity prevention programs, preferably in conjunction with a doctor from another department. For patients with periodontal diseases at risk of developing infectious endocarditis, treatment in accordance with existing recommendations should be considered before future dental treatment [2].

CONCLUSION

The above measures and approaches, taken jointly with representatives of all medical departments, will contribute to the maximum improvement of the dental and general health of the patient.

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