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RESEARCH OF CARDIOVASCULAR DISEASES IN THE PRACTICE OF A DENTIST

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ABOUT ARTICLE

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Abstract: More than 100 years ago, a link was discovered between oral infections, tooth extractions, and cardiovascular disease (infective endocarditis) [1], and by the mid-20th century the practice of routinely prescribing antibiotics to prevent endocarditis prior to invasive dental procedures appeared [2]. In the last two decades, oral infections have again been considered a cause of systemic disease, with particular attention focused on their association with periodontal disease (PO) and atherosclerotic vascular disease (AZS) [3, 4].

INTRODUCTION

The purpose of this review is to inform dentists about the factors that influence the development of atherosclerotic heart disease in periodontal disease without considering the association of periodontal disease 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. This is to inform them. Periodontal Disease Periodontal disease is a serious public health problem [7, 8]. Periodontal disease has a high prevalence, decreases quality of life and masticatory function, adversely affects esthetics, leads to

disability and tooth loss, is the cause of most cases of complete tooth loss, has economic consequences, and can adversely affect systemic health, including contributing to the development of atherosclerotic vascular disease. is a chronic disease. It is a chronic disease [9]. Periodontal disease can be prevented by adhering to good oral hygiene and taking preventive measures such as brushing, 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 or inflammation of the periodontal ligament apparatus, which includes the gingiva, periodontal ligament, and alveolar bone. Clinically, the gingival sulcus deepens, gingival pockets form, gingival attachment to the root surface is destroyed, and biofilm on the tooth surface migrates to the apical portion of the root, leading to connective tissue destruction, alveolar bone loss, and gingival loss [10]. Various microorganisms settle in the glycoprotein-containing layer (plaque) above and below the gingiva, forming the subgingival plaque layer. Supragingival plaque is colonized primarily by Streptococcus sanguis, Streptococcus oralis, Streptococcus mutans, Actinomyces naeslundii and Actinomyces odontolyticus Streptococcus mutans, Actinomyces naeslundii and Actinomyces odontolyticus. Subsequently, secondary colonizers such as Fusobacterium nucleatum are added, resulting in the formation of an aggregate of millions of Gram-positive and Gram-negative bacteria and cocci that form a biofilm. Over time, the microbiota at the gingival attachment site changes from a predominance of Gram-positive bacteria to a predominance of Gram-negative bacteria, including a greater number of commensal bacteria, Porphyromonas gingivalis, Tannerella forsythia forsythia), Treponema denticola, Selenomonas noxia, Campylobacter rectus, Agrigatibacter (formerly Actinobacillus Aggregatibacter (Actinobacillus) Actinomycetemcomitans microaerophiles), Prevotella intermedia, and Spirochetes). In addition, there is a wide range of other organisms such as Chlamydophila pneumoniae, Mycoplasma, Helicobacter pylori, Candida species, Epstein-Barr virus, and Cytomegalovirus. Barr virus, Cytomegalovirus, Herpesvirus, Amoebas, methane-producing protokaryotes (methanogenic bacteria related to archaea), and sulfate-reducing bacteria (SRB) are found in periodontal pockets. The following microorganisms are present in periodontal pockets. In periodontal pockets, there are interactions between bacteria and endothelial cells, signals are generated, and exchanges occur between microorganisms and neighboring cells of the immune system. Inflammatory cytokines and chemokines are isolated and attract dendritic cells, T lymphocytes, B lymphocytes, macrophages and neutrophils, which are involved in innate and acquired immune responses and inflammatory processes [10]. Microorganisms thought to be involved in PD, such as Porphyromonas gingivalis, Aggregatibacter (formerly Actinobacillus) actinomycetemcomitans, and Prevotella intermedia, invade the epithelial cells lining the gingival sulcus via receptor-mediated

endocytosis. They are attracted and absorbed. Thus, microorganisms producing endotoxins (e.g., 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 systemic immune response [11]. Cardiovascular Disease Cardiovascular disease based on atherosclerosis is the most common cause of morbidity and mortality in the adult population worldwide [12]. Atherosclerotic vascular disease causes ischemic heart disease (stable angina 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 layers of the arterial wall, followed by the oxidation and/or formation of biologically active substances, including malondialdehyde (MDA)-modified low-density lipoproteins (MDA-LDL). Circulating monocytes also differentiate into macrophages (xanthoma (foam) cells) that adhere to vascular endothelium and absorb lipoproteins. These accumulate in the form of fatty bands and grow into more complex fibrofibrous fatty plaques. Monocytes and T cells adhere indirectly to endothelial cell surfaces via chemical mediators such as type 1 intercellular adhesion molecule (ICAM-1), type 1 endothelial leukemia cell adhesion molecule (ELAM-1), and vascular endothelial adhesion molecule (VCAM-1). The foam cells eventually die, leaving behind lipoprotein-rich necrotic nuclei in the arterial wall, which migrate transiently into the smooth muscle cells of the arterial wall and proliferate in the intima (Tunica intima). Atherosclerotic plaques are foci of chronic inflammation. The formation of atherosclerotic plaques in carotid and coronary arteries results in the major adverse cardiovascular events, NMC and coronary heart disease. These occur when the fibrous membrane of the unstable atherosclerotic plaque ruptures, exposing subendovascular tissue 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 extensive myocardial ischemia or myocardial necrosis diagnosed electrocardiographically/biochemically. Random Data Many observational (case-control, cohort, and cross-sectional), epidemiologic, and invasive studies have confirmed an association between clinically diagnosed periodontal disease 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 for the relationship between periodontal disease and cardiovascular disease (excluding infective endocarditis), Behcet syndrome, Stevens-Johnson syndrome, and Sjogren's syndrome. A total of 537 articles published in dental/periodontal (61%) and medical journals (39%) were analyzed [10]. An overview of data on the definition and prevalence of cardiovascular disease and periodontal disease, the anatomy, pathophysiology, and microbiology of

periodontal disease, risk factors and pathogenesis of cardiovascular disease and periodontal disease, indirect and direct pathogenic mechanisms, and the association between cardiovascular disease and periodontal disease was presented. It was concluded that "periodontal disease is associated with atherosclerosis regardless of somatic pathology," but since there was no evidence of a causal relationship, "statements suggesting a causal relationship between periodontal disease and specific cases of atherosclerotic vascular disease are unfounded" [10]. In 2013, a consensus statement was issued by a working group of the European Federation of Periodontology and the American Academy of Periodontology (EFP/AAP) [9]. The consensus statement presented the results of biological, epidemiological, and first post-invasive studies. A review of longitudinal studies evaluating the occurrence of cardiovascular events was conducted and revealed a statistically significant excess risk of developing ACCP in patients with periodontal disease, independent of established cardiovascular risk factors. Due to the high prevalence of periodontal disease, this risk factor has been recognized as important from a public health perspective. We found only moderate evidence that treatment of periodontal disease reduces CRP levels and improves endothelial function. There was no evidence that lipid profiles are affected, but biomarkers of blood coagulation and endothelial cell activity, blood pressure, and subclinical atherosclerosis are improved with periodontal disease treatment. The working group concluded: "2. In vitro animal and clinical studies have confirmed the existence of interactions and biological mechanisms, but the invasive studies performed to date are insufficient to draw further conclusions; 3. Periodontal disease treatment is effective in the prevention of ASP There is no clear evidence that Well-designed invasive studies with clinical outcomes 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 included. Only studies that used periodontal probing, clinical communication loss, and/or radiographic assessment of alveolar bone loss as endpoints were included in the analysis. To compare the risk of developing ACCP in patients with atrial fibrillation with those without atrial fibrillation, we first assessed the risk of developing coronary heart disease (CHD, stable angina, CHD, and death) To evaluate the association between ST and ACCP, we assessed relative risk (risk, frequency, hazard ratio, odds) and age and gender Only studies that controlled for mixed effects were included. A total of 12 studies (6 coronary heart disease, 3 cardiovascular exacerbations, 2 coronary heart disease and cardiovascular exacerbations, and 1 peripheral vascular disease) were included in the review.Relationship between CT and ACC Inflammation is a trigger for plaque rupture and a component of the pathobiology of atherogenesis. Inflammation is also a trigger for plaque rupture. Furthermore, inflammation is an integral component of the etiology of periodontal disease. This is

because inflammation anywhere in the body results in an influx of inflammatory mediators into the systemic environment. Thus, when inflammatory processes are present simultaneously, they can influence each other. Several markers can be used to assess systemic inflammation: the inflammatory response characteristic of the acute phase, indicated by elevated levels of CRP and high-sensitivity CRP (hsCRP), is associated with many chronic diseases and is used to predict cardiovascular mortality and risk of myocardial infarction and is part of the Framingham Risk Scale [15]. However, it is unclear whether this acute phase protein is simply a marker of an inflammatory process or whether it is involved in the pathophysiology of atherosclerosis [13]. Acute phase proteins are present in newly formed atheromatous plaques and localize with activated complement. In its systematic review, the U.S. Preventive Services Task Force concluded that there is strong evidence that CRP is associated with the development of coronary heart disease, but that CRP should be directed toward treatment. As a result of this review, the U.S. Preventive Services Task Force concluded that there is insufficient evidence that CRP 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 disease and adds predictive information. Another biomarker of inflammation that has always been associated with cardiovascular risk is myeloperoxidase (MPO), an enzyme expressed on white blood cells and associated with both inflammation and oxidative stress. These two processes play an important role in the pathogenesis of coronary heart disease. Metalloproteinases such as lipoprotein-associated phospholipase A2, matrix metalloproteinase tissue inhibitor, fibrinogen, interleukin 6 [IL-6], soluble intercellular adhesion molecule type 1, macrophage cytokine-1 inhibitor, and soluble ligand type are all involved in the atherogenic plaque involved in the lysis of fibrous membranes, leading to the development of the most common cardiovascular disease CD40 [10].Levels of systemic inflammatory markers, including CRP, tumor necrosis factor (TNF-a), IL-1, IL-6 and IL-8, are increased in periodontal disease. As a result, periodontal disease, like chronic infection, contributes to increased levels of chronic inflammatory factors due to local tissue destruction, immune-inflammatory responses to periodontal pathogens, and systemic spread of inflammatory mediators secreted by these pathogens and their products. This may contribute to both the development of atherosclerosis and plaque rupture in patients with periodontal disease. Discussion Epidemiological studies on the role of periodontal disease 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)/American Academy of Periodontology

(AAP) and by 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 populations.

RESULTS

The results of the available studies show a trend for periodontal interventions to affect systemic inflammation and several markers of ACCP and endothelial function, which is not consistent with all the studies we analyzed. Furthermore, a transient decrease in inflammation and endothelial function was observed after exacerbation of periodontal disease. Because of the large gaps in our knowledge of the relationship between periodontal disease and ACCP, more basic, epidemiological, and invasive studies are needed. Due to public health concerns about this issue, all health care providers should be aware that periodontal disease is a risk factor for SLE, and prevention, diagnosis, and treatment of periodontal disease 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, or a burdensome family history, and who have not visited a treatment center in the past 12 months should be encouraged to do so. Patients with modifiable lifestyle-related risk factors for periodontal disease or ACCC should discuss these factors with their dentist as part of a comprehensive periodontal treatment plan. If possible, advice on smoking and obesity prevention programs should also be obtained, preferably in conjunction with other physicians. For patients with periodontal disease at risk of developing infective endocarditis, treatment according to existing recommendations should be considered prior to future dental treatment [2]. CONCLUSION: The above measures and approaches, taken in collaboration with representatives of all medical departments, will contribute to the maximum improvement of patients' dental and general health.

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