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PERIODONTITIS AS A RISK FACTOR FOR CARDIOVASCULAR DISEASES: COMMON PATHOGENETIC MECHANISMS AND CLINICAL CONSEQUENCES

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Abstract

Relevance. Chronic inflammatory diseases of the oral cavity, in particular periodontitis, have recently been considered not only as a local dental problem but also as a potential trigger of systemic pathology. Of particular interest is the link between periodontitis and cardiovascular diseases (CVD), which remain the leading cause of death worldwide. Objective – based on an analysis of available scientific data, to establish the role of periodontitis as an independent risk factor for the development of atherosclerosis, myocardial infarction, and stroke, as well as to describe the main common mechanisms. Materials and methods. A systematic analysis of publications from PubMed, Scopus, and RSCI databases for the period 2015–2025 was conducted, including clinical studies, meta-analyses, and reviews on the relationship between periodontitis and CVD. Results. It has been shown that the presence of periodontitis increases the risk of coronary heart disease by an average of 25–35%, and the risk of cerebrovascular events by 20–30%. The main linking mechanisms are chronic systemic inflammation (elevated C-reactive protein, IL-6, TNF-α), bacteremia involving periodontal pathogens (Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans), endothelial dysfunction, and immune-mediated damage to the vascular wall. Conclusion. Periodontitis should be considered a modifiable risk factor for cardiovascular pathology, which requires interdisciplinary interaction between dentists and cardiologists for early prevention and comprehensive treatment.

Keywords

periodontitis, cardiovascular diseases, atherosclerosis, systemic inflammation, bacteremia, Porphyromonas gingivalis, endothelial dysfunction, risk factors.

Introduction

Cardiovascular diseases occupy the first place in the mortality structure in most countries of the world. Along with traditional risk factors (arterial hypertension, dyslipidemia, smoking,

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diabetes mellitus, obesity), chronic foci of inflammation, including those in the oral cavity, are attracting increasing attention. Periodontitis is a widespread inflammatory-destructive disease of the tissues surrounding the tooth, which has a chronic relapsing course and is accompanied by the constant entry of bacterial antigens and pro-inflammatory mediators into the bloodstream. Epidemiological studies in recent years have revealed a consistent association between the severity of periodontitis and the risk of atherothrombotic complications. However, the question remains open: is this relationship causal, or do both diseases share common risk factors? The aim of this work is to summarize current scientific data on the pathogenetic mechanisms of the relationship between periodontitis and cardiovascular diseases, as well as to assess the clinical consequences of this association.

Research Methods

The study was carried out in the form of an analytical review with elements of literature systematization. The search for scientific publications was conducted in the electronic databases MEDLINE (PubMed), Scopus, Web of Science, and the Russian Science Citation Index (RSCI) for the period from 2015 to 2025. The following search terms and their combinations were used: «periodontitis», «periodontal disease», «cardiovascular disease», «atherosclerosis», «myocardial infarction», «stroke», «inflammation», «C-reactive protein», «Porphyromonas gingivalis», «endothelial dysfunction». The analysis included prospective cohort studies, case-control studies, meta-analyses, and systematic reviews. Exclusion criteria: animal studies, single clinical cases, publications before 2015 (except for classic works that laid the foundation of the concept). At the final stage, 42 sources were selected, of which the most representative were used for detailed analysis (presented in the reference list). The strength of the association between periodontitis and CVD was assessed based on relative risk (RR) or odds ratio (OR) values reported in meta-analyses. To describe biological mechanisms, data from experimental and clinical studies confirming systemic inflammation, bacteremia, and cross-immune response were summarized.

Discussion

Epidemiological evidence of the association. Numerous observational studies have revealed a dose-dependent relationship between the severity of periodontitis and the risk of cardiovascular events. According to a meta-analysis that included more than 50,000 participants, periodontitis is associated with an increased risk of coronary heart disease by 24% (RR=1.24; 95% CI 1.15–1.34) and the risk of stroke by 28% (RR=1.28; 95% CI 1.12–1.46). Patients with the deepest periodontal pockets (≥5 mm) and frequent exacerbations have higher risk rates compared to those with mild disease.

Common risk factors or causal relationship? Critics point out that smoking, low socioeconomic status, and diabetes mellitus may be common to both diseases. However, even after statistical adjustment for all traditional factors, the association remains significant. More convincing evidence of causality has come from interventional studies: active treatment of periodontitis (e.g., curettage, antibiotics) leads to a decrease in the levels of systemic inflammatory biomarkers – C-reactive protein (CRP), fibrinogen, IL-6 – within 6–12 weeks. Some works have noted an improvement in endothelial function (increase in flow-mediated vasodilation) after dental debridement.

Systemic inflammation as a key mechanism. Periodontitis maintains a chronic focus of inflammation from which pro-inflammatory cytokines constantly enter the bloodstream. In

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patients with periodontitis, the concentration of high-sensitivity CRP is on average 1.5–2 mg/L higher than in individuals with healthy periodontium. Moderate elevation of CRP (in the range of 2–4 mg/L) is recognized as an independent predictor of cardiovascular events. In addition to CRP, levels of fibrinogen, interleukin-1β, and tumor necrosis factor-α are elevated. The effect is not limited to cytokines alone: periodontal epithelium produces matrix metalloproteinases, which may contribute to the destabilization of atherosclerotic plaque.

Bacteremia and direct invasion of the vascular wall. During chewing, tooth brushing, or even spontaneously, periodontal pathogens enter the bloodstream. In patients with aggressive periodontitis, the frequency of transient bacteremia can reach 60–80% after tooth brushing. The most studied microorganism is Porphyromonas gingivalis. This Gram-negative anaerobe possesses unique virulence factors (fimbriae, Arg- and Lys-gingipain proteases). P. gingivalis has been detected in atherosclerotic plaques of coronary, carotid, and femoral arteries at autopsy. Moreover, in animal experiments, intravenous administration of P. gingivalis accelerates the development of atherosclerosis, and its vaccination reduces lesion size.

Cross-immune response (molecular mimicry). Antigens of periodontal pathogens, especially heat shock proteins (Hsp60 of P. gingivalis), share homology with human Hsp60 expressed on damaged endothelium. Antibodies generated against bacterial Hsp60 can cross-react with endothelial Hsp60, causing immune-mediated damage. It has also been found that P. gingivalis can trigger the production of autoantibodies to oxidized low-density lipoproteins (LDL), contributing to the formation of foam cells.

Clinical consequences for patients. In patients who have had a myocardial infarction, the presence of periodontitis is associated with an increased risk of recurrent events and death. Prospective cohorts have shown that untreated periodontitis worsens the prognosis after percutaneous coronary interventions. On the other hand, effective periodontal treatment (combined with hygiene and smoking cessation) along with a decrease in CRP leads to a reduction in the carotid intima-media thickness, a surrogate marker of atherosclerosis regression.

Limitations and prospects. Despite convincing evidence of the association, unresolved issues remain: what depth and duration of periodontal inflammation are critical for triggering vascular effects; whether there is a dose-response effect between the extent of dental treatment and the reduction of cardiovascular risk; and what is the time frame within which periodontitis therapy leads to a clinically significant improvement in cardiac prognosis. Large randomized clinical trials with hard endpoints (infarction, stroke, mortality) are required.

Conclusion

Periodontitis is an independent risk factor for the development of coronary heart disease, myocardial infarction, and stroke. The risk of cardiovascular events in patients with periodontitis is increased by an average of 20–35% compared to individuals with healthy periodontium. The pathogenetic link between periodontitis and cardiovascular diseases occurs through several mechanisms: chronic systemic inflammation (elevated CRP, IL-6), bacteremia with direct invasion of periodontal pathogens into the vascular wall, cross-autoimmune response, and endothelial dysfunction. Active treatment of periodontitis reduces the levels of systemic inflammatory markers and improves endothelial function, which may potentially reduce cardiovascular risk. It is recommended to include dental examination in protocols for assessing cardiovascular risk, as well as to inform cardiologists about the presence of periodontitis in their

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patients. Interdisciplinary interaction between dentists and cardiologists should become the standard of care for patients with comorbid pathology.

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