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CORRELATION BETWEEN CARDIOVASCULAR DISEASES AND PERIODONTITIS

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Abstract. Recent advances in biomedical research have established a substantial association between periodontitis and cardiovascular diseases (CVDs) [1,2]. Periodontitis, recognized as a chronic inflammatory condition affecting the supporting tissues of the teeth, has been increasingly implicated in the development of systemic inflammatory responses that adversely influence cardiovascular health [3,4].

Accumulating clinical evidence suggests that individuals with severe periodontal disease frequently exhibit significantly elevated serum levels of C-reactive protein (CRP), often reaching two- to three-fold higher concentrations compared with healthy subjects [5,6]. Elevated CRP has been widely acknowledged as an independent predictor of atherosclerotic progression and adverse coronary events [4].

Moreover, inflammatory mediators and bacterial endotoxins originating from periodontal lesions may disseminate through systemic circulation, thereby contributing to endothelial dysfunction and promoting atherogenesis [7,8]. Periodontal pathogens have also been associated with transient bacteremia, platelet activation, and thrombotic processes, further supporting the biological relationship between oral and cardiovascular diseases [12].

Keywords. Periodontitis, cardiovascular diseases, inflammation, atherosclerosis, endothelial dysfunction, periodontal infection, C-reactive protein, systemic inflammation.

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Introduction. Cardiovascular diseases continue to represent the leading cause of morbidity and mortality worldwide [1,2,3]. According to reports published by the World Health Organization, cardiovascular disorders account for approximately one-third of global deaths annually [4].

Simultaneously, periodontitis remains one of the most prevalent chronic inflammatory diseases affecting adults. Beyond its destructive effects on periodontal tissues and tooth retention, contemporary evidence indicates that periodontal disease may exert profound systemic consequences, particularly involving the cardiovascular system [5,6,7].

Large-scale epidemiological studies and meta-analyses have demonstrated that individuals suffering from advanced periodontitis possess a significantly increased risk of coronary artery disease, myocardial infarction, and cerebrovascular complications compared with periodontally healthy populations [8,9,10,11].

Consequently, elucidating the mechanistic and clinical relationship between periodontal inflammation and cardiovascular pathology has emerged as an important interdisciplinary focus in modern medical and dental research.

Bacterial factors and systemic inflammation in periodontitis. The pathogenesis of periodontitis is predominantly associated with anaerobic Gram-negative microorganisms, including *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythia*, and *Treponema denticola* [12,13,14].

These microorganisms produce a variety of virulence factors, such as lipopolysaccharides, proteolytic enzymes, and gingipains, which contribute to connective tissue destruction and amplification of inflammatory reactions. Activation of macrophages and monocytes

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subsequently stimulates the release of proinflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) [15,16].

Among these mediators, IL-6 plays a critical role in hepatic CRP synthesis, thereby intensifying systemic inflammatory burden and contributing to vascular injury [17,18].

Endothelial dysfunction and atherosclerotic mechanisms. Endothelial integrity is essential for maintaining vascular homeostasis, regulating vasodilation, and preventing thrombogenesis. Chronic periodontal inflammation has been shown to reduce nitric oxide (NO) bioavailability, consequently impairing endothelial-dependent vasodilation and promoting endothelial dysfunction [19,20].

Furthermore, periodontal inflammation enhances the expression of endothelial adhesion molecules, including VCAM-1 and ICAM-1, facilitating leukocyte adhesion and migration into vascular tissues [5,12]. Such alterations play a pivotal role in the initiation and progression of atherosclerotic plaque formation.

Importantly, molecular investigations have identified *Porphyromonas gingivalis* DNA within atherosclerotic lesions, strongly suggesting a direct contribution of periodontal pathogens to vascular pathology [21,22,23].

Periodontitis and hemostatic alterations. Patients diagnosed with periodontitis frequently demonstrate abnormalities in hemostatic regulation, including enhanced platelet aggregation and activation of coagulation pathways [7,10.24].

Elevated plasma fibrinogen concentrations observed in periodontal disease contribute to increased blood viscosity and heightened thrombotic susceptibility [11.25]. Clinical investigations further indicate that severe periodontitis may significantly elevate the risk of myocardial infarction in comparison with individuals without periodontal inflammation.

Immunological mechanisms. The molecular mimicry hypothesis proposes that certain bacterial antigens derived from periodontal pathogens may structurally resemble host vascular proteins. As a consequence, immune responses directed against bacterial components may inadvertently target endothelial tissues, thereby intensifying autoimmune-mediated vascular inflammation.

Additionally, activated T-lymphocytes contribute to persistent inflammatory activity within atherosclerotic plaques, increasing plaque instability and the likelihood of acute cardiovascular events.

Systemic effects of periodontal therapy. Periodontal treatment, particularly professional debridement combined with anti-inflammatory therapy, has been associated with a significant reduction in systemic inflammatory markers, including CRP, within several months following intervention.

Moreover, multiple clinical studies have demonstrated measurable improvements in endothelial function after periodontal therapy, commonly assessed through enhanced flow-mediated dilation (FMD).

These findings collectively support the concept that effective periodontal management may contribute not only to oral health improvement but also to cardiovascular risk reduction.

Conclusion. Current scientific evidence strongly supports the concept that periodontitis should be regarded as an independent and potentially modifiable risk factor for cardiovascular disease.

Chronic periodontal inflammation contributes to systemic inflammatory activation, endothelial dysfunction, atherosclerotic progression, and thrombotic complications. Importantly, the observed reduction in inflammatory biomarkers and improvement in vascular function following periodontal therapy further emphasize the clinical significance of this association.

Therefore, early diagnosis, preventive strategies, and comprehensive management of periodontal disease may play a substantial role in reducing cardiovascular morbidity and improving overall systemic health outcomes.

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