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NL Journal of Dentistry and Oral Sciences

(ISSN: 3049-1053)

Volume 3 Issue 2 April 2026

Editorial

Introspecting the Correlation between Periodontal Disease and Atherosclerotic Cardiovascular Disease through the Lens of the American Heart Association

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doi: 10.71168/NDO.03.02.145

Received Date: May 22- 2025 **Publication Date:** June 01- 2026

Atherosclerotic Cardiovascular Disease (ASCVD) is found as the common culprit of worldwide deaths, accounting for 30% of all deaths. Atherosclerotic cardiovascular disease (ASCVD) includes long-term health issues caused by the accumulation of plaque within the walls of blood vessels. This category encompasses ischemic heart disease, cerebrovascular disease, and peripheral vascular disease, all of which may lead to sudden clinical occurrences such as acute coronary artery disease (CAD), myocardial infarction (MI), and stroke. Periodontal Disease is a chronic multifactorial inflammatory disease which starts with accumulation of biofilm and plaque around periodontal tissues resulting in bleeding of gums, clinical attachment loss, deepening of pockets and destruction of periodontium. Chronic periodontal infections can lead to bacteremia, which may heighten the inflammatory load that promotes atherogenesis. The inflammation resulting from the direct actions of the oral microbiome may influence systemic inflammation within the walls of blood vessels through two mechanisms: the direct invasion of bacteria from the diseased and inflamed periodontal tissues into the bloodstream, and the translocation of bacteria mediated by phagocytes. The oral microbiome was indirectly associated with chronic systemic inflammation, antibody cross reactivity, and thrombotic factors. Individuals exhibiting elevated levels of the inflammatory marker CRP (C-reactive protein) may face an increased risk of cardiovascular incidents, including nonfatal myocardial infarction (MI) and mortality due to coronary heart disease. Additionally, proinflammatory cytokines like IL-6 (interleukin-6), IL-18 (interleukin-18), soluble C40 ligand, and TNF (tumor necrosis factor) have been linked to coronary heart disease. Patients suffering from periodontal disease exhibit elevated circulating concentrations of leptin (Proinflammatory), CRP, TNF, IL-1 (interleukin-1), IL-6, and IL-8 (interleukin-8),12,13,22 alongside reduced levels of adiponectin (Anti-inflammatory). Periodontitis also raises the Platelet activation factor (PAF) which plays an important role in Platelet aggregation and onset of atherosclerosis. Autoantibodies that cross-react with bacterial antigens, especially those targeting heat shock proteins produced under stress, are proposed as a potential mechanism through which periodontal infections could contribute to atherosclerosis. This mechanism is thought to involve endothelial damage resulting from an immune response to bacterial heat shock proteins. The cross-reactivity observed between Porphyromonas gingivalis and human HSP60 (heat shock protein 60) can lead to atherosclerotic changes due to the ensuing autoimmune response within the vascular endothelium. Bacterial indicators of oral dysbiosis have been linked to a heightened risk of subclinical atherosclerosis, existing and future coronary artery disease (CAD), as well as both incident and recurrent strokes. Pathogens associated with periodontal disease, including the gram-negative bacillus P. gingivalis, may play a role in inflammatory diseases. The identification of periodontal microorganisms (via DNA, RNA, or antigens) in samples of atheromatous plaque and vascular walls could facilitate the advancement of atherosclerosis and trigger a procoagulant response. Genetic studies have identified common susceptibility genes for ASCVD and periodontal disease, particularly the 9p21.3 locus on chromosome 9, which is consistently linked to cardiovascular disease and also associated with periodontal disease, suggesting shared risk factors for both conditions. Coronary artery disease (CAD) associated with multi risk factors like dislipidemia, hypertension, type 2 diabetes, disordered sleep, smoking, obesity, minimal physical activity etc. Chronic inflammatory conditions like low to high grade periodontitis plays an additive risk factor along with them as patients suffering from periodontal problems were proved to be more prone to cardiovascular or cerebrovascular diseases.

Existing research may overlook upstream causal factors. To clarify this, well-structured longitudinal studies and randomized controlled trials (RCTs) are needed to examine periodontal disease in at-risk groups, focusing on how periodontal treatment affects systemic inflammatory markers and outcomes related to atherosclerotic cardiovascular disease (ASCVD).

Citation: Dr. Nanda Kishore Ghoshal. "Introspecting the Correlation between Periodontal Disease and Atherosclerotic Cardiovascular Disease through the Lens of the American Heart Association". NL Journal of Dentistry and Oral Sciences 3.2 (2026): 01-02.