Journal of
Medical and Life Sciences
AL-NAJAM An International, Peer-reviewed, Quarterly publishing, Open access, Multi-disciplinary Research Journal

EDITORIAL OPEN ACCESS

Periodontal-Cardiometabolic Axis: How Oral Biology Shapes Critical Care Outcomes

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Citation: Yousuf A, Ali F. Periodontal-Cardiometabolic Axis: How Oral Biology Shapes Critical Care Outcomes. Al Najam J Med Life Sci. 2026;1(1):1–2.

Received: 22 February, 2026 **Revised:** 03 March, 2026 **Accepted:** 08 March, 2026

Periodontal disease is among the most prevalent chronic inflammatory diseases globally, that is characterized by unremitting dysbiosis of microbes and destruction of tooth supportive tissue [1]. Periodontitis is traditionally thought of as a localized oral disease but today is being perceived as a systemic inflammatory disease with extensive health consequences. The damaged periodontal pocket epithelium offers a direct pathway of entry of oral bacteria and their toxic by-products into the bloodstream leading to recurrent occurrences of bacteremia and systemic immune response [2]. This unending microbial challenge is central in influencing the so called periodontal-cardiometabolic axis (Figure 1).

Chronic periodontal inflammation favors the release of systemic inflammatory mediators such as interleukin-6, tumor necrosis factor-alpha and C-reactive protein that are highly implicated in endothelial dysfunction and atherogenesis [3]. These circulating mediators destroy vascular homeostasis, diminish nitric oxide levels of bioavailability and increase oxidative stress to form a biological environment where cardiovascular disease is favourable. Most recent molecular and epidemiological findings indicated that moderate to severe periodontitis patients have a greater number of systemic inflammatory indicators and are at a higher risk of coronary artery disease and ischemic stroke [4]. Oral microbial by-products have direct effects on cardiometabolic pathways in addition to having effects on inflammatory signaling. The major periodontal pathogens like Porphyromonas gingivalis and Fusobacterium nucleatum have the capability of spreading to the bloodstream and engaging with the endothelial cells in the vascular beds, macrophages and platelets [5]. Gram-negative oral bacteria lipopolysaccharides initiate Toll-like receptor signalling and nuclear factor-kB signalling and stimulate chronic low-grade inflammation and rapid atherosclerotic plaque [6]. The mechanistic proof that these biological mechanisms have in support of the part of oral microbiology in cardiovascular pathology is substantiated.

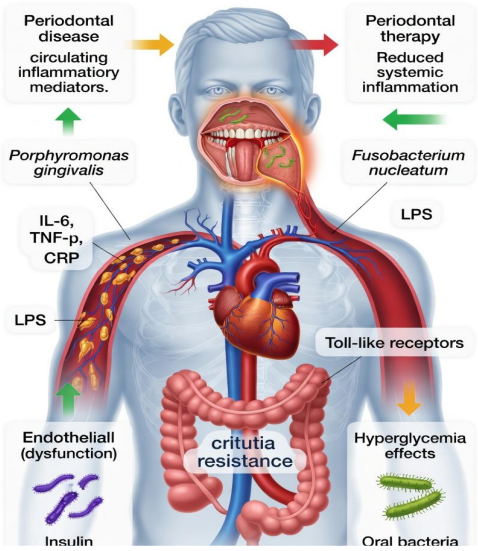

Figure 1. The periodontal-cardiometabolic axis linking oral *inflammation to systemic inflammation*, cardiometabolic disease.

The oral microbiome also interacts with the systemic metabolism in the oral-gut microbiota axis. New findings have shown that the gastrointestinal tract can be colonized by oral bacteria and restructure intestinal microbial colonies, which alters the generation of short-chain fatty acids structuring the bile acid metabolism and inflammatory metabolites [7]. Such microbial crosstalk has been attributed to insulin resistance, dyslipidemia and metabolic syndrome, and thus, supports the correlation between periodontal disease and cardiometabolic dysfunction [8]. A typical and two-way relationship is the case of diabetes mellitus and periodontal disease. Hyperglycemia affects the neutrophil activity, collagen metabolism and vascular integrity, making them the predisposing factors of periodontal disease and tissue erosion [9]. Periodontal inflammation on the other hand enhances insulin resistance by maintaining systemic inflammatory load and disrupting the insulin signaling pathways [10]. According to

recent clinical data, a person with inadequately managed type 2 diabetes and active periodontitis is at higher risk of cardiovascular events and complications significantly than a metabolically healthy person [11].

These are the biological interactions that are especially pertinent to the cases of critical illness. Inflammation on a systemic scale, loss of control over the immune system and metabolic imbalance are significant prognostic variables in intensive care units. Periodontal disease has the potential to add to the cumulative inflammatory burden and could enhance inflammatory responses in sepsis, acute cardiovascular events and metabolic decompensation [12]. The similar levels of C-reactive protein and pro-inflammatory cytokines found in severe periodontitis and severely ill patients indicate similar inflammatory pathways that may exacerbate organ dysfunction and slow down the recovery process [13]. The translocation of oral microbial organisms can also impact the intensive care outcomes through making the patients vulnerable to secondary infections and maladaptive immune activities. The poor oral hygiene and decreased salivary flow in critically unstable patients encourages the growth of oral microbes, which can act as a source of respiratory and systemic pathogens [14]. Despite the limited number of direct interventional trials, recent narrative and clinical reviews indicate that poor oral health correlates with longer hospitalization, increased risk of infection and adverse recovery trajectories on vulnerable hospitalized populations [15].

Understanding of the periodontal-cardiometabolic axis has led to the focus of the significance of combined medical and dental treatment. The beneficial effects of periodontal therapy on endothelial function, as well as the decrease of systemic inflammatory markers, have demonstrated that oral inflammation management could potentially help to reduce cardiometabolic risks on a broader level [16]. Integrating oral health assessment into cardiometabolic and critical care interventions could thus be a significant and changeable practice to enhance patient outcome. In conclusion, cardiometabolic diseases are serious biological risks of periodontal disease through the process of chronic inflammation, microbial dysbiosis, and immune-metabolic interactions The existing literature shows that oral biology does have a significant role in the formation of cardiovascular risk, the control of diabetes, and possibly the results in intensive care. The introduction of periodontal care into multidisciplinary cardiometabolic and critical care systems has the potential to decrease systemic complications and aid in the long-term recovery of high-risk groups of patients.

Acknowledgement: None
Conflict of Interest: None
Grant Support & Funding Source: None
Authors' Contribution: Both authors contributed equally as per ICMJE.

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