E CONF SERIES

International Conference on Medical Science, Medicine and Public Health
Hosted online from Jakarta, Indonesia
Website: econfseries.com 30th June, 2025

# IMMUNOLOGICAL AND MICROBIOLOGICAL CORRELATES OF PERIODONTAL DISEASE IN TYPE 2 DIABETES PATIENTS: A CLINICAL INSIGHT

Fozilova Umida Kamalovna
Bukhara State Medical Institute
Fozilova.umida@bsmi.uz
https://orcid.org/0009-0008-8405-7800

Khabibova Nazira Nasulloevna
Bukhara State Medical Institute Department of Therapeutic Dentistry
xabibova.nazira@bsmi.uz
https://orcid.org/0000-0001-9908-7078

**Abstract:**
Type 2 diabetes mellitus (T2DM) is associated with various systemic complications, including an increased risk of periodontal disease. This article investigates the immunological and microbiological mechanisms underlying periodontal deterioration in patients with T2DM. The analysis highlights the impact of chronic hyperglycemia on host immune response, microbial dysbiosis, and periodontal tissue destruction. Additionally, it examines the clinical and laboratory correlations that may inform preventive and therapeutic strategies.

**Keywords:** Type 2 diabetes, periodontitis, immune dysfunction, microbial dysbiosis, cytokines, glycemic control

## Introduction:

Type 2 diabetes mellitus (T2DM) is a globally prevalent metabolic disorder characterized by insulin resistance and chronic hyperglycemia. It is associated with a range of systemic and local complications, among which periodontal disease stands out due to its bidirectional relationship with glycemic control. Periodontitis is an inflammatory disease of the supporting structures of the teeth, and its severity

is markedly increased in individuals with diabetes. The pathophysiology of this interrelation involves a complex interplay between metabolic dysregulation, immune dysfunction, and microbial imbalance.

Immunological Dysregulation in T2DM and Periodontal Disease: The immune response in T2DM patients is notably altered due to the prolonged hyperglycemic environment. Neutrophils, which are primary responders in periodontal defense, exhibit impaired chemotaxis, phagocytosis, and reactive oxygen species (ROS) generation. Moreover, monocytes and macrophages are hyper-responsive to bacterial lipopolysaccharide (LPS), leading to exaggerated secretion of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-\u03b1), interleukin-1 beta (IL-1\u03b2), and interleukin-6 (IL-6). These cytokines contribute to persistent inflammation and destruction of periodontal tissues.

In T2DM patients, an imbalance in the regulatory and pro-inflammatory immune cells is commonly observed. An increased ratio of T-helper 17 (Th17) cells to regulatory T (Treg) cells skews the immune response towards chronic inflammation. Similarly, the predominance of classically activated (M1) macrophages over alternatively activated (M2) macrophages enhances tissue breakdown.

Microbial Dysbiosis and Diabetes: The subgingival microbiota in diabetic patients differs significantly from non-diabetic individuals. Hyperglycemia modifies the local environment, favoring the growth of pathogenic anaerobic bacteria such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola. The increased glucose concentration in gingival crevicular fluid and saliva serves as a nutrient source for these bacteria, promoting their virulence.

Studies have shown a higher bacterial load and altered microbial diversity in the periodontal pockets of diabetic patients. These dysbiotic communities interact with the dysregulated immune system to perpetuate chronic inflammation, leading to alveolar bone loss and clinical attachment loss.

Clinical and Laboratory Correlations: Clinical indicators such as bleeding on probing, pocket depth, and clinical attachment level are more severe in T2DM patients compared to non-diabetic controls. Biochemical markers in saliva and gingival crevicular fluid, including elevated levels of TNF-\u03b1, IL-6, and matrix

metalloproteinases (MMPs), corroborate the presence of active periodontal inflammation.

Another key marker is the reduced salivary concentration of lysozyme and lactoferrin, both of which are critical for innate immune defense. Their depletion in diabetic individuals signifies compromised antimicrobial protection. Concurrently, HbA1c levels correlate positively with periodontal disease severity, underlining the systemic-metabolic contribution to oral inflammation.

Therapeutic Implications and Preventive Strategies: The management of periodontal disease in T2DM requires an integrative approach. Mechanical debridement remains the cornerstone, but its efficacy is enhanced by adjunctive therapies targeting immune modulation and microbial control. Non-surgical periodontal therapy has been shown to reduce HbA1c by approximately 0.3-0.4% in several studies.

The use of antimicrobial agents, probiotics, and host modulation therapies, including subantimicrobial-dose doxycycline, can provide additional benefits. Moreover, enhancing glycemic control through medical management contributes to periodontal healing and reduces inflammation.

Conclusion: The interplay between immunological disturbances and microbial dysbiosis underlies the increased susceptibility and severity of periodontal disease in T2DM patients. Understanding these mechanisms enables more precise diagnostic and therapeutic approaches. Future research should aim to develop personalized treatment regimens that consider the unique immunometabolic profiles of diabetic patients.

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