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Bidirectional Impact of Type 2 Diabetes Mellitus and Chronic Periodontitis: A Comparative Study on Inflammatory Biomarkers and Glycemic Control Post-Non-Surgical Periodontal Therapy

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ABSTRACT

Background: Periodontitis and Type 2 Diabetes Mellitus (T2DM) are chronic inflammatory diseases that exhibit a bidirectional relationship, with each condition exacerbating the other. This study evaluates the effect of non-surgical periodontal therapy (NSPT) on systemic inflammation and glycemic control in patients with T2DM and chronic periodontitis.

Objective: To compare the changes in inflammatory biomarkers (IL-6, TNF-α, and CRP) and glycemic control (HbA1c) in T2DM patients and non-diabetic controls after NSPT.

Methods: A 3-month prospective cohort study was conducted on 60 patients divided into two groups: Group A (n=30) with T2DM and chronic periodontitis, and Group B (n=30) with chronic periodontitis only. Clinical periodontal parameters (PPD, CAL, BOP), serum inflammatory biomarkers, and HbA1c levels were measured at baseline and 12 weeks after NSPT.

Results: Both groups showed significant improvements in periodontal parameters ($p < 0.05$). Group A exhibited a statistically significant reduction in HbA1c (from 8.1% to 7.4%, $p < 0.01$), and inflammatory biomarkers decreased markedly in both groups. The magnitude of reduction in IL-6 and CRP was more pronounced in Group A.

Conclusion: NSPT improves both periodontal health and systemic inflammation. In T2DM patients, these improvements are accompanied by enhanced glycemic control, underscoring the importance of periodontal care in systemic disease management.

Keywords: *Periodontitis, Type 2 Diabetes Mellitus, Inflammatory Biomarkers, Non-surgical Periodontal Therapy, Glycemic Control, IL-6, CRP, HbA1c*

1. INTRODUCTION

Chronic diseases, particularly those involving inflammatory and metabolic dysfunctions, present an increasing global health burden. Among these, Type 2 Diabetes Mellitus (T2DM) and chronic periodontitis are two highly prevalent conditions that have been shown to exert a bidirectional influence on each other's development, progression, and management outcomes [1-3]. T2DM, characterized by insulin resistance and persistent hyperglycemia, affects over 537 million adults globally as of the 2021 IDF report, with projections indicating a sharp rise in the coming decades[4-6]. Meanwhile, chronic periodontitis, an infectious-inflammatory disease of the supporting structures of the teeth, affects approximately 50% of the adult population worldwide, contributing significantly to tooth loss and impaired oral function [7-9].

Mounting clinical and experimental evidence indicates that the relationship between T2DM and periodontitis is not merely associative but causal and reciprocal. Individuals with uncontrolled T2DM have an increased susceptibility to periodontal disease, marked by exaggerated inflammatory responses, delayed wound healing, and impaired neutrophil function [10-12]. In contrast, the systemic inflammatory burden caused by chronic periodontitis can worsen insulin resistance and contribute to poor glycemic control [13-18]. This bidirectional relationship is now considered a critical model in oral-systemic health, prompting collaborative approaches in both medical and dental care.

From a pathophysiological standpoint, several mechanisms underpin this interplay. Chronic hyperglycemia in diabetes facilitates the accumulation of advanced glycation end-products (AGEs), which engage with their receptor (RAGE) on various cells, leading to the overproduction of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) [19-21]. These mediators are not only elevated in periodontal tissues but also systemically, contributing to increased insulin resistance and endothelial dysfunction [15, 22-23]. Periodontal infection, in turn, exacerbates the host immune response, facilitating systemic inflammation and metabolic dysregulation—a process consistent with the inflammatory cytokine hypothesis [24-26].

Clinically, these insights have profound implications. Several interventional studies suggest that non-surgical periodontal therapy (NSPT)—comprising scaling and root planing (SRP)—may lead to improvements in both periodontal health and metabolic control, particularly evidenced by reductions in HbA1c levels, a key biomarker of glycemic status [27-29]. However, outcomes have been variable, and further research is needed to delineate the underlying immunoinflammatory changes that mediate these improvements and to evaluate their consistency across populations.

This study aims to expand on this growing area of evidence by conducting a comparative clinical investigation into the effects of NSPT on systemic inflammatory biomarkers (IL-6, TNF-α, CRP) and glycemic control (HbA1c) in patients with T2DM and chronic periodontitis, in comparison with systemically healthy individuals with periodontitis. The goal is to better understand the biological underpinnings of the periodontal-diabetes axis and to strengthen the case for integrated disease management protocols in dental and diabetic care settings.

2. MATERIALS AND METHODS

Study Design and Population

This was a prospective cohort study conducted over 3 months at a university-based dental hospital. Ethical approval was obtained from the Institutional Review Board (IRB#2025-PD-034).

Inclusion Criteria:

Aged 35–65 years

Diagnosed with chronic periodontitis (≥4 sites with PPD ≥5 mm)

T2DM group: HbA1c ≥7% and ≤10% under medical supervision

Exclusion Criteria:

Smoking

Pregnancy

Antibiotic therapy in the last 3 months

Periodontal therapy in the past 6 months

Study Groups

- **Group A**: 30 patients with T2DM and chronic periodontitis
- **Group B**: 30 patients with chronic periodontitis but no systemic disease

Clinical Measurements

At baseline and 12 weeks post-NSPT:

- **Periodontal Parameters**: Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL), Bleeding on Probing (BOP)
- **Biochemical Parameters**: HbA1c, IL-6, CRP, and TNF-α (measured by ELISA)

Intervention

All patients received non-surgical periodontal therapy, including scaling and root planing (SRP), oral hygiene education, and chlorhexidine mouth rinse for 2 weeks.

Statistical Analysis

Data were analyzed using SPSS v25.0. Paired t-tests and ANOVA were used for intra- and inter-group comparisons. Significance was set at $p < 0.05$.

3. RESULTS

Baseline Characteristics

Both groups were age- and gender-matched. The mean HbA1c in Group A was 8.1% at baseline.

Periodontal Improvements

Significant reductions in PPD and CAL were observed in both groups ($p < 0.01$). BOP decreased by over 40% in both groups.

Inflammatory Biomarker Changes

Post-treatment reductions:

- **IL-6**: Group A ↓ 28%, Group B ↓ 19%
- **CRP**: Group A ↓ 36%, Group B ↓ 22%
- **TNF-α**: Moderate but significant reduction in both groups ($p < 0.05$)

Glycemic Control (Group A only)

- HbA1c reduced from 8.1% ± 0.6 to 7.4% ± 0.5 ($p < 0.01$)

4. DISCUSSION

The findings of this study add meaningful insight to the growing body of literature addressing the complex bidirectional relationship between Type 2 Diabetes Mellitus (T2DM) and chronic periodontitis. Our data demonstrate that non-surgical periodontal therapy (NSPT) not only improves local periodontal clinical parameters such as probing pocket depth (PPD), clinical attachment loss (CAL), and bleeding on probing (BOP), but also significantly reduces systemic inflammatory burden—as evidenced by decreases in IL-6, TNF-α, and CRP. More notably, in patients with T2DM, these improvements are paralleled by a statistically and clinically significant reduction in HbA1c levels, highlighting the potential role of periodontal therapy as an adjunctive measure in metabolic disease management.

These results strongly support previous findings by Patricia A A O'Connell et al. [30] and S Gopalakrishnan Sundaram et al., where periodontal therapy led to reductions in systemic inflammatory markers and modest yet consistent improvements in glycemic control [31-32]. The observed HbA1c reduction of approximately 0.7% in the diabetic group of this study is clinically relevant, comparable to the effect of some oral hypoglycemic medications (e.g., DPP-4 inhibitors), thus emphasizing the systemic therapeutic benefits of periodontal care [33]. Moreover, this reduction meets the threshold established by the UK Prospective Diabetes Study (UKPDS), which showed that each 1% drop in HbA1c is associated with significant reductions in diabetic complications [34].

The biological plausibility of these outcomes lies in the inflammatory and immunomodulatory pathways shared by both diseases. Chronic hyperglycemia promotes the formation of advanced glycation end-products (AGEs), which, upon binding to RAGE receptors, trigger oxidative stress and upregulate pro-inflammatory cytokines that are also key mediators in periodontal tissue destruction. Similarly, periodontitis allows periodontal pathogens and inflammatory mediators to enter systemic circulation, further amplifying the cytokine cascade and worsening insulin resistance—a process often described as a feed-forward inflammatory loop [6-10].

An important strength of our study is the use of specific biomarker profiling (IL-6, CRP, TNF-α) to track systemic inflammation, thereby providing biochemical evidence to support clinical outcomes. Furthermore, by comparing diabetic and non-diabetic periodontitis patients, we highlight not only the differential inflammatory response in systemic disease but also the consistency of periodontal therapy outcomes across systemic health states [9-13].

Nevertheless, several limitations must be acknowledged. The follow-up period of 12 weeks, while sufficient for observing initial changes, does not account for long-term sustainability of improvements in glycemic control. Additionally, although ELISA-based assays provide high sensitivity, single-point serum biomarker assessments may not fully capture temporal variations or chronic inflammatory load. Finally, factors such as dietary habits, medication adherence, and microbiome diversity, which were not controlled in this study, may influence inflammatory and glycemic outcomes [7-12].

Despite these limitations, the study reinforces the view that periodontal health is not isolated from systemic physiology but is intrinsically linked to broader inflammatory and metabolic networks. These findings hold strong implications for interdisciplinary healthcare, urging for greater collaboration between dental and medical practitioners in the management of patients with chronic inflammatory diseases.

5. CONCLUSION

This study confirms that non-surgical periodontal therapy leads to significant improvements in both local periodontal health and systemic inflammatory status, and in the case of patients with T2DM, yields a measurable reduction in HbA1c levels. These results underscore the interconnected pathophysiology of periodontitis and diabetes mellitus and advocate for the inclusion of periodontal assessment and therapy as part of standard diabetic care protocols.

From a public health and clinical practice perspective, this research reinforces the need to reframe oral health as an integral component of systemic health. Future studies with longer follow-up periods, larger sample sizes, and microbiome-based profiling may further elucidate the biological mechanisms underpinning the observed associations.

In conclusion, managing periodontitis in diabetic patients is not only critical for preserving oral function but also represents a non-pharmacologic, cost-effective strategy to reduce systemic inflammation and improve glycemic outcomes—a compelling case for integrating dental care into holistic chronic disease management models.

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