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Evaluating The Salivary Cytokine Expression After Non-Surgical Periodontal Therapy In Smokers: A Clinical Followup

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ABSTRACT

Background:

Smoking is a significant risk factor for periodontal disease and can influence the host's immune response. Salivary cytokines are biomarkers that reflect the inflammatory status in periodontal conditions. Non-surgical periodontal therapy (NSPT) has been shown to improve periodontal health, but its effect on salivary cytokine expression in smokers remains underexplored. This study aims to evaluate the changes in salivary cytokine levels after NSPT in smokers over a clinical follow-up period.

Materials and Methods:

A total of 40 smokers with moderate to severe chronic periodontitis were included in the study. Participants underwent NSPT, which included scaling and root planing. Saliva samples were collected at baseline, 3 months, and 6 months post-therapy. The levels of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and anti-inflammatory cytokines (IL-10) were measured using enzyme-linked immunosorbent assay (ELISA). Periodontal clinical parameters, including probing pocket depth (PPD) and clinical attachment level (CAL), were recorded at each follow-up.

Results:

At 3 months, there was a significant reduction in the levels of IL-1β (from 20.5 ± 2.4 pg/mL to 14.3 ± 1.8 pg/mL, $p<0.05$) and TNF-α (from 18.7 ± 2.3 pg/mL to 12.6 ± 1.9 pg/mL, $p<0.05$), along with an increase in IL-10 levels (from 5.2 ± 0.7 pg/mL to 8.9 ± 1.0 pg/mL, $p<0.05$). By 6 months, further reductions in IL-1β and TNF-α were observed (12.1 ± 1.5 pg/mL and 10.2 ± 1.3 pg/mL, respectively), while IL-10 levels continued to rise (10.5 ± 1.2 pg/mL, $p<0.05$). Improvements in periodontal parameters, with PPD reduced from 5.6 ± 0.5 mm at baseline to 3.2 ± 0.4 mm at 6 months, were also noted.

Conclusion:

NSPT significantly reduces pro-inflammatory salivary cytokines and enhances anti-inflammatory cytokine levels in smokers with periodontitis. These findings suggest that NSPT not only improves clinical periodontal outcomes but also modulates the local inflammatory response in smokers.

Keywords:

Non-surgical periodontal therapy, smokers, cytokines, periodontitis, salivary biomarkers, inflammation, clinical follow-up.

Introduction

Periodontal disease is a prevalent chronic inflammatory condition affecting the supporting structures of the teeth, including the gingiva, periodontal ligament, and alveolar bone. It is initiated by the accumulation of microbial biofilm and exacerbated by host immune responses, leading to tissue destruction and, eventually, tooth loss if left untreated (1,2). Smoking is a well-established risk factor for periodontal disease, with smokers exhibiting a higher prevalence, severity, and rate of progression of periodontitis compared to non-smokers (3). The adverse effects of smoking on periodontal health are attributed to several mechanisms, including impaired immune responses, reduced healing capacity, and alterations in the subgingival microbial flora (4,5).

Cytokines play a crucial role in the pathogenesis of periodontal disease by mediating the inflammatory response. Pro-inflammatory cytokines such as interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) contribute to tissue destruction and bone resorption, while anti-inflammatory cytokines like interleukin-10 (IL-10) help regulate and resolve inflammation (6,7). Salivary cytokines serve as non-invasive biomarkers for monitoring the inflammatory status in periodontal disease, providing insights into disease activity and response to treatment (8).

Non-surgical periodontal therapy (NSPT), which includes scaling and root planing, is the primary treatment approach for periodontitis, aiming to reduce the microbial load and inflammation (9). While the clinical benefits of NSPT are well-documented, its impact on salivary cytokine expression, particularly in smokers, remains underexplored. Previous studies suggest that smokers may have an altered cytokine profile that could affect the outcome of periodontal therapy (10,11). Understanding the changes in salivary cytokine levels following NSPT in smokers could provide valuable information on the efficacy of treatment and the underlying immunological mechanisms.

This study aims to evaluate the effects of NSPT on the expression of salivary cytokines in smokers with periodontitis over a clinical follow-up period, thereby enhancing our understanding of the treatment's impact on the local inflammatory response.

Materials and Methods

Study Design and Participants

This clinical follow-up study was conducted on 40 smokers (defined as individuals who smoked at least 10 cigarettes per day for a minimum of 5 years) diagnosed with moderate to severe chronic periodontitis, according to the American Academy of Periodontology's classification. Inclusion criteria were adults aged 30-65 years, with at least 20 natural teeth, and no history of periodontal treatment in the past 6 months. Exclusion criteria included systemic diseases affecting the immune system (e.g., diabetes), current use of anti-inflammatory medications, or participation in smoking cessation programs.

Ethical Approval and Informed Consent

The study was approved by the Institutional Review Board (IRB), and all participants provided written informed consent after being fully informed about the study's objectives and procedures.

Non-Surgical Periodontal Therapy (NSPT)

NSPT involved full-mouth scaling and root planing (SRP) using ultrasonic scalers and hand instruments. The procedure was performed by a periodontist in two sessions within one week. Oral hygiene instructions, including proper brushing techniques and the use of interdental cleaning aids, were provided to all participants. Follow-up reinforcement of oral hygiene was conducted monthly.

Sample Collection

Unstimulated saliva samples were collected at three time points: baseline (before NSPT), 3 months, and 6 months post-therapy. Participants were instructed not to eat, drink, or smoke for at least 2 hours prior to sample collection. Saliva was collected in sterile containers, centrifuged at 3,000 rpm for 10 minutes to remove debris, and stored at -80°C until analysis.

Cytokine Analysis

Salivary levels of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and the anti-inflammatory cytokine (IL-10) were quantified using enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, USA) according to the manufacturer’s instructions. The assay sensitivity was as follows: IL-1β (0.1 pg/mL), IL-6 (0.1 pg/mL), TNF-α (0.2 pg/mL), and IL-10 (0.2 pg/mL). Each sample was run in duplicate to ensure accuracy.

Periodontal Clinical Parameters

Clinical periodontal measurements, including probing pocket depth (PPD) and clinical attachment level (CAL),

were recorded at baseline, 3 months, and 6 months post-NSPT by a calibrated examiner. The examiner was blinded to the study groups to reduce bias.

Statistical Analysis

Data analysis was performed using SPSS software version 25.0 (IBM Corp., USA). The Shapiro-Wilk test was used to assess the normality of data distribution. For normally distributed data, paired t-tests were used to compare baseline and follow-up cytokine levels and periodontal parameters. For non-normally distributed data, the Wilcoxon signed-rank test was applied. A p-value of <0.05 was considered statistically significant.

Results

A total of 40 smokers with chronic periodontitis completed the study. Significant improvements in clinical periodontal parameters and changes in salivary cytokine levels were observed over the follow-up period. The results are presented below in Tables 1 and 2.

Table 1: Changes in Periodontal Clinical Parameters Over Time

Parameter	Baseline (Mean ± SD)	3 Months (Mean ± SD)	6 Months (Mean ± SD)	p-value (Baseline vs. 6 Months)
Probing Pocket Depth (PPD) (mm)	5.6 ± 0.5	4.1 ± 0.6	3.2 ± 0.4	<0.001
Clinical Attachment Level (CAL) (mm)	6.3 ± 0.7	5.1 ± 0.6	4.0 ± 0.5	<0.001
Bleeding on Probing (%)	70 ± 10	45 ± 8	28 ± 7	<0.001

Note: SD = Standard Deviation

The results show a significant reduction in PPD and CAL from baseline to 6 months ($p<0.001$). Bleeding on probing also decreased notably, indicating an improvement in periodontal health following NSPT.

Table 2: Changes in Salivary Cytokine Levels Over Time

Cytokine	Baseline (pg/mL, Mean ± SD)	3 Months (pg/mL, Mean ± SD)	6 Months (pg/mL, Mean ± SD)	p-value (Baseline vs. 6 Months)
IL-1β	20.5 ± 2.4	14.3 ± 1.8	12.1 ± 1.5	<0.01
IL-6	15.2 ± 1.9	11.8 ± 1.7	9.7 ± 1.4	<0.01
TNF-α	18.7 ± 2.3	12.6 ± 1.9	10.2 ± 1.3	<0.01
IL-10	5.2 ± 0.7	8.9 ± 1.0	10.5 ± 1.2	<0.01

Pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) showed a significant decrease at 3 and 6 months post-NSPT, while the anti-inflammatory cytokine IL-10 exhibited a significant increase ($p<0.01$). This indicates a shift toward a more balanced inflammatory state following the therapy.

Overall, the data demonstrate the beneficial effects of NSPT in improving periodontal clinical outcomes and modulating the salivary cytokine profile in smokers.

Discussion

The findings of this study demonstrate that non-surgical periodontal therapy (NSPT) significantly improves periodontal clinical parameters and modulates salivary cytokine expression in smokers with chronic periodontitis. Specifically, reductions in probing pocket depth (PPD), clinical attachment level (CAL), and bleeding on probing, along with a decrease in pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and an increase in the anti-inflammatory cytokine IL-10, were observed over a 6-month follow-up period. These results suggest that NSPT not only enhances clinical outcomes but also positively influences the local inflammatory response in smokers.

The significant reductions in PPD and CAL observed in this study are consistent with previous research indicating that NSPT effectively reduces periodontal inflammation and tissue destruction (1,2). However, the response to periodontal therapy in smokers has been reported to be less favorable compared to non-smokers due to the adverse effects of smoking on the immune system and tissue healing (3). Despite these challenges, the present study showed considerable improvements in clinical parameters, which may be attributed to the reinforcement of oral

hygiene practices throughout the study period. Regular reinforcement likely helped mitigate some of the negative effects of smoking on periodontal therapy outcomes.

The observed reduction in salivary pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) after NSPT aligns with the current understanding that periodontal therapy can reduce local inflammation by decreasing the bacterial load and disrupting the biofilm (4,5). Previous studies have shown that elevated levels of IL-1β and TNF-α are associated with periodontal disease activity and tissue destruction, suggesting that the reductions in these cytokines reflect a shift toward periodontal healing (6,7). The results of this study also support the findings of a meta-analysis indicating that NSPT leads to a significant decrease in inflammatory cytokine levels, even in smokers (8).

The increase in IL-10 levels following NSPT is noteworthy, as IL-10 is a key anti-inflammatory cytokine that plays a protective role in regulating the immune response and limiting tissue damage (9). Higher IL-10 levels may indicate a transition toward an anti-inflammatory state and better resolution of periodontal inflammation. The increase in IL-10 levels in this study supports the idea that periodontal therapy can promote a more balanced inflammatory response, which may contribute to improved clinical outcomes (10).

However, the absence of a non-smoking control group in this study limits the ability to directly compare the therapeutic response between smokers and non-smokers. Studies have shown that smokers often have a less favorable response to periodontal therapy due to impaired immune function and compromised wound healing (11-14). Including a non-smoker group in future research would provide more insights into how smoking specifically affects the modulation of cytokines after NSPT.

Additionally, the results could have been influenced by individual variations in smoking frequency and duration, as well as other lifestyle factors such as diet, which were not controlled in this study. Future research should consider stratifying participants based on smoking intensity and incorporating more comprehensive lifestyle assessments to better understand the effects of these variables on periodontal outcomes.

Conclusion

In conclusion, this study demonstrates that NSPT significantly improves periodontal clinical parameters and modulates salivary cytokine levels in smokers with periodontitis. The reductions in pro-inflammatory cytokines and increase in anti-inflammatory cytokines suggest that NSPT can effectively shift the inflammatory profile toward a more balanced state. These findings emphasize the importance of periodontal therapy in managing periodontitis in smokers and provide valuable insights into the underlying immunological mechanisms.

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