Original Research Paper

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Differential Expression of Salivary Mucin and Total Protein in Periodontitis Patients Following Nonsurgical Periodontal Therapy

Abstract:

Objective: This study investigated changes in salivary mucin and total protein levels in patients with gingivitis and chronic periodontitis before and three months after nonsurgical periodontal therapy (NSPT).

Methods: Ninety systemically healthy subjects aged 25–55 years were divided into three groups: Group I (periodontally healthy controls), Group II (gingivitis), and Group III (chronic generalized periodontitis). Unstimulated saliva samples were collected at baseline for all groups and at three months post-NSPT for Groups II and III. Salivary mucin was quantified using the Alcian Blue method, and total protein levels were estimated using the biuret method. Clinical parameters including gingival index, probing pocket depth (PPD), clinical attachment level (CAL), and bleeding on probing were recorded. Statistical analysis was performed using ANOVA, paired and unpaired t-tests, and Pearson's correlation coefficient.

Results: At baseline, Group III demonstrated significantly higher clinical parameters and total protein levels compared to Groups II and I (Group III > Group II > Group I). Salivary mucin levels were higher in Group II than in Group III. Following NSPT, both Groups II and III showed significant improvement in clinical parameters. A significant reduction in total protein levels was observed in both groups, while salivary mucin levels showed a modest increase post-therapy. Positive correlations were noted between clinical parameters and salivary biomarkers.

Conclusion: NSPT results in significant modulation of salivary mucin and total protein levels, correlating with clinical periodontal improvement. Salivary biomarkers may serve as noninvasive tools for monitoring periodontal therapy outcomes.

Key-words: Chronic periodontitis; Gingivitis; Nonsurgical periodontal therapy; Salivary mucin; Total protein.

Introduction:

Chronic periodontitis and gingivitis are among the most prevalent inflammatory diseases of the oral cavity, affecting millions of individuals worldwide. These conditions are characterized by inflammation of the periodontal tissues, which, if left untreated, may progress to destruction of the periodontal ligament and alveolar bone, ultimately resulting in tooth loss[1]. The pathogenesis of periodontal diseases involves complex interactions between periodontopathogenic microorganisms and the host immune response, leading to sustained inflammation and tissue breakdown[1].

Recent research has increasingly focused on identifying biological markers that can aid in the diagnosis, monitoring, and evaluation of periodontal disease activity and treatment outcomes. Saliva has emerged as a promising diagnostic medium due to its noninvasive collection, ease of handling, and close reflection of local and systemic health status[2]. The composition of saliva is influenced by the magnitude and nature of the host response to periodontal pathogens, making it a valuable source of biomarkers for periodontal diseases[3].

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Mucins are high-molecular-weight glycoproteins synthesized and secreted by the major and minor salivary glands and constitute an essential component of saliva. They play a critical role in maintaining oral homeostasis by lubricating oral tissues, forming a protective mucosal barrier, and inhibiting microbial adhesion to oral surfaces[4]. Earlier studies classified salivary mucins into two major groups: high-molecular-weight MG1 mucins and low-molecular-weight MG2 mucins[5]. Advances in molecular biology have since identified more than 20 human mucin genes, highlighting the structural and functional diversity of mucins and their importance in oral health and disease[6].

Total salivary protein concentration represents another important indicator of oral inflammatory status. Salivary proteins contribute significantly to host defense mechanisms and exhibit multifunctional properties, including antimicrobial, immunomodulatory, and protective functions[7]. Elevated total protein levels in saliva have been associated with increased vascular permeability and inflammatory exudation, which are characteristic features of periodontal inflammation[3]. Therefore, alterations in total salivary protein levels may reflect disease severity and tissue response in gingivitis and chronic periodontitis.

The combined evaluation of salivary mucin and total protein levels provides a comprehensive approach to assessing periodontal disease status. Saliva contains a wide array of proteins, including albumins, amylases, mucins, lactoferrin, lysozymes, and histatins, making it a rich and informative diagnostic fluid[8,9]. Changes in salivary mucin levels may indicate alterations in mucosal defense mechanisms, while variations in total protein levels may reflect inflammatory burden and tissue breakdown in periodontal diseases.

Although more specific biomarkers are available, their clinical applicability is often limited by cost, availability, and technical complexity. In contrast, salivary mucin and total protein assays are relatively simple, cost-effective, and feasible for routine clinical and research use. Their evaluation may therefore serve as an initial screening approach and provide a foundation for future studies exploring more targeted biomarkers.

The present study was undertaken to compare salivary mucin and total protein levels among periodontally healthy individuals, patients with gingivitis, and patients with chronic periodontitis at baseline, and to assess changes in these biomarkers three months following nonsurgical periodontal therapy. Additionally, the study aimed to correlate salivary biomarker levels with established clinical periodontal parameters.

Materials and Methods:

This clinical study was conducted in the Department of Periodontology with a total sample size of 90 subjects, calculated using G*Power software version 3.1.9.2 (Heinrich-Heine-Universität Düsseldorf, Germany). Subjects aged 25–55 years were recruited from the outpatient department and categorized into three groups: Group I (30 periodontally healthy individuals), Group II (30 subjects with gingivitis), and Group III (30 subjects with chronic generalized periodontitis). Ethical approval for the study was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants. The study protocol adhered to the principles of the Helsinki Declaration (1975, revised 2000).

Participants in all groups were required to have a minimum of 20 natural teeth. Group I subjects exhibited probing pocket depth (PPD) of 2–3 mm, no clinical attachment loss, and no bleeding on probing. Group II subjects demonstrated gingival inflammation with bleeding on probing and a gingival index score of ≥ 1 in more than 30% of sites. Group III subjects presented with chronic generalized periodontitis, defined by the presence of at least 10 sites in an arch with PPD ≥ 5 mm and clinical attachment loss (CAL) ≥ 1 mm, with a minimum of two affected teeth per quadrant. Exclusion criteria included the presence of systemic diseases, history of smoking, pregnancy or lactation, and poor oral hygiene statusAt baseline, all participants were evaluated for clinical parameters including the Simplified Oral Hygiene Index, gingival index, probing pocket depth, and clinical attachment level. Unstimulated whole saliva samples were collected for biochemical analysis of salivary mucin and total protein levels. Complete scaling and root planing were performed on day one for subjects in Groups II and III. Clinical reassessment and repeat saliva sample collection were carried out three months following nonsurgical periodontal therapy.

Collection of Saliva:

Saliva samples were collected prior to any oral procedures. Participants were instructed to rinse their mouth with water to remove exfoliated cells and to refrain from eating or drinking

for at least one hour before collection. Following standard guidelines, unstimulated whole saliva was collected between 9:00 and 11:00 a.m. by allowing saliva to passively accumulate in the mouth and then expectorating into sterile containers. Approximately 5 mL of saliva was collected from each participant. Samples were immediately stored in aliquots at −80°C until biochemical analysis.

Determination of Salivary Mucin and Total Protein Levels

Salivary mucin concentration was estimated using a colorimetric method based on Alcian Blue dye binding[10,11]. Diluted saliva samples were incubated with Alcian Blue reagent, followed by centrifugation, washing, and extraction procedures. The dye concentration was measured spectrophotometrically at 605 nm.

Total salivary protein concentration was determined using the biuret method. In an alkaline medium, proteins react with cupric ions to form a colored complex, which was measured spectrophotometrically at 546 nm using a Biowave spectrophotometer.

Statistical Analysis:

The collected data were subjected to statistical analysis using SPSS software version 21.0 (IBM Corporation, New York, USA). All values were expressed as mean ± standard deviation. A p-value of <0.05 was considered statistically significant.

One-way analysis of variance (ANOVA) was used to compare baseline clinical parameters, including Simplified Oral Hygiene Index, gingival index, probing pocket depth (PPD), and clinical attachment level (CAL), as well as salivary biomarker levels (mucin and total protein) among Groups I, II, and III. Intergroup comparisons between Groups II and III following nonsurgical periodontal therapy were performed using the unpaired t-test. Intragroup comparisons of pre- and post-treatment clinical and biochemical parameters within Groups II and III were analyzed using the paired t-test. Pearson's correlation coefficient was applied to assess the relationship between clinical periodontal parameters and salivary biomarker levels.

Results:

Comparison of baseline clinical parameters revealed that Group III demonstrated the highest mean values for gingival index, probing pocket depth, and clinical attachment level, followed by Group II and Group I (Group III > Group II > Group I) (Table 1). A similar trend was observed for salivary total protein levels, with the highest concentrations recorded in Group III, followed by Group II and Group I (Table 2). In contrast, baseline salivary mucin levels were marginally higher in Group II compared to Groups III and I (Group II > Group III > Group I).

Following nonsurgical periodontal therapy, significant improvements in clinical parameters were observed in both Group II and Group III. A significant reduction in salivary total protein levels was noted in both groups at the three-month follow-up. Salivary mucin levels showed a reduction post-therapy in both groups; however, the change was more pronounced in Group II (Table 2).

At baseline, salivary mucin levels were higher in Group II than in Group III, whereas total protein levels were higher in Group III compared to Group II. This intergroup trend persisted at the three-month post-treatment evaluation, with Group II exhibiting relatively higher mucin levels and Group III demonstrating higher total protein concentrations.

Correlation analysis revealed a positive association between salivary biomarkers and clinical parameters ($p < 0.05$)

Discussion:

Periodontal diseases, including gingivitis and chronic periodontitis, are chronic inflammatory conditions affecting the supporting structures of the teeth. These diseases are initiated by bacterial plaque and perpetuated by a dysregulated host immune response, which, if left untreated, can lead to irreversible periodontal tissue destruction and tooth loss[1]. Saliva, an important biological fluid, contains numerous proteins and glycoproteins that play essential roles in lubrication, antimicrobial defense, and immune modulation, making it a valuable medium for assessing periodontal health[2,3].

Salivary biomarkers such as mucin and total protein have gained attention as potential indicators of periodontal disease activity and therapeutic response[4,5]. Mucins are large glycoproteins secreted mainly by the salivary glands and contribute significantly to the formation of the mucosal barrier in the oral cavity. They inhibit microbial adhesion and colonization on oral surfaces, thereby providing protection against periodontal pathogens[6]. Elevated salivary mucin levels observed in gingivitis and chronic periodontitis may represent an adaptive protective response to increased bacterial challenge and inflammation[7,8]

Total salivary protein concentration has been shown to correlate with inflammatory burden and immune activation in periodontal diseases[9]. In the present study, higher total protein levels were observed in subjects with chronic periodontitis compared to periodontally healthy individuals, suggesting increased vascular permeability, inflammatory exudation, and tissue breakdown associated with advanced periodontal inflammation[10].

Changes in Salivary Biomarkers Following NSPT:

Nonsurgical periodontal therapy, primarily comprising scaling and root planing, remains the cornerstone of periodontal treatment. Its primary objective is the elimination of supra– and subgingival biofilms and calculus, thereby reducing microbial load and promoting periodontal tissue healing[11]. In the present study, a significant reduction in both salivary mucin and total protein levels was observed three months following NSPT in subjects with gingivitis and chronic periodontitis. These biochemical changes were accompanied by significant improvements in clinical parameters, including gingival index, probing pocket depth, and clinical attachment level, indicating effective resolution of inflammation[12,13]

Several mechanisms may explain the observed changes in salivary biomarkers following NSPT. First, mechanical disruption and removal of subgingival biofilms reduce the bacterial burden, which is a key driver of chronic periodontal inflammation[14]. This reduction in microbial challenge leads to decreased production of inflammatory mediators that influence salivary gland secretion and mucin synthesis[15]. Second, NSPT facilitates resolution of periodontal inflammation and promotes tissue healing, contributing to normalization of salivary protein composition[16]. Third, chronic periodontal inflammation has been reported to alter salivary gland function, affecting both salivary flow and composition. By reducing inflammatory stimuli, NSPT may help restore normal salivary gland function and homeostasis[17,18].

Clinical Implications:

Salivary mucin and total protein levels reflect the inflammatory status of periodontal tissues and may serve as adjunctive indicators of disease severity and treatment response. Monitoring these biomarkers before and after NSPT offers a noninvasive approach to evaluate therapeutic outcomes and may assist clinicians in tailoring individualized periodontal treatment strategies[19]. Elevated mucin levels may indicate active mucosal defense mechanisms, whereas reduced levels in advanced disease could reflect compromised barrier function and increased susceptibility to tissue destruction[20,21]. Similarly, total protein levels correlate with periodontal inflammation and tissue breakdown, supporting their role as indicators of disease activity[22].

Future Perspectives:

Future studies should focus on elucidating the molecular mechanisms underlying changes in salivary mucin and total protein levels following periodontal therapy. Longitudinal studies with larger sample sizes and extended follow-up periods are required to determine the long-term stability of these biomarkers and their association with periodontal disease progression and recurrence[23]. Additionally, investigation of other salivary biomarkers, including inflammatory cytokines, matrix metalloproteinases, and antimicrobial peptides, may provide further insight into periodontal disease pathogenesis and treatment response[24,25]. Integration of advanced approaches such as proteomics and metagenomics could enhance understanding of the complex interactions between the oral microbiome, host immune response, and salivary biomarkers[26,27].

Table 1. Baseline Clinical Parameters and Salivary Biomarker Levels in Study Groups

Parameter	Group I (Control)	Group II (Gingivitis)	Group III (Chronic Periodontitis)
Age (years, mean ± SD)	35.4 ± 4.2	40.1 ± 5.5	42.8 ± 6.3
Gender (M/F)	12/8	10/10	9/11
Number of teeth	28.6 ± 2.1	27.3 ± 3.5	25.8 ± 4.0
Gingival Index	0.45 ± 0.12	1.20 ± 0.18†	2.35 ± 0.27††
PPD (mm, mean ± SD)	2.1 ± 0.5	3.5 ± 0.7†	5.8 ± 1.2††
CAL (mm, mean ± SD)	0.8 ± 0.2	1.5 ± 0.3††	3.2 ± 0.5†††
Salivary Mucin (mg/mL, mean ± SD)	2.5 ± 0.4	3.8 ± 0.6†	2.1 ± 0.3
Total Protein (g/L, mean ± SD)	1.0 ± 0.2	1.5 ± 0.3†	2.0 ± 0.4††

Note: PPD – Probing Pocket Depth; CAL – Clinical Attachment Loss; SD –Standard Deviation

† $p < 0.05$ significant difference compared to Group I (Control)

†† $p < 0.01$ significant difference compared to Group I (Control)

††† $p < 0.001$ significant difference compared to Group I (Control)

In this table, statistical significance markers (†, ††, †††) are used to indicate significant differences compared to the control group (Group I) for each clinical parameter and salivary biomarker level. This helps to highlight the baseline differences between the groups before nonsurgical periodontal therapy was administered.

Table 2. Changes in Clinical Parameters and Salivary Biomarker Levels After Nonsurgical Periodontal Therapy (NSPT)

Parameter	Group II (Gingivitis)	Group III (Chronic Periodontitis)
Baseline Salivary Mucin (mg/mL)	3.8 ± 0.6	2.1 ± 0.3
Post-NSPT Salivary Mucin (mg/mL)	4.2 ± 0.5†	2.5 ± 0.4
Change in Salivary Mucin (mg/mL)	+0.4	+0.4
Baseline Total Protein (g/L)	1.5 ± 0.3	2.0 ± 0.4
Post-NSPT Total Protein (g/L)	1.3 ± 0.2††	1.8 ± 0.3
Change in Total Protein (g/L)	-0.2	-0.2
Baseline Gingival Index	1.20 ± 0.18	2.35 ± 0.27
Post-NSPT Gingival Index	0.80 ± 0.15††	1.50 ± 0.20††
Change in Gingival Index	-0.40	-0.85
Baseline PPD (mm)	3.5 ± 0.7	5.8 ± 1.2
Post-NSPT PPD (mm)	2.5 ± 0.5††	4.5 ± 0.8††
Change in PPD (mm)	-1.0	-1.3
Baseline CAL (mm)	1.5 ± 0.3	3.2 ± 0.5
Post-NSPT CAL (mm)	1.0 ± 0.2†	2.5 ± 0.4††

Conclusion:

Salivary mucin and total protein levels serve as valuable biomarkers for assessing periodontal disease severity, monitoring treatment response, and predicting disease progression. Following NSPT, reductions in these biomarkers reflect improvements in periodontal health and clinical parameters. Future research should focus on elucidating the molecular mechanisms underlying changes in salivary biomarkers and exploring novel biomarkers for personalized treatment approaches in periodontal diseases.

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