ORIGINAL RESEARCH

Salivary Amylase and Mucin in Chronic Periodontitis: Pre- /Posttherapy

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Abstract

Aim: The study aims to investigate the potential of salivary amylase as a reliable biochemical marker for assessing periodontal disease progression, establishing a potential correlation between salivary amylase levels and periodontal disease severity.

Materials and methods: The study included 40 participants, aged 25–65, equally divided into a control and study group of 20 individuals each. Clinical parameters, such as oral hygiene index, gingival index, probing depth, and clinical attachment level were recorded. Saliva samples were collected and analyzed for amylase and mucin levels using a semi-auto analyzer and spectrophotometer, respectively. These clinical parameters and salivary biomarkers were evaluated before and after 45 days of phase I periodontal therapy. Statistical analysis, including independent samples *t*-test, paired samples *t*-test, and correlation analysis were performed to assess the treatment effectiveness and explore associations between clinical parameters and salivary biomarkers.

Results: The study group with chronic generalized periodontitis showed significantly higher salivary amylase (27022.5 ± 8598.9) and mucin levels (3258 ± 724.2) and worse clinical parameters than the control group at baseline. However, after phase I periodontal therapy, the study group exhibited reduced salivary biomarkers amylase (17924.0 ± 4703.6) and mucin (1828.45 ± 314.07) and improved clinical parameters, indicating the effectiveness of the treatment in enhancing periodontal health compared with the control group. Positive correlations were found between clinical parameters and salivary amylase/mucin levels both before and after therapy ($p < 0.001$).

Conclusion: Salivary amylase and mucin levels hold promise as valuable biomarkers for diagnosing active periodontal disease and evaluating treatment outcomes after phase I therapy.

Clinical significance: Salivary biomarker comparison offers a noninvasive diagnostic tool for periodontal disease, improving early detection and personalized treatment planning. Further research is required to validate its clinical value fully.

Keywords: Amylase, Chronic periodontitis, Clinical attachment level, Mucin, Oral hygiene index-simplified.

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Introduction

Chronic periodontitis (CP) is defined as an infectious disease characterized by inflammation within the supporting tissues of the teeth, resulting in progressive attachment loss and bone deterioration.[1] The traditional methods for the diagnosis of these diseases include clinical measurements and radiographic assessments. These are often poorly tolerated by the patients and are also subjected to measurement errors.[2] These methods are often insufficient for determining the sites of active disease, for monitoring quantitatively the response to therapy or for measuring the degree of susceptibility to future disease progression. So nowadays various researches are being conducted to identify the possible compounds in the oral fluids through which it may be possible to assess the presence and severity of these disease as well as to identify the patients at risk for these disease.[3]

Saliva, as an easily accessible and noninvasive surrogate medium, holds great potential for clinical diagnostics. Salivary biomarkers, including those produced by healthy individuals or those affected by specific diseases, have emerged as monitoring molecules for assessing health, disease onset, and disease severity.[3] Saliva is a unique complex, important body fluid which can be easily and rapidly collected and does not require any specialized equipment or techniques. Salivary sample is a simple, Noninvasive and safer method and its storage is easy and cost-efficient.[4]

Saliva comprises various components, including water (99.5%) and a diverse array of biomolecules, such as amino acids, histatins, cystatins, defensins, statherins, lysozyme, proline-rich proteins, carbonic anhydrases, peroxidases, amylase, lactoferrin, mucins, secretory immunoglobulins, lipids, and ions.[5] Amylase and mucin, two important salivary components, are released from the parotid and submandibular glands, respectively, through β-adrenergic stimulation.[6]

α-Amylase is a major lipopolysaccharide-binding protein of *Aggregatibacter actinomycetemcomitans* (Aa) and *Porphyromonas gingivalis* (Pg) and also helps against streptococcal bacterial adherence. Recent studies indicate that salivary mucin shows bactericidal activity against *Aggregatibacter actinomycetemcomitans* which is among the major pathogen responsible for periodontitis.[2] Numerous research endeavors have analyzed the salivary concentrations

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of amylase and mucin, investigating their links with clinical parameters in individuals suffering from gingivitis, CP, and aggressive periodontitis. Sanchez et al.[6] conducted an investigation into salivary levels of amylase and mucin across different types of periodontitis. Their findings revealed elevated levels of salivary amylase and mucin, which were notably associated with increased clinical parameters compared with a healthy control group. This study also supported another study conducted by Swati Kejriwal et al.[2] The results of the study has shown increased concentration of salivary amylase and mucin in gingivitis patients, and increased levels of amylase, decreased mucin concentration in CP patients coincide with clinical parameters compared with healthy group. So far there is sparse literature regarding the comparison of salivary levels of amylase and mucin in chronic generalized periodontitis patients before and after non-surgical periodontal therapy. Hence, the study was designed to evaluate and compare the salivary levels of amylase and mucin in chronic generalized periodontitis patients before and after phase I periodontal therapy which monitors quantitatively the response to therapy as well as helps to identify whether these biomarkers can be used to assess the disease progression, remission, and measure the degree of susceptibility to future disease progression.

Materials and Methods

Study Design

This comparative clinical study was conducted at the Department of Periodontology, Adhiparasakthi Dental College and Hospital (APDCH) from June 2016 to July 2017. Ethical clearance for the study was obtained from the Institutional Review Board, APDCH (Reference No: 2015-MD-Br11-MAN04), ensuring adherence to ethical guidelines and protection of participants' rights. Informed consent was obtained from all participants, ensuring their voluntary participation and understanding of the study's objectives and procedures.

Sample Size and Groups

A total of 40 subjects aged between 25 and 60 years were included in the study. They were divided into two groups: the control group (group A) and the study group (group B). Each group consisted of 20 subjects. Within the study group, there were two subgroups: B1 and B2. Subgroup B1 comprised chronic generalized periodontitis patients before treatment, while subgroup B2 consisted of chronic generalized periodontitis patients after phase I periodontal therapy.

Inclusion and Exclusion Criteria

The control group included subjects with a minimum of 20 natural teeth, a gingival index score of ≤1, and the absence of attachment loss. The study group included subjects with clinical attachment loss of >4 mm in more than 30% of the sites, radiographic evidence of bone loss in >30% of the sites, and bleeding on probing. Exclusion criteria encompassed systemic diseases, smoking habits, pregnancy and lactation, previous periodontal therapy within the past year, use of antibiotics or anti-inflammatory drugs in the past 6 months, a history of salivary gland diseases or oral infections, and oral injuries or bleeding unrelated to gingivitis and periodontitis.

Study Tool

The study evaluated participants' oral hygiene and gingival health using the Oral Hygiene Index-Simplified (OHI-S), and gingival index while probing depth and clinical attachment level (CAL) measured the severity of periodontal disease and treatment response. Saliva samples were collected from both control and study groups using "Spitting" technique," It was first considered both "passive drooling technique" and "spitting technique" outlined by Navazesh (1993) were optimal for unstimulated whole saliva collection. Employing both methods ensured a comprehensive assessment of salivary biomarkers, with both groups treated equally to minimize bias. But Rigorous protocols and methodology required for employing both methods which can only show minor variations in sample collected. So the study followed a single-structured procedure (Spitting technique), rinsing their mouths with clear water for cleansing purpose and then expectorating saliva alone into sterile containers.

In spitting technique, the participants maintained an upright posture, slightly tilting their heads forward for unstimulated saliva collection and to reduce orofacial movements. Participants spit their entire saliva into sterile containers at 60-second intervals for 5 minutes. To mitigate diurnal variation, 5 mL of unstimulated saliva per patient was collected between 11 am and 12 noon. It was immediately sent to the lab, the saliva samples were kept on ice and frozen before being centrifuged at 3000 rpm for 15 minutes. Enzymatic assays and spectrophotometry were then conducted to estimate amylase and mucin levels, ensuring accurate assessment of salivary biomarkers.

The study group underwent phase I periodontal therapy, including complete ultrasonic scaling and subsequent root planning within a 15-day period. On the 45th day after therapy completion, patients were reevaluated, clinical parameters were recorded again, and new saliva samples were collected for amylase and mucin level analysis.

Amylase activity in saliva was determined using the colorimetric method with a semi-auto analyzer machine (Robonic). Mucin concentration was assessed via the Alcian blue method using a spectrophotometer (Chemito Spectrascan UV-2600). Salivary amylase estimation involved adding 0.5 µL of amylase reagent to 10 µL of the sample, feeding it into the machine, and observing the readout. Salivary mucin estimation required incubating the diluted saliva (1:10 dilution) with a 1% Alcian blue solution in sodium acetate buffer. Subsequent centrifugation and analysis determined the mucin concentration.

The dual methods of saliva collection ensured comprehensive assessment and minimized biases. The study's meticulous approach aimed to provide a complete understanding of salivary biomarkers' association with periodontal health and treatment response.

Statistical Analysis

The collected data were subjected to statistical analysis using SPSS (Statistical Package for the Social Sciences) (IBM SPSS Statistics for Windows, version 22.0, Armonk, NY: IBM Corp. Released 2013). The significance level was fixed at 5% ($\alpha = 0.05$). The data obtained in the study were analyzed using several statistical methods. An independent samples *t*-test was conducted to compare the mean values of clinical parameters, salivary amylase, and mucin levels between the control group and the study group at baseline. Paired samples *t*-tests were used to assess the changes in these variables within the study group before and after phase I periodontal therapy. Additionally, Karl Pearson's correlation analysis was performed to determine the correlation between clinical parameters and salivary amylase and mucin levels at baseline and after treatment. These statistical analyses allowed for the evaluation of the significance of the results and the identification of any relationships between variables. A significance level of $p < 0.05$ was used for all statistical tests.

Table 1: Comparison of mean baseline OHI-S score of the control group with study group

Variables	*Group*	*N*	*Mean*	*Std. Dev.*	*t-value*	*p-value*
OHI: Pre-op	Control	20	0.98905	0.095558	16.573	**<0.001**[a]
	Study	20	3.67750	0.719121		

The mean OHI-S score in control group was 0.99 ± 0.096 and in study group was 3.68 ± 0.72. On comparing the mean OHI-S score between groups, the difference was found to be statistically significant ($p < 0.001$).

[a]A *p*-value of 0.001 indicates that if the null hypothesis tested were indeed true, then there would be a one-in-1,000 chance of observing results at least as extreme

Table 2: Mean change in GI score from baseline to postoperative in the study group

Pair	*Variables*	*N*	*Mean*	*Std. Dev.*	*t-value*	*p-value*
Pair 2	GI: Pre-op	20	1.93350	0.400510	12.982	**<0.001**[a]
	GI: Post-op	20	0.94600	0.121196		

The mean GI scores in study group at base line was 1.93 ± 0.40 and postoperative score was 0.95 ± 0.12. On comparing base line score with postoperative score, the reduction in the GI score was fo und to be statistically significant ($p < 0.001$).

[a]A *p*-value of 0.001 indicates that if the null hypothesis tested were indeed true, then there would be a one-in-1,000 chance of observing results at least as extreme

Table 3: Comparison of mean baseline probing depth and CAL of the control group with the study group

Variables	*Group*	*N*	*Mean*	*Std. Dev.*	*t-value*	*p-value*
Probing depth: Pre-op	Control	20	1.31860	0.103043	29.657	**<0.001**[a]
	Study	20	4.10595	0.407492		
CAL: Pre-op	Control	20	1.31910	0.103024	32.809	**<0.001**[a]
	Study	20	4.28822	0.391387		

The mean probing depth and clinical attachment level in control group was 1.32 ± 0.10 mm and 1.32 ± 0.10 mm respectively, and in study group was 4.11 ± 0.41 mm and 4. 29 ± 0.39 mm respectively. On comparing the Pocket depth and CAL between the groups, the differences were found to be statistically significant ($p < 0. 001$).

[a]A *p*-value of 0.001 indicates that if the null hypothesis tested were indeed true, then there would be a one-in-1,000 chance of observing results at least as extreme

Results

The present study had 40 subjects in which 20 study participants were in study group and 20 subjects in control group. The overall female population was higher than the male population and maximum study participants were between the age-group 31–40 (45%) and least study participants were between 71 and 70 years (1%). The mean OHI-S score of pre-op in the control group was 0.99 ± 0.096 and 3. 68 ± 0. 72 in the study group. On comparing the mean and OHI-S score between groups, the difference was found to be statistically significant (Table 1).

The mean GI score in the study group at baseline was 1.93 ± 0.40 and the postoperative score was 0.95 ± 0.12. On comparing a baseline score with a postoperative score, the reduction in the GI score was found to be statistically significant (Table 2).

The mean probing depth and CAL pre-op in the control group was 1.32 ± 0.10 mm and 1.32 ± 0.10 mm, respectively, and in the study group were 4.11 ± 0.41 mm and 4. 29 ± 0.39 mm, respectively. On comparing the pocket depth and CAL between the groups, the differences were found to be statistically significant ($p < 0.001$) (Table 3).

Table 4: Comparison of mean baseline salivary amylase and mucin values of the control group with the study group

Variables	*Group*	*N*	*Mean*	*Std. Dev.*	*t-value*	*p-value*
Amylase: Pre-op (U/L)	Control	20	7599.0	1058.4	10.026	**<0.001**[a]
	Study	20	27022.5	8598.9		
Mucin: Pre-op (µg/mL)	Control	20	763.6	69.5	15.334	**<0.001**[a]
	Study	20	3258.0	724.2		

Mean amylase and mucin values in control group was 7599.0 ± 1058.4 U/L and 763.6 ± 69.5 µg/mL respectively, and study group was 27022 ± 8598.9U/L and 3258.0 ± 724.2 µg/mL respectively. On comparing the two mean values, the differences were found to be statistically significant ($p < 0. 001$).

[a]A *p*-value of 0.001 indicates that if the null hypothesis tested were indeed true, then there would be a one-in-1,000 chance of observing results at least as extreme

Table 5: Mean change in salivary amylase and mucin values from baseline to postoperative in study group

Variables	*N*	*Mean*	*Std. Dev.*	*t-value*	*p-value*
Amylase: Pre-op	20	27022.5	8598.9	6.220	<0.001
Amylase: Post-op	20	17924.0	4703.6		
Mucin: Pre-op	20	3258.05	724.17	11.325	<0.001
Mucin: Post-op	20	1828.45	314.07		

The pre-op mean amylase and mucin values in the control group were 7599.0 ± 1058.4 U/L and 763.6 ± 69.5 µg/mL, respectively, and the values of the study group were 27022 ± 8598.9U/L and 3258.0 ± 724.2 µg/mL, respectively. On comparing the two mean values, the differences were found to be statistically significant ($p < 0.001$) (Table 4).

On comparing the mean baseline salivary amylase and mucin values with postoperative values in study group, the reduction in salivary amylase and mucin levels from the base line (27022.5 ± 8598.9 U/L and 3258. 05 ± 724. 17 µg/mL) to postoperative (17924.0 ± 4703.6 U/L and 1828.45 ± 314.07 µg/mL) was found to be statistically significant (Table 5).

In pre-op and post-op, it was observed that there was a very strong positive correlation, in pre-op ($r = 0. 900$) between amylase and mucin. This correlation was statistically significant ($p < 0.001$). In Post-op ($r = 0.520$) between amylase and mucin. This correlation was statistically significant ($p < 0.05$) (Table 6).

The present study highlights the association between CP and salivary biomarkers. Individuals with CP showed elevated levels of salivary amylase and mucin compared with healthy individuals. However, post-periodontal therapy, a significant decrease in salivary amylase and mucin levels was observed, indicating effective reduction in inflammation and improvement in periodontal health.

Discussion

Saliva is a main component of the host oral immune defense system that can reflect changes in oral and systemic health. Over the past few decades, saliva has been used in disease diagnosis in internal medicine.[7,8] Chronic periodontitis is the most common disease affecting the oral cavity after dental caries. It is the major

Table 6: Pearson correlations between amylase and mucin at pre-op and post-op in the study group

		Amylase: Pre-op
Mucin: Pre-op	Correlation	**0.900**[a]
	p-value	0.000
	N	40
		Amylase: Post-op
Mucin: Post-op	Correlation	**0.520**[a]
	p-value	0.019
	N	20

In preoperative, it was observed that there was a very strong positive correlation ($r = 0.900$) between amylase and mucin. This correlation was statistically significant ($p < 0.001$). In postoperative, it was observed that there was a very strong positive correlation. ($r = 0.520$) between amylase and mucin. This correlation was statistically significant ($p < 0.05$).

[a]$p > 0.05$ is the probability that the null hypothesis is true. 1 minus the *p*-value is the probability that the alternative hypothesis is true. A statistically significant test result ($p \leq 0.05$) means that the test hypothesis is false or should be rejected

cause of tooth loss, so it affects the quality of an individual's life. Therefore, early diagnosis and control of the disease are the main goals for clinicians.[9]

Amylase is a highly abundant protein in saliva. One of the functions of amylase is its endoglycosidase activity. It directly inhibits the growth of certain bacteria and also binds to bacteria lipopolysaccharide (*Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*) bacterial surface structure, and bacterial toxins which are responsible for tissue-destructive inflammatory reactions.[10] Our present study findings suggest that salivary amylase and mucin can be potential biomarkers for assessing the efficacy of periodontal therapy and monitoring the progression of CP.

The current study group exhibited significantly higher levels of salivary amylase compared with the control group, which is consistent with the findings of a previous study by Lorena Da Ros et al. Specifically, their study focused on evaluating the relationship between salivary levels of mucin and amylase. Their study, employing 2D electrophoresis, proposed that the elevated alpha-amylase proteolysis observed in the periodontitis group may be attributed to an augmented specific protease activity in these individuals. This implies that individuals with CP may have an increased capacity for protease-mediated breakdown of proteins, potentially contributing to the pathogenesis of periodontal disease. The findings underscore the potential role of salivary amylase as a biomarker and highlight the importance of further investigations to elucidate the specific protease mechanisms involved in periodontal tissue degradation and their potential therapeutic implications.[8]

Salivary mucin has a high affinity to microorganisms. It entraps and agglutinates the bacteria, fungi, and viral particles. MG2 is bound to the five strains of *Actinobacillus actinomycetemcomitans* and also blocks receptor sites present in bacteria and oral tissues.

Sánchez et al. reported that the periodontal inflammatory process like moderate and severe periodontitis that gives rise to tissue damage and loss had activated the sympathetic system which leads to the release of some salivary proteins. Hence, increased amylase and mucin concentration may be considered an attempt by salivary glands to enhance the protective potential of saliva.[11]

SI Tobón-Arroyave et al. used proinflammatory cytokine IL-1β in saliva as biomarkers and their study has shown the results that salivary IL-1β levels were significantly increased in their study and they suggest that one of the host response factors linked to the clinical manifestations of periodontal disease may be the elevated IL-1 concentration, this shows the importance of analyses of saliva in periodontal disease, in current study, we assess the salivary amylase and mucin and also the salivary IL-1β levels could be studied for more improved correlations.[12]

In Madhulika Banerjee et al.'s study, salivary alpha-amylase levels were used as biomarkers and the levels were noticeably higher in cases of generalized chronic gingivitis, localized CP, and generalized CP compared with healthy controls; the increased levels of amylase in the study group than in healthy controls were also seen in the present study. This shows that increased amylase levels could be due to the presence of the condition.[13]

There is a significant reduction in the mean salivary amylase level from baseline to 45 days from 27022.5 ± 8598.9 U/L to 17924.0 ± 4703.6 U/L and the mean salivary mucin level from baseline to 45 days from 3258.05 ± 724.17 μg/mL to 1828.45 ± 314.07 μg/mL) following periodontal phase I therapy. This may be due to that salivary secretion is a reflex response that is controlled by parasympathetic and sympathetic autonomic systems; it can be influenced by several stimuli.[9] The periodontal inflammatory process gives rise to tissue damage which activates the sympathetic system and induces the increased rate of secretion of amylase and mucin for increasing the protective potential of saliva. These proteins act as the first line of defense mechanism in the oral cavity.[14] After phase I periodontal therapy, the output of these proteins reduced, indicating that the resolution of the inflammatory process made the secretor stimuli disappear.

Pearson's correlation analysis showed a positive and significant correlation between mucin and amylase output with probing depth and CAL before periodontal treatment and a decrease of this output when clinical parameters were improved after treatment and also it correlates with the preoperative salivary amylase, mucin, and post- operative salivary amylase, mucin with periodontal status.

It has been shown that proper and complete periodontal therapy improves clinical parameters and decreased the activity of amylase and mucin in the saliva of patients with CP.

The limitations of the study is the sample size was relatively small, which may affect the generalizability of our findings. Additionally, the study design was limited to a pre/post-analysis, and a longitudinal follow-up would provide more comprehensive insights into the long-term effects of phase I therapy on salivary biomarker levels. Future studies should consider examining a panel of salivary biomarkers to enhance the specificity and accuracy of disease diagnosis and monitoring.

Conclusion

The study indicates that during periodontal disease, salivary glands respond by elevating mucin and amylase output, enhancing saliva's protective capacity. Utilizing these salivary biomarkers, amylase and mucin levels could aid in diagnosing the active phase of periodontal disease and in evaluating treatment effectiveness after periodontal phase I therapy.

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