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Original Article

Assessing Periodontal Health in Tobacco Users: A Salivary Analysis

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Abstract

Background: The risk of periodontal disease is considered to be high among tobacco users compared to those not consuming tobacco in any form. A rise in salivary enzyme levels reflects the destruction of healthy tissues, making it a potential clinical biomarker. Thus, the aim was to assess periodontal health and examine salivary enzyme levels and their correlation with tobacco use.

Methods: Unstimulated saliva was collected using a standard protocol from 128 individuals who visited the outpatient department (OPD) of a dental hospital, divided into four groups. Clinical assessment was done using the gingival index (1963) (GI) and the community periodontal index (1982) (CPI).

Findings: There was a strong correlation between tobacco use and salivary enzyme levels. Tobacco chewers had the most elevated salivary enzyme levels, followed by smokers and passive smokers. Significant periodontal deterioration presenting as an increase in probing depth and clinical loss of attachment (LOA) was seen among the chewers (3.22 ± 0.87 and 2.16 ± 1.27, respectively) and smokers (3.16 ± 0.80 and 1.63 ± 1.38, respectively), which was more than passive smokers (2.75 ± 0.80 and 0.84 ± 0.30, respectively) and was considered significant between all the groups ($P = 0.001$).

Conclusion: Tobacco use in either form affects the severity of periodontal diseases and the levels of salivary enzymes. Thus, salivary enzymes are regarded as reliable biochemical indicators of periodontal tissue damage and can be used as motivators to quit tobacco usage. Additionally, passive smoking was found to have a negative impact on periodontal health and can be considered a risk factor.

Keywords: Passive smoker, Periodontitis, Periodontal disease, Salivary enzymes, Tobacco

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Introduction

Smoking tobacco stands out as the leading environmental contributor to mortality and morbidity, holding the dubious distinction of being the most widespread and detrimental use of tobacco products. Periodontal disease emerges as a prevalent concern among the array of inflammatory diseases with intricate origins and multifaceted causes.[1] After oral cancer, periodontitis looms as the subsequent oral disease linked to tobacco use, further accentuating the detrimental impact of this habit on oral health. A recent systematic analysis concluded that smoking dramatically raises the incidence of periodontitis by 85%.[2] A study conducted on various groups, including smokers, smokeless tobacco users, and dual users, in Central India found 43%, 68%, and 82% prevalence of periodontitis among them, respectively.[3] Periodontal disease results from inflammatory destruction of the periodontal tissue and alveolar bone, which support the teeth. The repercussions of prolonged and severe inflammation in the periodontal region go beyond mere discomfort, often resulting in tooth loss, consequently impacting essential oral functions such as mastication, speech, and facial esthetics.[4] It has been documented as widely prevalent globally, with gingivitis reported to approach 100% in certain studies.[5,6,7] Systemic health conditions, including diabetes and cardiovascular disease, have been related to the persistent inflammation brought on by periodontal illnesses. Furthermore, the spread of oral bacteria through the bloodstream may contribute to respiratory infections and adverse pregnancy outcomes.[8] Tobacco users constitute a notably high-risk group due to the well-established association between tobacco consumption and the development of periodontal diseases, highlighting their heightened vulnerability to these oral health issues.[4,5] It affects the function and growth of periodontal cells, such as the fibroblasts and cells of the periodontal membrane and ligament, resulting in cell

death (apoptosis). It also exacerbates periodontal disease by impairing the immune response, inhibiting immune defenses, and intensifying inflammation. This happens due to reduced T-lymphocytic activity and proliferation, decreased phagocytic activity, and increased production of pro-inflammatory cytokines and oxygen radicals by monocytes. Furthermore, there is a decline in antibody levels against periodontal pathogens and impaired adhesion of human periodontal ligament fibroblasts, eventually damaging and destroying the alveolar bone.[3,9]

Tobacco consumption, whether through smoking or smokeless means, is considered a significant public health concern globally. Saliva, the initial biological fluid exposed to cigarette smoke, is the first line of defense against oxidative stress.[10] Four hundred of the more than 4000 distinct chemicals found in cigarette smoke have been shown to cause cancer. These substances break down the protective proteins, enzymes, and macromolecules in saliva, depriving it of its protective function and leaving the mouth lining vulnerable to inflammatory or degenerative changes.[11] A non-smoker who is exposed to cigarette smoke regularly in a closed space is referred to as a passive smoker or an involuntary smoker. Another definition of a passive smoker is someone who inhales at least one cigarette every day or spends two hours a day in the air that has been contaminated by cigarette smoke.[12] They are acknowledged to have a higher chance of developing periodontitis. Passive smoking, both at home and in the workplace, has been linked to the development of numerous systemic diseases, including cancer, cardiovascular diseases, and serious respiratory conditions. There is no risk-free level of exposure to second-hand smoke.[13] A significant review conducted in 2007 by Johnson and Guthmiller also suggested a potential connection between periodontal disease and passive smoking. After adjusting for other variables, the odds of developing periodontal disease were 1.6 times higher for individuals who were exposed to passive smoking than for those who were not.[14] Odisha leads with the highest consumption of smokeless tobacco, accounting for 42.9% of the adults, according to results reported by GATS-2.[15]

Conventional approaches to diagnosing periodontal disease rely on identifying clinical indicators of inflammation, i.e., clinical parameters like clinical attachment level, recession, bleeding of pockets, and periodontal probing pocket depth, typically the outcome of tissue destruction. This could cause a delay in the disease's detection, which has prompted the quest for more suitable substitutes.[16,17] Furthermore, in smokers, the gingiva tends to bleed less. It appears more hardened, linked with deeper periodontal pockets, potent furcation involvement, greater loss of attachment (LOA), and accelerated bone loss, compared to non-smokers.[18] Thus, before the clinical symptoms appear, salivary biomarkers can be used to predict the existence and severity of periodontitis.[19] Additionally, saliva offers several advantages over other diagnostic methods, including being non-invasive, requiring smaller sample sizes, ensuring strong patient compliance, cost-effectiveness, easy storage and transport, and greater sensitivity with correlation to blood levels.[20] The anti-carcinogenic properties of saliva are largely due to its antioxidant system, which includes various enzymes and molecules that get released into the gingival crevicular fluid and saliva when periodontal tissues are damaged. This comprehensive array of salivary biomarkers, such as uric acid, aspartate aminotransferase, alanine aminotransferase, acid phosphatase, lactate dehydrogenase, and alkaline phosphatase (ALP), is crucial for the early detection of periodontal diseases.[21] In the context of our research, the focus lies on monitoring salivary alkaline phosphatase (S-ALP), salivary lactate dehydrogenase (S-LDH), and salivary uric acid (S-UA) as inflammatory biomarkers in saliva to identify tissue injury in the tobacco user population.

Research indicates that smokers' salivary antioxidant defense system against the buildup of oral stress is suppressed.[22] While little research has been done on the salivary levels of UA. LDH, ALP, and creatine kinase between individuals who smoke and those who do not, there is a significant research gap comparing all three categories, namely tobacco chewers, smokers, and passive smokers, and their salivary levels.[13,23,24] Thus, this study seeks to address this gap by examining and comparing the salivary levels of enzymes ALP, LDH, and antioxidant UA and their impact on periodontal parameters measuring gingival bleeding (GI), periodontal pocket depth (community periodontal index, CPI) and clinical LOA among tobacco users.

Materials and Methods

A cross-sectional, institution-based study was conducted among tobacco users attending the outpatient department (OPD) of a private dental college and hospital in Bhubaneswar. The sample size for the study was calculated using the G*Power 3.1.9.7 software (Heinrich-Heine-University, Düsseldorf, Germany). One hundred twenty-eight participants were calculated for a sample size of 32 per group with 80% power and a 5% significance level. The age group consisted of individuals over 18 years, regardless of periodontal health status. The participants were selected after providing informed consent according to the inclusion and exclusion criteria. Thus, 32 tobacco chewers, 32 smokers, 32 passive smokers, and 32 individuals with no habit of chewing or smoking were included using purposive sampling techniques. The Institutional Review Board of the Institute of Dental Sciences assessed the study protocol and provided ethical clearance (SOA/IDS/IRB/22/42) before the commencement of the study.

Inclusion criteria:

- Group 1 (tobacco chewers): subjects who have been

using chewable tobacco daily for at least a year[25];
- Group 2 (smokers): subjects who had smoked for at least a year, more than 20 cigarettes or beedis each week[25];
- Group 3 (passive smokers): Subjects who were exposed to at least two cigarettes/day on ≥ 5 days/week for at least five years[26] or exposed to air polluted with cigarette smoke for a minimum of 2 hours daily[27];
- Group 4 (control group): Subjects who have never used tobacco in their lifetime.

Exclusion criteria

Participants with a history of xerostomia, long-term pharmaceutical use, periodontal therapy within the last six months, cardiovascular issues, and potentially malignant illnesses of the mouth were excluded.

Sample collection

Saliva samples were collected using sterile sample bottles between 9 am and 12 pm to minimize diurnal variations in the samples. Saliva was collected only from those participants who had not eaten or drunk in the last hour before commencing the saliva collection process. Subjects were instructed to accumulate saliva on the floor of their mouth and then let it drool into the sample bottle (Figure 1). At least three milliliters (3 mL) of unstimulated saliva was collected and immediately placed in an ice bag for transport to the laboratory, where enzyme levels were measured. The specimen jars were coded according to the grouping of participants to maintain confidentiality.

Figure 1. Collection of saliva

Clinical examination

GI,[28] CPI, and LOA scores derived from the WHO Community Periodontal Index of Treatment Needs (CPITN) were among the clinical indicators assessed.[29] Except for third molars, data were gathered at four locations surrounding each tooth, the mesial, buccal, distal, and lingual, using a CPI probe. The examiner applied the clinical scoring criteria to ten patients. The results were compared to those obtained by another faculty member from the same department. The kappa value for inter-examiner reliability was found to be 0.86, showing a high level of agreement between observations. The study was recorded by one of the department's postgraduate students, who had also taken part in all of the training activities and examination procedures.

Biochemical analysis

The collected saliva was then transported to the hospital's biochemistry laboratory to determine the enzyme levels. The enzymes analyzed were S-SALP, S-LDH, and S-UA. Alkaline phosphatase estimation was done using LiquiCHEK alkaline phosphatase (SL) (manufactured by Agappe Diagnostics Ltd., Kerela- 683 562) consisting of alkaline phosphatase (SL) reagent 1 and alkaline phosphatase (SL) reagent 2. For lactate dehydrogenase analysis, an LDH (P-L) kit (manufactured by Coral Clinical Systems, Goa-403 722) consisting of a buffer and starter reagent was used. For uric acid analysis, a uric acid kit (manufactured by Coral Clinical Systems, Goa- 403 202) with one buffer reagent and one enzyme reagent, along with standard uric acid (8 mg/dL), was used. Centrifugation of saliva was done at 1000 rpm for a duration of 10 minutes, and the resultant supernatant was used for biochemical assessment, which was done spectrophotometrically in a BTS 350 semi-autoanalyzer (manufactured by Biosystem 350) (Figures 2-4). The technician analyzing the samples was unaware of the category to which each saliva sample belonged, as the specimen jar identities were kept undisclosed. The reading acquired on the screen (in IU/L) was then recorded and analyzed statistically.

Statistical analysis

The data obtained were compiled using Microsoft Office Excel, version 2019 (Microsoft Corp., Redmond, WA, USA). They were subjected to statistical analysis using the Statistical Package for the Social Sciences version 26.0 (IBM SPSS Statistics, Armonk, NY, USA). Normality testing was done using the Kolmogorov-Smirnov test. The study used descriptive statistics (mean, standard deviation, and percentage) and inferential statistics (Kruskal-Wallis test and Spearman's correlation). The Kruskal-Wallis test was used to evaluate the salivary enzyme levels between the groups. Spearman's correlation was used to analyze the relationship between salivary

enzymes and clinical parameters. The significance level was set at $P < 0.05$, and the results were considered highly significant when $P < 0.01$.

Results

Table 1 shows that among the 128 surveyed individuals, the males were predominantly tobacco smokers 23.4% and tobacco chewers 15.6%, while 9.4% were female tobacco users and only 1.6% were smokers. Additionally, 12.5% of the surveyed population, regardless of gender, were passively exposed to smoking. The mean age of participants in the tobacco user groups ranged from 36.06 ± 6.7 to 49.84 ± 9.2.

Findings from the biochemical analysis revealed significantly distinct mean salivary levels across different groups. Among tobacco chewers, the salivary level of the enzyme ALP was highest, at 62.12 ± 7.14; ALP levels were 48.94 ± 5.59 among smokers and 37.57 ± 3.71 among passive smokers. Similarly, the mean salivary level of LDH among tobacco chewers was 681.22 ± 108.75, followed by smokers (582.95 ± 49.23) and passive smokers (483.29 ± 26.58). Additionally, in the tobacco chewers group, the mean salivary level of the antioxidant UA was 2.71 ± 0.17, while it was 2.37 ± 0.14 for smokers and 2.59 ± 0.97 among passive smokers.

The outcomes derived from examining clinical periodontal parameters showcased significant results in the mean CPI and LOA scores across distinct groups of tobacco exposure. Specifically, the mean CPI and LOA scores were 3.22 ± 0.87 and 1.31 ± 1.46 among tobacco chewers, 3.00 ± 1.04 and 1.31 ± 1.65 among smokers, and 2.59 ± 0.97 and 0.47 ± 0.98 among passive smokers, respectively. Furthermore, the mean GI scores exhibited

Figure 2. Laboratory centrifuge

Figure 3. Analysis of salivary enzymes using semi-autoanalyzer

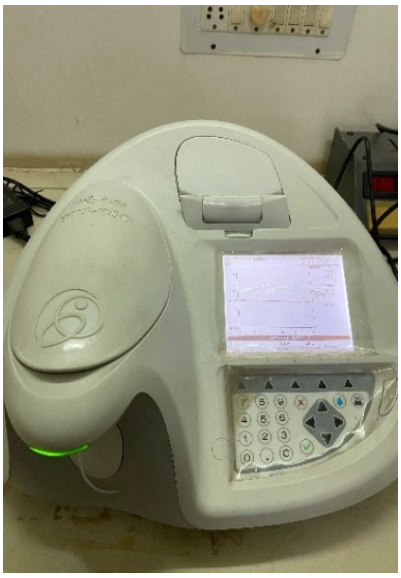

Figure 4. Assessment of results

Table 1. Mean distribution of age, salivary enzymes, and clinical parameters

Type	Gender (N = 128) n (%)		Age (mean ± SD)	ALP (IU/L) (mean ± SD)	LDH (IU/L) (mean ± SD)	UA (IU/L) (mean ± SD)	CPI score (mean ± SD)	LOA (mm) (mean ± SD)	GI (mean ± SD)
	Male n (%)	Female n (%)							
Chewer	20 (15.6)	12 (9.4)	49.84 ± 9.2	62.12 ± 7.14	681.22 ± 108.75	2.71 ± 0.17	3.22 ± 0.87	2.16 ± 1.27	2.22 ± 0.49
Smoker	30 (23.4)	2 (1.6)	40.81 ± 10.8	48.94 ± 5.59	582.95 ± 49.23	2.37 ± 0.16	3.16 ± 0.80	1.63 ± 1.38	1.81 ± 0.47
Passive smoker	16 (12.5)	16 (12.5)	36.06 ± 6.7	37.57 ± 3.71	483.29 ± 26.58	2.26 ± 0.13	2.75 ± 0.80	0.84 ± .030	1.70 ± 0.54
Control	15 (11.7)	17 (13.3)	39.22 ± 9.4	21.99 ± 5.95	390.4 ± 55.16	2.19 ± 0.29	2.63 ± 0.75	0.81 ± 0.98	1.66 ± 0.74
P value				0.001	0.001	0.001	0.001	0.001	0.001

GI, Gingival index; LOA, Loss of attachment; CPI, community periodontal index; LDH, lactate dehydrogenase; UA, uric acid.

significant variations among the groups, reaching the highest level in the tobacco users group with a mean of 2.22 ± 0.49.

After recording variations in salivary enzymes and antioxidant levels among the groups (comprising tobacco chewers, smokers, and passive smokers), a post hoc analysis was conducted. The mean rank resulting from the analysis illustrates that tobacco chewers exhibited the highest mean rank among all salivary enzymes and antioxidants, followed by smokers and passive smokers. Furthermore, a statistically significant association was identified among the various salivary enzymes and antioxidants, elucidating potential interconnections within the observed biochemical profiles.

The results from Table 2 indicate a significant break to moderate correlation between the activities of these enzymes and antioxidants and the clinical periodontal parameters (CPI, LOA, and GI) in the tobacco chewer, smoker, and passive smokers groups. S-ALP was strongly associated with the LOA across these groups.

Figure 5 illustrates that the tobacco chewer group exhibited a higher prevalence of participants with a CPI score of 4, but was not significantly associated between periodontitis and tobacco use. Among the smokers and passive smokers, a preponderance of CPI score 3 indicates susceptibility to potential periodontal deterioration, possibly influenced by nicotine's vasoconstrictive activities on periodontal tissues. Figure 6 illustrates that LOA score 1 was frequent in the passive smoker group compared to the non-smoker group. Conversely, LOA scores 2 and 3 were less frequent in the passive smoker group compared to the non-smoker group. In Figure 7, the substantial occurrence of score 3 in the chewer group highlighted a marked severity of gingival inflammation, with the lowest frequency among the passive smoker group. Moderate gingival inflammation was seen among the chewers, followed by the passive smokers. The elevated prevalence of score 3 underscored the need for attention to preventive measures and oral health interventions in individuals engaging in tobacco use.

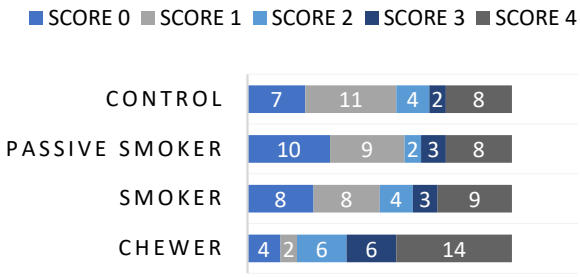

Figure 5. CPI scores among the groups

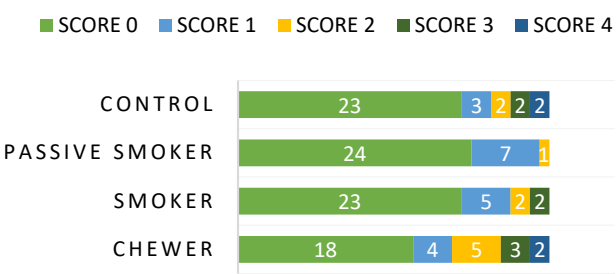

Figure 6. LOA scores among the groups

Discussion

The study measured and compared tobacco users' salivary enzyme levels and clinical parameters. The study surveyed 128 individuals, revealing that males predominantly smoked or chewed tobacco, while females had lower usage rates, with 12.5% of both genders exposed to passive smoking. Biochemical analyses showed elevated salivary enzyme levels (ALP, LDH) and antioxidant (UA) in tobacco users, particularly among chewers. Clinical periodontal parameters (CPI, LOA, and GI) were highest in tobacco chewers, followed by smokers and passive smokers. Correlation analysis of biochemical markers and clinical parameters showed a moderate positive relation between S-ALP and LOA.

Saliva, explored as a diagnostic tool, is easily accessible, and its collection is non-invasive, offering biomarkers like genetic information and proteins. It can help diagnose oral and systemic disorders at the chairside as it contains hormones, antibodies, enzymes, and other compounds. Many constituents enter saliva from the blood, reflecting physiological functioning. This non-invasive method generally results in better patient compliance, is cost-effective, and is nearly as accurate as blood tests.[30] The descriptives of the studied population reveal statistically significant findings, except for smoking, which was found to be low among females due to the societal stigma as it is often considered taboo in the cultural context. A noteworthy rise in S-ALP values across all groups was observed compared to the normal level (12 IU/L).[30,31]

Table 2. Spearman's correlation analysis between salivary enzymes and clinical parameters

Spearman's rho (ρ)		CPI	LOA	GI
S-ALP	Correlation coefficient	0.434*	0.451*	0.348*
S-LDH	Correlation coefficient	0.388*	0.392*	0.306*
S-UA	Correlation coefficient	0.217*	0.234*	0.191*

GI, Gingival index; LOA, Loss of attachment; CPI, community periodontal index; S-LDH, salivary lactate dehydrogenase; S-UA, salivary uric acid; S-ALP, salivary alkaline phosphatase .
*Correlation is significant at $P<0.05$.

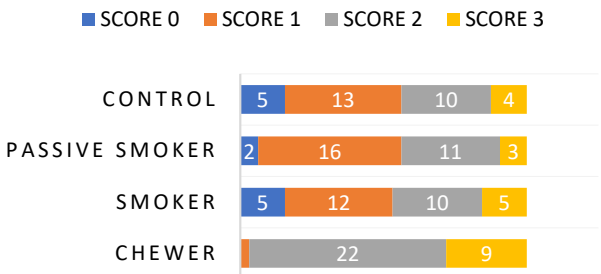

Figure 7. GI scores among the groups

Similar findings were observed for S-LDH, consistent with Ibraheem et al.[13] In our study, we noted a statistically significant increase in uric acid in all groups with the highest mean level among the tobacco chewers (2.71) followed by smokers (2.37), and similar results were found in a study by Pullishery et al.[25] Uric acid, which is a key non-enzymatic antioxidant in saliva, constitutes about 85% of its antioxidant capacity and diminishes by over 1/3 of normal levels in smokers, highlighting the impact of tobacco use on antioxidant defense mechanisms.

In investigating the correlation between periodontal parameters and salivary levels, this study revealed a weak to moderate yet statistically significant correlation, which was contradictory to the results by Ibraheem et al.[13] The increased enzymatic activity in periodontitis plays a crucial role in facilitating the regeneration of compromised periodontal tissues. Furthermore, salivary enzymes actively contribute to protective functions by breaking down and eliminating bacteria or debris within the oral environment. The augmented blood flow to inflamed regions stimulates the salivary glands, resulting in an overall increase in saliva production and changes in its enzymatic composition.

The CPI scores, which provide insights into periodontal health through assessments of gingival bleeding, dental calculus, and periodontal pocket depth, suggest a distinct association between periodontitis and tobacco use. Compared to smokers and non-smokers, tobacco chewers had higher rates of periodontal destruction. The results were in agreement with the studies by Karemore et al and Ling et al.[3,32] This could be brought on by the combined effects of an extended period of chewing the smokeless tobacco product and its absorption rate into the oral mucosa and also the higher concentration of irritants in it.[33] Among smokers (2.3%) and passive smokers (2.3%), the prevalent CPI score of 3 signifies an increased vulnerability to potential periodontal deterioration, but the risk was lower in comparison to the tobacco chewers (4.7%). A study by Vaishnavi Devi and Leelavathi found that smokeless tobacco users had probing depths of 1–4 mm and 5–8 mm compared to smokers. However, smokers exhibited greater probing depths of 8–12 mm and above 12 mm compared to smokeless tobacco users.[34] This inclination is likely attributable to the vasoconstrictive effects of nicotine on the periodontal tissues, suggesting a noteworthy correlation between smoking habits and the risk of periodontal issues. Second-hand tobacco smoke, containing nicotine, carcinogens, and toxins, could also contribute to this scenario. The average range of nicotine levels in the air in smokers' residences and in places where smoking is allowed is usually 2 to 10 micrograms/m^3.[35] The results align closely with studies by Ibraheem et al, Rezaei and Sariri, and Sanders et al, confirming comparable trends in periodontal pocket depth and suggesting a relationship between second-hand smoke and periodontal tissue.[13,36,37] Additionally, the non-tobacco user group, comprising only seven subjects with a score of 0, prompts consideration of other factors like oral hygiene practices, genetics, diet, and overall health, emphasizing the multifaceted nature of periodontal well-being within this specific group.

The minimal loss of attachment score of 0, observed among the tobacco chewers group, raises questions regarding the general health of their periodontal tissues. There was an elevation in LOA score 1 within the passive smoker group when compared to the smokers and non-smokers groups. Conversely, LOA scores 2 and 3 showed a decrease in the passive smokers group in comparison to the smokers and non-smokers groups. This discrepancy could be a result of specific characteristics like dietary or oral hygiene practices of the study population or variations in the amount or duration of exposure among individuals, aligning with the results of Ibraheem et al.[13] Also, the notable prevalence of score 3 in the chewers group underscores an escalated severity of gingival inflammation characterized by redness and hypertrophy, indicating a substantial impact on their gingival health. This observation is indicative of the potential adverse effects of tobacco chewing on gingival tissues. Furthermore, the trend continues in the smoker's group, reinforcing the notion that tobacco use, whether through chewing or smoking, is directly linked to compromised gingival health. The elevated prevalence of score 3 underscores the need for attention to preventive measures and oral health interventions in individuals engaging in tobacco use. Gingival score 2, indicating moderate gingivitis, was highest among the tobacco chewers (17.2%), followed by passive smokers (8.6%), which was in alignment with the results of a study by Vaishnavi Devi and Leelavathi, for the tobacco chewers, followed by smokers.[34] Severe gingivitis was mostly reported for tobacco chewers (7%) followed by smokers (3.9%), which was in alignment with the results by Kulkarni et al,[38] While Vaishnavi Devi and Leelavathi reported higher frequency of gingivitis in smokers followed by smokeless tobacco users.[34] Smoking reduces blood flow to the periodontium, leading to decreased bleeding on probing and a masked presentation of gingival inflammation. In contrast, smokeless tobacco directly contacts and irritates gingival tissues, causing localized inflammation and more pronounced gingivitis. Smoking also causes immuno-inflammatory imbalances and oxidative stress, which can accelerate inflammation and infection susceptibility, but its vascular effects can mask gingivitis signs.

The results of this study revealed variations in the levels of salivary enzymes and antioxidants among all groups. Notably, the passive smoker group showed significantly greater enzyme levels than the control non-tobacco user group, exceeding the normal range. This observation suggests that enzymes are likely released

from polymorphonuclear cells during inflammation, as well as from osteoblasts and periodontal ligament fibroblasts during processes such as bone development and periodontal tissue repair.[13] In the active stages of periodontitis, the destruction of alveolar bone leads to the release of intracellular contents, including ALP, into the gingival crevicular fluid and saliva.[39] Furthermore, there is a reasonable basis to infer that second-hand smoke might cause similar systemic effects on periodontal tissues as direct smoking. This suggests that both active and passive smoking impact health similarly, though the extent of these effects may differ.[13]

One strong aspect of our study is the simultaneous consideration of all three groups of tobacco users and establishing their connection with periodontitis. By doing so, we aimed to provide a more comprehensive understanding of the relationship between salivary levels, enzymatic profiles, and periodontal health, filling a notable gap in existing research. Several limitations are evident in the current study. Firstly, the study design is cross-sectional, which might restrict the ability to establish causation. Additionally, categorizing participants into groups lacked consideration for their periodontal health status. Also, dietary habits and oral hygiene practices, two significant determinants of oral health, were not systematically considered in the study design. The omission of comprehensive assessments regarding other salivary enzymes, such as malondialdehyde or thiocyanate, further limits the study's ability to capture a holistic picture of the factors influencing periodontal health. Future research should address these limitations through more rigorous study designs, including longitudinal approaches and comprehensive assessment protocols, to enhance the validity and generalizability of findings.

Conclusion

Periodontal disease, being multifactorial, involves both microbial challenges and host responses. It is influenced by various factors, including lifestyle habits like tobacco consumption. The study found that males have a greater prevalence of tobacco use than females and that they are significantly more exposed to passive smoking. Biochemical studies demonstrated that tobacco chewers had more periodontal tissue damage and alveolar bone loss than non-smokers, as evidenced by larger pocket depth and higher clinical attachment levels. It is also possible to conclude that passive smoking harms periodontal health, even if it is not severe. Thus, cigarette smoking, including both active and passive forms, is a well-established risk factor that can exacerbate the development of periodontal disease. Elevated salivary concentrations of enzymes can be particularly informative, reflecting oxidative stress of the tissues and serving as a potent indicator of the inflammatory status of the periodontium. Higher levels of ALP, LDH, and UA can quantitatively estimate the inflammatory status of gingival and periodontal tissues, revealing periodontal destruction, such as periodontal pockets, gingival bleeding, and suppuration, potentially leading to earlier disease diagnosis than clinical parameters. Biomarkers should be promoted to improve clinical practice and thus be utilized to assist clinical indices of inflammation. Correlation analysis confirms the association between salivary enzyme levels and clinical periodontal parameters, particularly between S-ALP and LOA, indicating greater periodontal attachment loss and highlighting the enzyme's potential role as a biomarker for periodontal disease severity. Given the limited research on salivary enzyme levels in tobacco chewers assessing periodontal status, our findings carry weight and can be considered credible in this understudied context. Salivary diagnostics can offer a non-invasive and accessible means of assessing the inflammatory status of the periodontium. By leveraging biomarkers, clinicians can enhance their ability to diagnose and monitor the impact of tobacco usage on oral health. This approach aligns with a broader trend in embracing salivary diagnostics as a valuable tool in future clinical assessments, marking a progressive step toward a more comprehensive and personalized approach to patient care.

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Author's Contribution

Conceptualization: Dharmashree Satyarup, Jagruti Nanda.
Data curation: Jagruti Nanda, Priyanca Panigrahi.
Formal analysis: Subhashree Ray, Priyanca Panigrahi.
Methodology: Dharmashree Satyarup, Jagruti Nanda, Subhashree Ray.
Resources: Subhashree Ray.
Supervision: Dharmashree Satyarup, Subhashree Ray.
Writing–original draft: Jagruti Nanda.
Writing–review & editing: Dharmashree Satyarup, Jagruti Nanda.

Competing Interests

The authors have no conflict of interest to declare.

Ethical Approval

This institutional-based cross-sectional study was granted ethical approval from the Institutional Review Committee (IRB) and Institutional Ethical Committee (IEC) at the Institute of Dental Sciences, Bhubaneswar (SOA/IDS/IRB/22/42) before its initiation. Participants provided informed consent, and their confidentiality was safeguarded throughout the study.

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