RESEARCH PAPER

Periodontal Disease Progression in Smokers, Chewers and Dual Tobacco Users: A Comparative Case–Control Study

Dr. Sumit Narang[1], Dr. Amit Kumar Singh[1], Dr. Srijan Sunar[1], Dr. Mehwash Haque[1], Dr. Ananya Saxena[1], Dr. Deeksha Dubey[1]

[1] Department of Periodontology and Oral Implantology, People's Dental Academy, People's University, Bhanpur, Ayodhya Bypass, Bhopal, Madhya Pradesh – 462037, India.

ABSTRACT

Background and Objectives:

Tobacco consumption in smoked and smokeless forms is a well-established risk factor for periodontal destruction. Comparative clinical evidence evaluating smokers, chewing tobacco users, dual users and non-users remains limited. This study aimed to assess and compare periodontal disease severity across these groups.

Materials and Methods:

A case–control study was conducted involving 200 participants divided equally into four groups: healthy controls, chewing tobacco users, smoking tobacco users and dual tobacco users. Periodontal parameters recorded included Gingival Index, Plaque Index, Probing Pocket Depth, Clinical Attachment Loss and tooth loss. Statistical analysis was performed using ANOVA followed by Tukey post-hoc testing.

Results:

Dual tobacco users demonstrated the highest severity across all periodontal parameters, followed by smokers and chewers. Healthy controls exhibited minimal periodontal changes. Intergroup differences were statistically significant ($p < 0.001$).

Conclusions:

Combined tobacco use accelerates periodontal destruction more aggressively than single-form exposure. Targeted cessation counseling and preventive periodontal care are warranted.

Keywords: Periodontal disease; Tobacco smoking; Smokeless tobacco; Dual tobacco use; Clinical attachment loss

How to cite this article: Sumit Narang. Periodontal Disease Progression in Smokers, Chewers and Dual Tobacco Users: A Comparative Case–Control Study. Int J Drug Deliv Technol. 2026;16(76s):1610-1614. DOI: 10.25258/ijddt.16.76s.151.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Periodontal disease is a chronic inflammatory condition involving the gingiva, periodontal ligament, cementum and alveolar bone and remains a leading cause of tooth loss in adults. Its onset and progression are influenced by a complex interaction of microbial challenge and host response, modified by environmental, behavioral and genetic factors. Among these, tobacco use represents one of the most significant and preventable risk determinants.

Cigarette smoking is consistently identified as the strongest environmental risk factor for periodontitis, with extensive evidence demonstrating its association with increased disease prevalence, severity and rate of progression [1,2]. Periodontitis is characterized by progressive destruction of the periodontal ligament and alveolar bone, resulting in periodontal pocket formation, gingival recession and eventual tooth loss [3]. Smoking alters the host–microbial equilibrium by impairing immune surveillance, reducing gingival blood flow through nicotine-induced vasoconstriction and accelerating connective tissue and bone breakdown, thereby masking clinical signs of inflammation while promoting deeper periodontal destruction [4].

Smokeless tobacco use has also been implicated in periodontal breakdown, although its disease pattern differs from that of smoking. Chewing tobacco is commonly associated with localized gingival recession, attachment loss and periodontal pocketing, particularly at the site of quid placement [5]. The direct chemical irritation, abrasive injury and sustained exposure to tobacco-specific nitrosamines contribute to connective tissue degradation and alveolar bone loss. Experimental and clinical studies suggest that smokeless tobacco influences inflammatory enzyme activity and redox balance within periodontal tissues, thereby modifying disease expression [6].

Tobacco exposure further disrupts both innate and adaptive immune responses. Cigarette smoke affects cell-mediated and humoral immunity, with studies of gingival crevicular fluid demonstrating reduced levels of cytokines, enzymes and polymorphonuclear leukocytes in smokers compared with non-smokers [7]. Neutrophil function is particularly affected; smoking impairs chemotaxis and phagocytosis, limiting effective elimination of periodontal pathogens. In heavy smokers, excessive generation of reactive oxygen species

contributes to oxidative stress and direct periodontal tissue injury [8].

While the individual effects of smoking and smokeless tobacco on periodontal health have been well documented, comparative clinical data assessing smokers, chewers and individuals who use both forms concurrently remain limited. Dual tobacco users are exposed to both systemic and localized pathogenic mechanisms, raising the possibility of a synergistic effect on periodontal destruction that is not fully understood.

The present study aims to evaluate and compare periodontal outcomes among smoking tobacco users, chewing tobacco users, dual tobacco users and healthy individuals, thereby providing clinically relevant evidence on the differential and combined impact of tobacco habits on periodontal disease progression.

MATERIALS AND METHODS

Study Design and Study Population

This research complied with ethical standards, following the Helsinki Declaration (1975, revised in 2013) and relevant national regulations. A case–control study was conducted to evaluate periodontal disease severity among individuals with different forms of tobacco exposure. A total of 200 participants were enrolled and categorized into four groups of equal size. Group A comprised healthy individuals with no history of tobacco use (n = 50). Group B included chewing tobacco users (n = 50), Group C consisted of smoking tobacco users (n = 50) and Group D included individuals with concurrent smoking and chewing tobacco habits (dual users; n = 50).

Sample Size Determination

The sample size was calculated based on previously reported standard deviation values for periodontal parameters. Assuming a detectable mean difference of 1 mm, a standard deviation of 0.5 mm, a confidence level of 95% and adequate study power, the required sample size was estimated to be 50 participants per group.

Inclusion and Exclusion Criteria

Participants aged between 30 and 45 years with a diagnosis of chronic periodontitis were included in the study. Individuals in the tobacco-exposed groups had a documented history of smoking, chewing tobacco, or both for a minimum duration of three years. Only systemically healthy individuals were enrolled.

Participants were excluded if they had discontinued tobacco use within the preceding six months, had received antibiotic therapy during the previous two months, were pregnant or lactating, or had a history of oral malignancy. Individuals undergoing orthodontic or periodontal therapy, as well as those with bleeding disorders or receiving anticoagulant medication, were also excluded.

Clinical Parameters Assessed

Periodontal status was assessed using standardized clinical parameters, which served as dependent variables in the analysis. These included the Gingival Index (GI), Plaque Index (PI), Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL) and mean tooth loss. All measurements were recorded following established clinical criteria.

Instruments and Clinical Examination

Clinical examinations were performed using standard diagnostic instruments, including a UNC-15 periodontal probe and a Community Periodontal Index (CPI) probe. All instruments were sterilized using autoclave procedures prior to use to maintain infection control standards.

Statistical Analysis

All collected data were compiled and analyzed using standard statistical software. Descriptive statistics were calculated for all clinical parameters and results were expressed as mean ± standard deviation. Intergroup comparisons of periodontal parameters among healthy controls, chewing tobacco users, smoking tobacco users and dual tobacco users were performed using one-way analysis of variance (ANOVA). $p < 0.001$ interpreted as highly significant.

RESULTS

The mean periodontal clinical parameters recorded across the four study groups are presented in **Table 1, Figure 1.**. Healthy controls demonstrated minimal periodontal involvement, with low mean Gingival Index (0.7 ± 0.2), Plaque Index (0.8 ± 0.3), Probing Pocket Depth (2.1 ± 0.4 mm), Clinical Attachment Loss (1.8 ± 0.3 mm) and negligible tooth loss (mean = 0.1).

Chewing tobacco users showed increased periodontal impairment compared with healthy individuals, with mean Gingival Index and Plaque Index values of 1.4 ± 0.4 and 1.9 ± 0.6, respectively. Mean Probing Pocket Depth and Clinical Attachment Loss in this group were 3.6 ± 0.5 mm and 3.2 ± 0.6 mm, along with a mean tooth loss of 1.2.

Smoking tobacco users exhibited more pronounced periodontal destruction than chewers. The mean Probing Pocket Depth was 4.2 ± 0.6 mm and mean Clinical Attachment Loss was 4.0 ± 0.7 mm. Higher Gingival Index (1.8 ± 0.5) and Plaque Index (2.2 ± 0.7) scores were also observed, with a mean tooth loss of 2.0.

Dual tobacco users demonstrated the most severe periodontal breakdown across all evaluated parameters. This group recorded the highest mean Gingival Index (2.4 ± 0.6), Plaque Index (2.8 ± 0.8), Probing Pocket Depth (5.1 ± 0.7 mm) and Clinical Attachment Loss (5.4 ± 0.8 mm), along with the greatest mean tooth loss (3.1).

One-way analysis of variance revealed statistically significant differences among the four groups for all clinical parameters ($p < 0.001$). Post-hoc Tukey analysis confirmed that dual tobacco users differed significantly from healthy controls, chewing tobacco users and smoking tobacco users across all outcomes (**Table 1**).

DISCUSSION

The present study demonstrates a graded pattern of periodontal destruction that closely corresponds to the form and intensity of tobacco exposure. Individuals using tobacco in any form exhibited significantly poorer periodontal parameters than healthy controls, with the greatest severity observed among dual tobacco users. This gradient supports the concept that different tobacco habits exert distinct yet overlapping pathogenic effects on periodontal tissues.

Smoking tobacco was associated with more extensive periodontal destruction than chewing tobacco, reflected by higher mean probing pocket depth and clinical attachment loss values. Smokers in the present study exhibited a mean probing pocket depth of 4.2 ± 0.6 mm and a clinical attachment loss of 4.0 ± 0.7 mm, compared with 3.6 ± 0.5 mm and 3.2 ± 0.6 mm, respectively, among chewing tobacco users. These findings are consistent with previous reports attributing smoking-related periodontal damage to systemic immunosuppression, impaired vascular perfusion and accelerated alveolar bone loss [8,9]. Inhaled tobacco smoke affects both innate and adaptive immune responses, reducing effective bacterial clearance while promoting connective tissue breakdown.

Chewing tobacco users, while demonstrating less generalized destruction than smokers, showed clear evidence of periodontal deterioration when compared with healthy individuals. The disease pattern in chewers is often site-specific, corresponding to the area of quid placement, where prolonged chemical exposure and mechanical irritation contribute to localized gingival recession and attachment loss. The comparatively lower mean tooth loss observed in chewers (1.2 teeth) relative to smokers (2.0 teeth) suggests a predominantly localized rather than systemic mode of tissue injury, as reported in earlier clinical and epidemiological studies [10].

Gingival and plaque indices were higher among smokers than chewers; however, this observation must be interpreted cautiously. Nicotine-induced vasoconstriction in smokers is known to mask overt clinical signs of gingival inflammation, producing deceptively lower bleeding scores despite advanced periodontal destruction [11]. In contrast, chewing tobacco often produces visible gingival changes, including recession and mucosal alterations, due to direct chemical and abrasive effects on the gingival tissues. These habit-specific differences highlight the limitations of relying solely on inflammatory indices without considering underlying attachment loss.

Dual tobacco users demonstrated the most severe periodontal outcomes across all measured parameters, indicating a synergistic effect of combined tobacco exposure. This group recorded the highest mean gingival index (2.4 ± 0.6), plaque index (2.8 ± 0.8), probing pocket depth (5.1 ± 0.7 mm) and clinical attachment loss (5.4 ± 0.8 mm). Notably, the mean attachment loss in dual users was nearly three times higher than that observed in healthy controls, confirming accelerated breakdown of the periodontal supporting structures. The highest mean tooth loss (3.1 teeth) further underscores the cumulative and irreversible nature of periodontal damage associated with concurrent smoking and chewing habits [12–14].

The findings of this study emphasize that dual tobacco use poses a substantially greater periodontal risk than single-form tobacco consumption. Exposure to both systemic effects of smoking and localized effects of smokeless tobacco likely amplifies inflammatory dysregulation, oxidative stress and tissue destruction. These results highlight the need for targeted periodontal screening, habit-specific risk assessment and aggressive tobacco cessation counseling, particularly for individuals with multiple tobacco habits.

Future studies incorporating longitudinal designs, biochemical markers of inflammation and oxidative stress and microbiological profiling would further clarify the mechanistic pathways underlying the synergistic effects observed in dual tobacco users.

CONCLUSION

Within the limitations of this case–control study, tobacco use was associated with a clear increase in periodontal disease severity, with the extent of destruction varying according to the form of exposure. Dual tobacco users exhibited the greatest periodontal breakdown, followed by smokers and chewing tobacco users, indicating a synergistic detrimental effect of combined habits. Smoking alone produced more extensive periodontal damage than chewing tobacco, suggesting a stronger systemic impact on periodontal tissues. These findings reinforce the need for early identification of tobacco-related periodontal risk and the integration of targeted tobacco cessation and preventive strategies into routine dental care.

CONFLICT OF INTEREST

We hereby certify that we do not have any financial or personal relationships that could bias the content of this work

AUTHOR'S CONTRIBUTIONS

Conceptualization, **S.N.**; methodology, **A.K.S.** and **S.S.**; software, not applicable; validation, **M.H., A.S. and D.D.**; formal analysis, **S.S. and A.S.**; investigation, **A.K.S., S.S. and M.H.**; resources, **S.N.**; data curation, **S.S. and D.D.**; writing—original draft preparation, **A.K.S.**; writing—review and editing, **S.N., A.S. and D.D.**; visualization, **D.D.**; supervision, **S.N.**; project administration, **S.N.**; funding acquisition, not applicable.

ACKNOWLEDGEMENTS

The authors acknowledge all participants and institutional support staff involved in the study.

Abbreviations

GI – Gingival Index
PI – Plaque Index
PPD – Probing Pocket Depth
CAL – Clinical Attachment Loss

TABLES

Table 1: Mean Clinical Parameters Across Groups

Parameter	Healthy	Chewers	Smokers	Dual Users
GI	0.7±0.2	1.4±0.4	1.8±0.5	2.4±0.6
PI	0.8±0.3	1.9±0.6	2.2±0.7	2.8±0.8
PPD(mm)	2.1±0.4	3.6±0.5	4.2±0.6	5.1±0.7
CAL(mm)	1.8±0.3	3.2±0.6	4.0±0.7	5.4±0.8
Tooth Loss (mean)	0.1	1.2	2.0	3.1

FIGURES

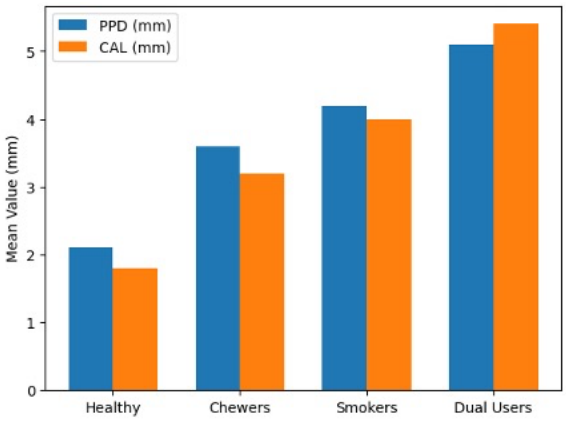

Figure 1A. Comparison of mean probing pocket depth (PPD) and clinical attachment loss (CAL) among healthy controls, chewing tobacco users, smoking tobacco users, and dual tobacco users.

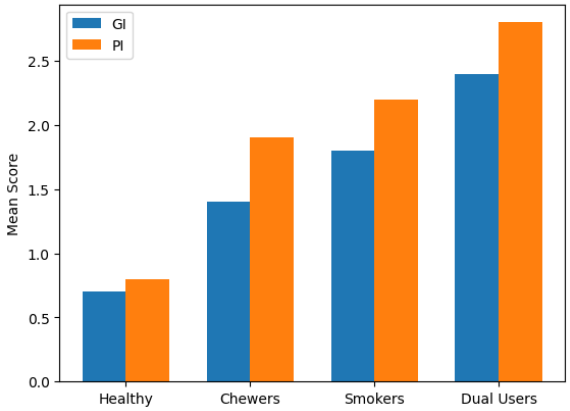

Figure 1B. Comparison of mean gingival index (GI) and plaque index (PI) among healthy controls, chewing tobacco users, smoking tobacco users, and dual tobacco users.

REFERENCES

1. Gautam DK, Jindal V, Gupta SC, Tuli A, Kotwal B, Thakur R. Effect of cigarette smoking on the periodontal health status: A comparative, cross sectional study. J Indian Soc Periodontol 2011;15:383-7.
2. Mittal S, Dani N, Abullais SS, Al-Qahtani NA, Shah K. Effect of smoking and tobacco chewing on periodontal disease and non-surgical treatment outcome: A clinical and biochemical study. J Int Acad Periodontol 2017;20:12-8.
3. Chu YH, Tatakis DN, Wee AG. Smokeless tobacco use and pe- riodontal health in a rural male population. J Periodontol 2010 81: 848–854.
4. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. J Clin Periodontol. 2002;29:771–76.
5. Maddipati Sreedevi, Alampalli Ramesh, Chini Dwarakanath. Periodontal status in smokers and nonsmokers: A clinical, microbiological and histopathological study. Int J Dent. 2012.
6. Gautam DK, Jindal V, Gupta SC, Tuli A, Kotwal B, Thakur R. Effect of cigarette smoking on the periodontal health status: A comparative, cross sectional study. J Indian Soc Periodontol 2011;15:383-7.
7. Katuri KK, Alluri JK, Chintagunta C, Tadiboina N, Borugadda R, Loya M, et al. Assessment of periodontal health status in smokers and smokeless tobacco users: A cross-sectional study. J Clin Diagn Res 2016;10:C143-6
8. Dave, Anuja Kashyapbhai; Dave, Bela; Thakker, Viral; Joshi, Nancy; Katariya, Mahendrakumar; Patel, Harsh1. Evaluation of Periodontal Health Among Tobacco Chewers, Smokers and Non-Tobacco Users: A Case–Control Study. Advances in Human Biology 11(Suppl 1):p S111-S115.
9. Anand P, Mishra S, Nagle D, Kamath N, Kamath K, Anil S. Patterns of Periodontal Destruction among Smokeless Tobacco users in a Central

Indian Population. In.: Research Square Platform LLC; 2021.
10. Nasir, S.M., Sultana, T., Hashmi, S. et al. Patterns and predictors of periodontal disease and tooth loss among users of smokeless tobacco. BMC 23, 428.
11. Sinha DN, Gupta PC. Tobacco use among students in Oris- sa and Uttar Pradesh. Indian Pediatr 2005;42:846–847.
12. Sinha DN, Gupta PC, Pednekar M. Tobacco use among students in Bihar (India). Indian J Public Health 2004;48: 111–117.
13. SubramanianSV,NandyS,KellyM,GordonD,DaveySmith G. Patterns and distribution of tobacco consumption in India: cross sectional multilevel evidence from the 1998–9 nation- al family health survey. BMJ 2004;328:801–806.