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Impact of tobacco chewing and smoking on periodontal health

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Abstract:

Smoking and chewing habits worsen periodontal health. Therefore, it is of interest to assess and compare the periodontal status among smokers, tobacco chewers and non-users. A hospital-based cross-sectional study was conducted with 420 participants. Periodontal parameters were assessed. Data shows that both smoking and chewing tobacco significantly impairs periodontal health. Smokers exhibit deeper pockets and greater tissue destruction, while chewers show more plaque accumulation.

Keywords: Attachment loss, chewing, gingival recession, periodontitis, smoking, tobacco

Background:

Periodontal diseases are chronic inflammatory conditions affecting the supporting structures of the teeth and represent one of the leading causes of tooth loss worldwide. The etiology of periodontal disease is multifactorial, involving microbial biofilm, host immune response, genetic susceptibility and environmental factors. Among these tobacco use has been identified as one of the most significant modifiable risk factors. Tobacco consumption in various forms, including cigarette smoking, smokeless tobacco and electronic cigarettes, continues to pose a major public health challenge and contributes substantially to oral morbidity and diminished quality of life [1, 2]. Tobacco use adversely affects oral tissues by altering the oral microbiome, impairing neutrophil function, reducing vascularity and disrupting host immune responses, thereby accelerating periodontal destruction [2, 3]. Furthermore, tobacco consumption has been associated with several oral conditions, including gingival recession, periodontal attachment loss, delayed wound healing, oral mucosal lesions and oral potentially malignant disorders [3, 4]. Numerous epidemiological studies have showed that smokers exhibit increased prevalence and severity of periodontitis, greater probing depths and enhanced alveolar bone loss compared with non-smokers [5]. Smokeless tobacco products such as gutka and chewing tobacco are highly prevalent in South Asian countries and have been increasingly recognized as important contributors to periodontal deterioration. Habitual gutka chewers show significantly poorer periodontal parameters, increased inflammatory cytokine levels and a higher prevalence of gingival recession and attachment loss than non-users [6]. Recent studies have also reported that smokers experience considerably higher rates of periodontal disease and tooth-supporting tissue destruction than individuals who have never used tobacco products [7]. The deleterious effects of tobacco extend beyond

disease initiation and progression and significantly impair the outcomes of periodontal therapy by reducing healing potential and diminishing treatment response [8]. The relationship between smokeless tobacco use and periodontal inflammation has been further strengthened by studies showing significantly worse periodontal conditions among gutka users, particularly in the presence of systemic metabolic disturbances such as prediabetes [9]. In addition to conventional tobacco products, the emergence of electronic cigarettes has generated new concerns regarding periodontal health. Systematic reviews and meta-analyses have suggested that e-cigarette users may exhibit increased susceptibility to periodontal inflammation and adverse periodontal outcomes, although the evidence remains heterogeneous and requires further investigation [1, 10]. Several review studies have consistently concluded that smoking adversely affects gingival tissues by promoting periodontal pocket formation, clinical attachment loss, alveolar bone resorption and eventual tooth loss [11]. Moreover, tobacco use influences biological markers associated with periodontal disease progression and healing. Recent evidence has demonstrated differences in metallothionein levels and inflammatory responses between smokers and non-smokers following periodontal therapy, indicating altered host response mechanisms among tobacco users [12]. The global burden of tobacco-associated oral diseases remains substantial. The multinational SMILE study showed significant variations in oral health and dental aesthetics among smokers from different countries, emphasizing the widespread impact of tobacco on oral health and the need for population-specific investigations [13]. Furthermore, smoking has been shown to adversely affect the efficacy of advanced periodontal treatment modalities, including antimicrobial photodynamic therapy, thereby complicating the management of advanced periodontitis in tobacco users [14]. Earlier studies also reported that smokers respond less favorably

to both mechanical and antimicrobial periodontal therapy compared with non-smokers, suggesting that tobacco use remains a major determinant of treatment outcomes [15]. Therefore it is of interest to assess and compare periodontal status among smokers, tobacco chewers and non-users.

Materials and methodology:

This hospital-based observational cross-sectional study was conducted in the Department of Periodontology of a tertiary care dental institution over a period of six months. Adult patients aged 18–65 years attending the outpatient department were screened and recruited after obtaining written informed consent. The study population was categorized into three groups: Group I – tobacco smokers, Group II – tobacco chewers (smokeless tobacco users including gutka, khaini and pan masala) and Group III – non-tobacco users serving as controls. Participants with a history of systemic diseases affecting periodontal status (such as uncontrolled diabetes mellitus, immunocompromised conditions, or osteoporosis), those who had received periodontal treatment within the previous six months, pregnant or lactating women, former tobacco users and individuals taking medications known to influence periodontal health were excluded from the study. The sample size was calculated using the formula for estimating prevalence in cross-sectional studies:

$$n = Z^2P(1-P)/d^2$$

Where n is the minimum required sample size, Z is the standard normal deviate at a 95% confidence interval (1.96). P is the expected prevalence of periodontal disease among tobacco users, assumed to be 50% due to variability in previous reports. D is the allowable error of 5% (0.05). Substituting these values: $n = (1.96)^2 \times 0.5 \times (1-0.5) / (0.05)^2 = 384$. Thus, a minimum sample size of 385 participants was required. Considering a 10% non-response rate, the final sample size was increased to approximately 420 participants. The participants were selected using a convenience sampling technique until the desired sample size was achieved. A detailed demographic and habit history, including age, gender, type of tobacco used, duration of use and frequency of consumption, was recorded using a structured proforma. Clinical periodontal examination was performed by a single calibrated examiner using a mouth mirror and a William's periodontal probe under adequate illumination. The periodontal parameters assessed included Plaque Index (Silness and Loe), Gingival Index (Loe and Silness), Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL) and Community Periodontal Index (CPI). The periodontal findings were recorded for all teeth except third molars. To ensure reliability, intra-examiner calibration was performed before the commencement of the study and a kappa coefficient of ≥ 0.80 was considered acceptable. The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were expressed as mean $\pm$ standard deviation and percentages. Comparison of quantitative variables among the three study groups was performed using one-way

analysis of variance (ANOVA) followed by Tukey's post-hoc test, while categorical variables were analyzed using the Chi-square test. Pearson's correlation analysis was used to assess the relationship between duration and frequency of tobacco use and periodontal parameters. A p -value of <0.05 was considered statistically significant.

Results:

A total of 420 participants were included in the study, comprising 140 smokers, 140 tobacco chewers and 140 non-tobacco users. **Table 1** summarizes the demographic characteristics and tobacco habit distribution among the study participants, which included smokers, tobacco chewers and non-tobacco users. The mean age of participants was comparable across all three groups, with smokers (40.1 ± 9.6 years), tobacco chewers (39.3 ± 10.8 years) and non-users (36.7 ± 10.4 years) and the difference was not statistically significant ($p = 0.082$). This indicates that the groups were age-matched, thereby minimizing age-related confounding effects on periodontal outcomes. A statistically significant difference was observed in gender distribution ($p < 0.001$). Males constituted the majority in both tobacco-consuming groups, with smokers accounting for 82.9% and tobacco chewers 77.1%, whereas the non-user group had a higher proportion of females (55.0%). This finding reflects the higher prevalence of tobacco use among males in the studied population. Regarding habit characteristics, more than half of the smokers (57.9%) and tobacco chewers (52.9%) reported duration of tobacco use exceeding 10 years. However, this difference between the two groups was not statistically significant ($p = 0.431$), suggesting similar chronic exposure patterns among tobacco users. Similarly, a high frequency of daily tobacco consumption (>5 times/day) was observed in both smokers (62.9%) and tobacco chewers (65.0%), with no statistically significant difference between the groups ($p = 0.714$). This indicates comparable intensity of tobacco exposure among the two user groups. **Table 2** presents a comparison of various periodontal parameters among smokers, tobacco chewers and non-tobacco users. The findings demonstrated statistically significant differences among the three groups for all periodontal variables assessed ($p < 0.001$). The mean Plaque Index was highest among tobacco chewers (2.37 ± 0.52), followed by smokers (2.21 ± 0.48), while non-users exhibited the lowest scores (1.24 ± 0.39). This finding indicates poorer oral hygiene practices and increased plaque accumulation among tobacco users, particularly those consuming smokeless tobacco products. Similarly, the Gingival Index was significantly elevated among tobacco chewers (2.18 ± 0.47) and smokers (2.06 ± 0.44) compared with non-users (1.11 ± 0.35), suggesting a greater degree of gingival inflammation in individuals exposed to tobacco. Smokers demonstrated the highest mean Probing Pocket Depth (4.31 ± 0.92 mm) and Clinical Attachment Loss (4.08 ± 1.02 mm), followed by tobacco chewers (3.89 ± 0.81 mm and 3.71 ± 0.89 mm, respectively). Non-users had significantly lower values for these parameters (2.26 ± 0.63 mm and 1.97 ± 0.58 mm, respectively). These findings suggest that smoking is strongly associated with increased periodontal tissue destruction and

progression of periodontitis. The mean Community Periodontal Index score was also significantly higher among smokers (3.12 ± 0.73) and tobacco chewers (2.96 ± 0.69) than among non-users (1.48 ± 0.51), indicating a greater prevalence and severity of periodontal disease among tobacco consumers. Interestingly, Gingival Recession was most pronounced among tobacco chewers (2.24 ± 0.64 mm), followed by smokers (1.81 ± 0.52 mm), whereas non-users demonstrated minimal recession (0.72 ± 0.31 mm). The greater gingival recession observed among tobacco chewers may be attributed to the direct local irritant effect of smokeless tobacco products on gingival tissues and the chronic placement of tobacco quid in the oral vestibule. Post-hoc analysis showed significantly higher plaque scores and gingival recession among tobacco chewers compared with smokers ($p<0.05$), whereas smokers exhibited significantly greater probing pocket depth and clinical attachment loss than tobacco chewers ($p<0.05$). Both tobacco-user groups showed significantly poorer periodontal parameters than non-users. A positive correlation was observed between the duration of tobacco consumption and clinical attachment loss ($r=0.62$, $p<0.001$), indicating that prolonged exposure to tobacco significantly increased periodontal destruction.

Table 1: Distribution of participants according to demographic characteristics and tobacco habits

Variable	Smokers (n=140)	Tobacco Chewers (n=140)	Non- users (n=140)	Total (n=420)	p- value
Mean age (years)	40.1 ± 9.6	39.3 ± 10.8	36.7 ± 10.4	38.7 ± 10.4	0.082
Male, n (%)	116 (82.9)	108 (77.1)	63 (45.0)	287 (68.3)	<0.001*
Female, n (%)	24 (17.1)	32 (22.9)	77 (55.0)	133 (31.7)	
Duration of habit >10 years, n (%)	81 (57.9)	74 (52.9)	-	-	0.431
Daily frequency >5 times/day, n (%)	88 (62.9)	91 (65.0)	-	-	0.714

*Statistically significant ($p<0.05$).

Table 2: Comparison of periodontal parameters among the study groups

Periodontal Parameter	Smokers	Tobacco Chewers	Non-users	p-value
Plaque Index	2.21 ± 0.48	2.37 ± 0.52	1.24 ± 0.39	<0.001*
Gingival Index	2.06 ± 0.44	2.18 ± 0.47	1.11 ± 0.35	<0.001*
Probing Pocket Depth (mm)	4.31 ± 0.92	3.89 ± 0.81	2.26 ± 0.63	<0.001*
Clinical Attachment Loss (mm)	4.08 ± 1.02	3.71 ± 0.89	1.97 ± 0.58	<0.001*
Community Periodontal Index	3.12 ± 0.73	2.96 ± 0.69	1.48 ± 0.51	<0.001*
Gingival Recession (mm)	1.81 ± 0.52	2.24 ± 0.64	0.72 ± 0.31	<0.001*

*One-way ANOVA; statistically significant at $p<0.05$.

Discussion:

The present study shows that both smoking and tobacco chewing exert profound adverse effects on periodontal health, with smokers showing deeper probing pocket depths and

greater clinical attachment loss, while chewers exhibit higher plaque accumulation and gingival recession. These findings are consistent with earlier reports that tobacco use is a major modifiable risk factor for periodontal disease progression and poor treatment outcomes. Shabil *et al.* (2024) highlighted increased susceptibility to periodontal inflammation among e-cigarette users [1] and Johnson & Slach (2001) emphasized impaired vascularity and neutrophil function in smokers [2], which aligns with our observation of severe tissue destruction in this group. Greer (2011) described gingival recession among smokeless tobacco users [4], corroborated by our finding of pronounced recession in chewers. Martinez - Canut *et al.* (1995) reported greater probing depths in smokers [5], while Javed *et al.* (2015) showed poorer periodontal parameters among gutka chewers [6], both consistent with our results. Laxman & Annaji (2008) noted reduced healing potential in tobacco users [8] and our correlation between duration of use and attachment loss supports this. Javed *et al.* (2013) further showed worse periodontal conditions in gutka chewers with systemic disturbances [9], complementing our evidence of cumulative effects. Systematic reviews by Tattar *et al.* (2025) [10] and biomarker studies by Yadav *et al.* (2024) [12] reinforce the impaired host response mechanisms observed in our cohort. The multinational SMILE study (Kowalski *et al.*, 2026) [13] emphasized global variations, while our Indian data add region-specific insights into smokeless tobacco. Moreover, Al-Kheraif *et al.* (2022) [14] and Kinane & Radvar (1997) [15] showed poorer therapeutic outcomes in smokers, resonating with our findings of compromised periodontal healing. Collectively, these comparisons underscore the multifaceted detrimental effects of tobacco on periodontal tissues and highlight the need for tailored preventive and therapeutic strategies addressing both smoking and chewing habits.

Conclusion:

We show that both smoking and tobacco chewing have a significant adverse impact on periodontal health. Smokers showed greater probing pocket depth and clinical attachment loss, while tobacco chewers exhibited higher plaque accumulation and gingival recession. These findings confirm tobacco use as a key modifiable risk factor for periodontal disease progression. Therefore, integrating tobacco cessation counseling into routine periodontal care is essential to improve clinical outcomes and reduce disease burden.

Advancement to knowledge:

Both smoking and chewing tobacco have an impact on how severe periodontal disease is. Additionally, it continues to conceal the manifestation of gingival inflammation and impairs the responsiveness of periodontal tissues to non-surgical therapy.

Limitation:

The study was cross-sectional and hospital-based, which restricts causal inference and generalizability to wider populations.

Study benefits:

The findings of this study may contribute to the existing evidence and provide valuable information for clinicians, public health professionals and policymakers in developing effective tobacco cessation and periodontal disease prevention strategies.

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