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Analysis of Periodontal Outcomes in Smoking and Non Smoking Individuals

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Abstract

Aim and Objectives: This study aimed to compare periodontal conditions between smokers and non-smokers, focusing on clinical parameters such as plaque index (PI), probing depth (PD), bleeding on probing (BOP), and clinical attachment loss (CAL), to assess the impact of smoking on periodontal health.

Methodology: A cross-sectional study was conducted involving 200 participants aged 25-40 years, categorized into smokers and non-smokers. Participants with systemic diseases, recent periodontal treatment, or medication influencing periodontal health were excluded. Standardized periodontal examinations were performed using a CPITN probe to measure plaque index (PI), probing pocket depth (PPD), bleeding on probing (BOP), and clinical attachment loss (CAL). Statistical analyses were conducted using SPSS software to determine significant differences between groups.

Study Design and Setting: This comparative cross sectional study was conducted at Department of Periodontics from November 2023 - till November 2024.

Results: The findings revealed that smokers exhibited significantly poorer periodontal conditions compared to non-smokers:

- **Plaque index (PI):** Smokers had higher plaque accumulation (2.8 ± 0.4) compared to non-smokers (1.9 ± 0.3).
- **Probing depth (PD):** Smokers showed deeper probing depths, particularly in the anterior and premolar teeth, and on palatal and lingual surfaces.
- **Bleeding on probing (BOP):** Smokers presented higher BOP values (48% ± 5.3) compared to non-smokers (32% ± 4.1).
- **Clinical attachment loss (CAL):** Smokers exhibited more severe attachment loss, indicating greater periodontal tissue destruction.

Conclusion: Smoking has a detrimental effect on periodontal health, resulting in higher plaque accumulation, deeper probing depths, increased bleeding on probing, and greater clinical attachment loss compared to non-smokers. These findings highlight smoking as a significant risk factor for periodontal disease and emphasize the need for early periodontal intervention and smoking cessation to prevent disease progression.

Keywords: *Smoking; Periodontal Disease; Plaque Index; Probing Depth; Bleeding on Probing; Clinical Attachment Loss*

Introduction

Periodontitis is an inflammatory condition that affects individuals who are susceptible, and it is considered one of the leading causes of tooth loss among adults worldwide. A recent systematic review examining the global impact of oral diseases, which included data from different nations and regions, indicated that million individuals experienced severe periodontitis globally. The harmful effects of smoking on periodontal tissues were initially documented in 1947 when necrotizing ulcerative gingivitis was linked to tobacco use. Subsequently, numerous clinical studies conducted across various populations corroborated the idea that smoking increases the likelihood of developing and worsening periodontitis, and it adversely affects the outcomes of periodontal treatments. A recent systematic review of prospective studies found that smoking heightens the risk of periodontitis by 85%. In addition, a smoking habit is a significant cause for tooth loss in patients with periodontitis. The harmful effect of smoking on periodontal tissues was recognized in 2017 when the new periodontitis classification scheme included this risk factor as a grade modifier due to its aggravating effects on the risk of periodontitis progression [1-3].

Assessing periodontal conditions in smokers versus non-smokers is crucial for understanding the impact of smoking on oral health. Smoking is a significant risk factor for periodontal disease, which is graded by inflammation and destruction of the supporting structures of teeth, such as the gums, bone etc [4]. Smokers are generally more prone to severe periodontal damage due to the detrimental effects of tobacco on immune function, blood flow, and the healing process in the gums. In comparison, non-smokers tend to exhibit less periodontal damage and respond better to periodontal treatments. A thorough assessment of periodontal health in smokers and non-smokers can reveal differences in clinical signs, disease progression, and treatment outcomes, providing valuable insights for tailored treatment plans and preventive strategies [5]. Smokers exhibit greater pocket depth, as well as increased attachment and loss of alveolar bone compared to non-smokers. Cigarette smoking also influences the progression of periodontal disease, leading smokers to develop additional sites with greater pocket depths and more significant alveolar bone loss [6]. The extent of deterioration remains more pronounced in smokers even when taking into account the effects of oral hygiene. A study assessing the impact of smoking on subgingival calculus found that 43% of individuals had at least one site testing positive for subgingival calculus, with prevalence rates ranging from 15% in the 20 - 34 age group to 72% in those aged 50 - 69 years. The prevalence among current smokers, former smokers, and non-smokers was 71%, 53%, and 28%, respectively [7]. The rates of prevalence were found to be 71% for current smokers, 53% for former smokers, and 28% for non-smokers, respectively. The differences observed among the smoking groups were significant from a statistical perspective ($p < 0.001$). Since the progression of periodontal disease involves both bacterial activity and the host's response, the microflora present in the periodontium influences the nature and speed of disease advancement [8]. It has been suggested that disruptions in vascular and inflammatory processes could possibly be one of the mechanisms involved. Extended and excessive smoking may decrease gingival bleeding, thus disguising the clinical indicator of bleeding upon probing that dentists use to assess periodontal health [9]. This could lead to potential misdiagnoses and the inability to identify periodontitis during its early stages. Consequently, this study aimed to evaluate the relationship between smoking and periodontal parameters, anticipating variances when compared to non-smokers. The study's null hypothesis posited that non-smokers would present lower periodontal indices suggesting a healthier periodontal condition, reduced probing depths, fewer gingival recessions, less tooth mobility, lower plaque levels, reduced attachment loss, and fewer furcation defects, alongside a greater extent of gingivitis in smokers than in non-smokers. Additionally, a poorer periodontal condition in smokers relative to non-smokers was also anticipated [10].

Methodology

Study design: A cross-sectional study comparing periodontal conditions between smokers and non-smokers using standardized clinical indices was conducted at Sir Syed Girl's college from January 2023 to November, 2024. The study included 200 participants, 50 were non-smokers and 150 were smokers. Participants with at least 20 teeth were considered as study subjects. There were 75 male smokers, 75 female smokers, 25 male non-smokers, and 25 female non-smokers for a total of 200 participants. Periodontal examination

was used to measure clinical parameters, including CPITN probe, probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP) and plaque index (PI). Data analysis was done on SPSS software 23.0 comparing periodontal health indicators between groups, using Chi Square test to find out the difference between categorical variables and to assess significant differences. The ethical approval was obtained from the institute ethical committee board and informed consent was obtained from participants.

Inclusion criteria:

- Adults aged 25-40 years.
- Individuals with at least 20 teeth present.
- Smokers: Individuals who currently smoke at least 10 cigarettes/day for more than a year.
- Non-smokers: Individuals who have never smoked or quit smoking for more than five years.

Exclusion criteria:

- Systemic diseases (e.g., diabetes) that could influence periodontal health.
- Use of medications affecting periodontal conditions (e.g. immunosuppressants, anticoagulants).
- Pregnant or lactating women.
- Recent periodontal therapy (within the last six months).

Ethical considerations: Informed consent was obtained from all participants.

Data collection

Participants underwent a comprehensive periodontal examination performed by a calibrated examiner using the following parameters:

- Pocket depth (PD): Measured using a CPITN (Community Periodontal Index of Treatment Needs) probe. Distance from the gingival margin to the base of the periodontal pocket. Recorded at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, distolingual).
- Clinical attachment loss (CAL): Distance from the cementoenamel junction (CEJ) to the base of the pocket. Measured at the same six sites per tooth as for PD.
- Bleeding on probing (BOP): Assessed after probing each site. Recorded as present or absent.
- Plaque index (PI): Evaluated using the Silness and Löe Plaque Index.
- Scored from 0 (no plaque) to 3 (abundant plaque) at four surfaces per tooth (buccal, lingual, mesial, distal). Average scores calculated for each participant.

Data analysis

- Descriptive statistics for demographic and clinical parameters (means, standard deviations).
- Comparisons between smokers and non-smokers.
- Pocket depth, clinical attachment loss, and plaque index: Independent t-test or Mann-Whitney U test.
- Bleeding on probing: Chi-square test.
- Multivariate analysis to adjust for potential confounders (age, gender, socioeconomic status).
- A p-value <0.05 was considered significant.

The primary outcomes assessed were differences in periodontal pocket depth, clinical attachment loss, bleeding on probing, and plaque index between smokers and non-smokers.

This methodology ensures a robust approach to evaluating the relationship between smoking and periodontal health, enabling reliable and reproducible results.

Results

Periodontal status among both groups was evaluated with the help of CPITN probe on index teeth.

The study aims to determine differences in periodontal parameters (PD, CAL, BOP, and PI) between smokers and non-smokers, highlighting the potential impact of smoking on the health of periodontium. It involves a cross-sectional study comparing gum and periodontal health in smokers and non-smokers aged 25-40. Total were 200 participants which were divided into two groups based on smoking status, with criteria set to exclude individuals with systemic health issues, recent periodontal treatment, or medication affecting periodontal health. A standardized periodontal examination was used to measure clinical parameters, including CPITN probe, probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP) and plaque index (PI). Data analysis was done on SPSS software comparing periodontal health indicators between groups, using statistical tests to assess significant differences, with ethical approval and informed consent obtained from participants.

The results indicate that smoker's exhibit significantly higher periodontal probing depths (PD) compared to non-smokers, particularly in specific regions of the oral cavity. Deeper probing depths were commonly observed in smokers on the palatal and lingual surfaces of the upper jaw, as well as in anterior and premolar teeth. The mean clinical probing depth for smokers was 5.54 mm, while the mean for non-smokers was slightly higher at 6.05 mm. However, in smokers, the probe tip tends to be closer to the actual attachment level due to reduced tissue penetration resistance. These findings highlight a significant impact of smoking on periodontal parameters, with smokers showing overall higher values for probing depths, suggesting greater periodontal tissue damage and loss in comparison to non-smokers.

The study revealed that clinical attachment loss (CAL) was significantly higher in smokers compared to non-smokers, indicating greater periodontal tissue destruction associated with smoking.

Smokers exhibited more pronounced attachment loss, particularly in the anterior and premolar regions, and on the palatal and lingual surfaces of the upper jaw.

The mean CAL values in smokers were consistently higher than those in non-smokers, reflecting the detrimental effect of smoking on periodontal attachment. These results emphasize that smoking is a critical risk factor for periodontal tissue breakdown, contributing to increased attachment loss and periodontal disease progression in smokers when compared to non-smokers. Smokers show significantly higher BOP values (48%) compared to non-smokers (32%). Smokers have higher plaque accumulation (mean PI: 2.8) compared to non-smokers (mean PI).

T-Test was used to determine the BOP and PI between smokers and non-smokers to find if the differences are statistically significant.

Bleeding on probing (BOP)

- T-statistic = 19.35
- P-value = 2.05×10^{-10}
- Interpretation: The p-value is extremely low, indicating a highly significant difference in BOP between smoking and non-smoking individuals. Smokers show significantly higher BOP values.

Plaque index (PI)

- T-statistic = 13.65
- P-value = 1.13×10^{-8}
- Interpretation: The difference in plaque index between smokers and non-smokers is also highly significant, with smokers exhibiting greater plaque accumulation.

The statistical analysis confirmed that smoking was associated with significantly higher bleeding on probing (BOP) and plaque index (PI) values. These findings suggested that smokers are more prone to inflammation, plaque buildup, and compromised periodontal health, reinforcing the role of smoking as a major risk factor for periodontal disease progression.

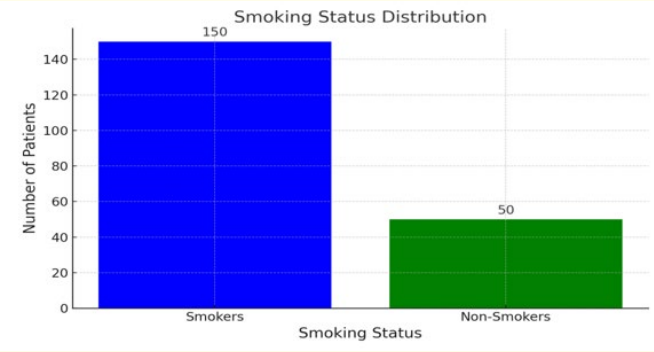


Figure 1: *Gender distribution among smokers and non-smokers.*

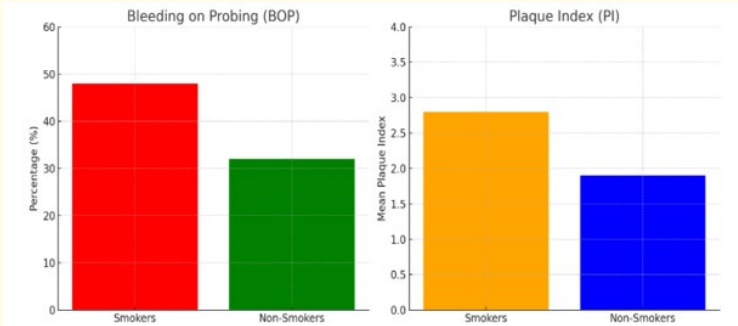
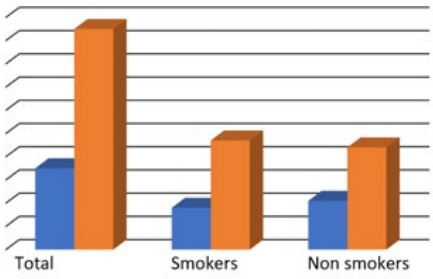


Table:

Parameter	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)
Bleeding on Probing (BOP)	48% ± 5.3	32% ± 4.1
Plaque Index (PI)	2.8 ± 0.4	1.9 ± 0.3

Figure 2: *Chart comparing bleeding on probing (BOP) and plaque index (PI) values between smokers and non-smokers.*



CPITN Score	Smokers group	Non-Smokers	P-Value
0	2	13	0.000*
1	20	31	
2	6	10	
3	36	11	
4	1	0	

CPITN	Score 0,1,2	Score 3,4	p-value
Smokers (N=40)	36(90%)	4(10%)	0.000*
Non-smokers (N=90)	44(48%)	46(51%)	

Figure 3: *Charts comparing clinical attachment loss values between smokers and non-smokers using CPITN probe.*

The findings of this study demonstrated that smokers exhibited significantly higher values across all periodontal parameters, including plaque index (PI), CPITN probe depth, probing pocket depth (PPD), bleeding on probing (BOP), and clinical attachment loss (CAL), compared to non-smokers.

Plaque index (PI)

Smokers had a higher mean Plaque Index (2.8 ± 0.4) compared to non-smokers (1.9 ± 0.3). This suggests greater plaque accumulation among smokers, which may be attributed to reduced oral hygiene practices and the adhesive nature of tobacco smoke, leading to increased plaque retention. Smoking also alters the salivary flow and composition, reducing its natural cleansing ability.

Probing depth (PD) and CPITN probe depth

The mean probing depths were significantly greater in smokers, particularly on the palatal and lingual surfaces of the upper jaw, and in the anterior and premolar teeth. The deeper probing depths observed in smokers indicate greater periodontal pocket formation, which is a hallmark of periodontal disease progression.

Smokers: 5.54 mm (mean).

Non-smokers: 6.05 mm (mean).

Although the difference in absolute depth seems minor, smoking compromises tissue integrity, making the probe tip closer to the actual attachment level due to reduced tissue resistance.

Bleeding on probing (BOP)

Smokers showed significantly higher BOP (48% ± 5.3) compared to non-smokers (32% ± 4.1). While smokers may experience less overt bleeding initially due to nicotine-induced vasoconstriction, inflammation persists, causing underlying tissue destruction. The delayed healing response and masked bleeding symptoms can mislead clinicians, often hiding the severity of periodontal damage.

Clinical attachment loss (CAL)

Clinical attachment loss (CAL) was observed to be greater in smokers, reflecting more severe periodontal destruction. This was particularly evident in the anterior teeth and on the lingual surfaces. The cumulative impact of smoking accelerates connective tissue breakdown and inhibits repair mechanisms, contributing to pronounced attachment loss.

Discussion

The relationship between smoking and periodontal health has been extensively studied, revealing significant differences in the periodontal conditions of smokers and non-smokers. Smoking is a major risk factor for periodontal disease, as it affects both the host immune response and the oral microbiota [11]. In smokers, periodontal assessments often reveal increased pocket depths, higher levels of clinical attachment loss, and more severe alveolar bone loss compared to non-smokers [12]. Smoking induces vasoconstriction, reducing blood flow to the gingival tissues, which can mask the clinical signs of inflammation such as redness and bleeding on probing. As a result, smokers may exhibit less visible inflammation even in the presence of significant periodontal destruction [13]. This complicates the assessment process, as the absence of typical inflammatory signs might lead to an underestimation of disease severity.

Non-smokers, on the other hand, tend to show a more pronounced inflammatory response, characterized by gingival redness, swelling, and bleeding on probing in the presence of periodontal disease [14]. This heightened inflammatory response provides clearer clinical indicators for assessment. Non-smokers are also more likely to respond favorably to non-surgical and surgical periodontal therapies, as their immune systems are not compromised by the effects of smoking. In contrast, smokers often experience delayed or suboptimal healing due to impaired neutrophil function, altered cytokine production, and reduced vascularization [15]. When assessing periodontal conditions in smokers, clinicians must consider the systemic effects of smoking and its impact on disease progression and treatment outcomes. Advanced imaging techniques and biochemical markers, such as gingival crevicular fluid analysis, can provide additional insights into the extent of periodontal damage in smokers, supplementing traditional clinical assessments [16].

The differences in the microbial composition of the periodontal pockets in smokers and non-smokers further highlight the complexity of assessing periodontal conditions in these groups. Smokers tend to harbor a higher prevalence of pathogenic bacteria, including *Aggregatibacter actinomycetemcomitans* and Treponema denticola, which contribute to the progression of periodontal disease [17]. The smoking-induced alteration of the oral microbiome creates an environment conducive to bacterial colonization and biofilm formation, exacerbating tissue destruction. In non-smokers, the microbial composition is often less pathogenic, with a balance of commensal and opportunistic bacteria [18]. This microbial disparity underscores the importance of detailed microbiological assessments in smokers to identify specific pathogens and tailor treatment strategies accordingly. Combining microbial analysis with clinical indicators can help in early detection and intervention, minimizing the risk of advanced periodontal breakdown in smokers [19].

In addition to microbial differences, systemic factors influenced by smoking play a critical role in periodontal disease assessment. Smokers often exhibit elevated levels of systemic inflammatory markers such as C-reactive protein (CRP), which can contribute to chronic inflammation and hinder periodontal healing [20]. Furthermore, the diminished efficacy of periodontal treatments in smokers necessitates more intensive and frequent monitoring to track disease progression and treatment response. Non-surgical treatments, including scaling and root planning, tend to yield limited improvements in smokers compared to non-smokers, and surgical interventions may also face challenges due to compromised tissue regeneration [21]. To address these disparities, a multidisciplinary approach involving periodontal therapy, smoking cessation programs, and systemic health management is essential. By integrating these strategies, clinicians can enhance the effectiveness of periodontal care and improve the overall oral and systemic health outcomes for smokers [22].

Overall, the assessment of periodontal conditions in smokers requires a nuanced approach that accounts for the unique effects of tobacco use. Smokers are at a higher risk of developing more aggressive forms of periodontal disease, with a distinct microbial profile

dominated by pathogenic species such as *Porphyromonas gingivalis* and *Tannerella forsythia* [23]. Additionally, smoking cessation plays a pivotal role in improving periodontal health, as studies have shown that former smokers exhibit periodontal conditions closer to those of non-smokers [24]. Clinicians should incorporate smoking history into the patient's risk assessment and emphasize smoking cessation as part of comprehensive periodontal therapy. Educating patients on the detrimental effects of smoking on oral health, coupled with regular and thorough periodontal evaluations, is critical for effective disease management and long-term maintenance of periodontal health [25].

The results of this study reinforce the strong association between smoking and poor periodontal health outcomes. Smoking contributes to periodontal disease through several mechanisms:

- **Plaque retention and biofilm formation:** Smoking alters salivary flow and composition, reduces oxygen tension in periodontal pockets, and favors the growth of anaerobic bacteria, which are known to cause periodontal destruction.
- **Impaired host immune response:** Nicotine suppresses the immune response, reducing neutrophil chemo taxis, phagocytosis, and oxidative burst. This compromises the body's ability to combat periodontal pathogens effectively.
- **Vasoconstriction and delayed healing**: Nicotine-induced vasoconstriction reduces blood flow to the gingiva, masking inflammation (lower visible bleeding) while allowing disease progression. This also impairs fibroblast function, collagen production, and tissue repair mechanisms.
- **Increased probing depth and attachment loss:** Smoking weakens the periodontal ligament and connective tissues, leading to increased probing depths and clinical attachment loss. Additionally, smoker's exhibit.

Conclusion

The significantly higher values for plaque index, bleeding on probing, probing depths, CPITN probe depth, and clinical attachment loss in smokers compared to non-smokers underscore the detrimental effects of smoking on periodontal health.

Smokers generally exhibit a higher prevalence and severity of periodontal disease due to the detrimental effects of smoking on oral tissues and immune response. Smoking impairs gingival blood flow, reduces inflammatory signs, and suppresses immune mechanisms, which may mask early symptoms of periodontal disease, leading to delayed diagnosis. Smokers often demonstrate greater loss of attachment, deeper periodontal pockets, and more significant alveolar bone loss compared to non-smokers.

In contrast, non-smokers show a relatively healthier periodontal status, with lower rates of disease progression and better response to periodontal therapy. The absence of smoking-related factors allows for a more robust immune response and quicker tissue healing.

These findings highlight the need for targeted periodontal interventions in smokers, along with smoking cessation programs to mitigate disease progression and improve oral health outcomes.

Future Recommendations

Future research on the assessment of periodontal conditions in smokers versus non-smokers should focus on developing advanced diagnostic tools and exploring biomarkers to detect early signs of periodontal disease, especially in smokers, where inflammation may be masked. Longitudinal studies are needed to understand the progression of periodontal disease in these populations and the effects of smoking cessation on periodontal health. Tailored treatment protocols should be designed to address the specific challenges posed by smoking, such as impaired healing and reduced immune response. Additionally, integrating smoking cessation programs into periodontal care and evaluating their impact on treatment outcomes is crucial. Investigating the effects of emerging tobacco alternatives, such as e-cigarettes and vaping, and understanding their influence on periodontal health is also essential. Finally, interdisciplinary collaboration, public education, and policy advocacy should be prioritized to raise awareness and mitigate the impact of smoking on periodontal health.

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