

## Periodontal Disease in Smokers

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### ABSTRACT

**Background:** A number of published studies demonstrated that smoking represents an increased risk for the development and progression of periodontal disease. Smoking affects the microbiota composition, immune response, and the healing capacity of the periodontium. The aim of this study is to determine the status of periodontal disease in smokers and have an overview about the need for smoking cessation and awareness programs to provide better periodontal health.

**Methods:** A cross-sectional observational study was carried out on 231 patients visiting the dental OPD of Bir Hospital who were smokers using convenience sampling. CPI modified index was carried out, and a comparison and analysis of the impact of different smoking status on periodontitis was done.

**Results:** Out of 231 smokers, 229 individuals (99.13%) had gingival bleeding, 209 individuals (90.47%) had periodontal pockets greater than 3mm and 198 individuals (85.71%) had loss of attachment more than 3mm.

**Conclusions:** Gingival bleeding, periodontal pocket and loss of attachment were significantly more prevalent among smokers, highlighting the importance of smoking cessation and awareness programs.

**Keywords:** Gingival bleeding; loss of attachment; periodontal pocket; periodontitis; smoking.

### INTRODUCTION

Periodontitis is an infection-driven inflammatory disease of the tooth supporting connective tissues and alveolar bone.<sup>1</sup> It is caused by an aberrant host response against oral and dental plaque bacteria. Smoking and other unfavourable lifestyle factors significantly impair the host response.

Smoking represents an increased risk for the development and progression of periodontal disease. Multiple studies have stated that increased pocket depth measurements, attachment loss and alveolar bone loss are more prevalent in smokers.<sup>2-4</sup> Severe rate of periodontal disease might be due to greater amounts of plaque accumulation and higher prevalence of periodontal pathogens in subgingival plaque of smokers than non- smokers.<sup>5</sup>

Smoking is a well-recognized risk factor for periodontal disease and remains a significant public health concern. So, the aim of this study is to determine the status of

periodontal disease in smokers visiting the dental OPD of Bir Hospital and have overview about the need for smoking cessation and awareness programs to provide better periodontal and oral health.

### METHODS

A cross-sectional observational study was conducted at Dental OPD, Department of Dental Surgery, NAMS, Bir Hospital to evaluate the periodontal disease status in smokers. Ethical approval was obtained on January 28, 2020 from Institutional Review Board, Ethical Committee of NAMS, Bir Hospital, Kathmandu, Nepal. (NAMS-IRB 1075/076/077). Sample size was calculated to be 231 by using formula  $n = Z_{\alpha}^2 PQ / d^2$ . Data collection was carried out from January 2020 to May 2020.

Inclusion criteria comprised patients aged 15-59 years visiting the dental OPD who were current or former smokers<sup>6</sup> with more than 10 natural teeth in the mouth. The smokers were further categorized as light, intermediate, and heavy smokers.<sup>7</sup> Exclusion

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criteria were participants who had received periodontal treatment; were on antibiotics; had systemic/ topical steroidal anti-inflammatory drugs or prophylactic antibiotics for the last 4 months. Other systemic conditions that could influence periodontal status (like diabetes mellitus, malignant tumors, rheumatic heart disease, heart valve prolapse, infarction, leukemia, renal diseases, syndromic disorders, pregnant women and lactating mothers, mentally handicapped) were also excluded.

Written informed consent was obtained from all patients after a thorough explanation of the nature, risks, and benefits of the clinical investigation and associated procedures. Smoking status determined what type of smoker he/she was (former, current, light, moderate and heavy) based on number of sticks of cigarette and duration of smoking.<sup>8</sup>

Patients were interviewed with the help of proforma based on “WHO Oral Health Assessment form, 2013”<sup>9</sup> and were clinically assessed for periodontal status. The assessment of the periodontal status was carried out using the Community Periodontal Index (CPI) modified.<sup>9</sup>

The examination was performed with the aid of standardized Community Periodontal Index (CPI) probe (‘TSR 621’ WHO Periodontal probe). CPI probe has a ball tip of 0.5mm diameter, black band between 3.5 mm to 4.5mm from the tip and rings at 8.5mm and 11.5mm from the tip. The scoring criteria for CPI modified are as follows:

Gingival bleeding scores, 0= Absence of condition, 1= Presence of condition, 9= tooth excluded, X= tooth not present.

Pocket scores 0= Absence of condition, 1= Pocket 4-5mm, 2= Pocket 6mm or more, 9= tooth excluded, X= Tooth not present.

Loss of attachment scores, 0= 0-3mm, 1= 4-5mm, (CEJ within black band), 2= 6-8mm (CEJ between upper limit of black band and 8.5mm ring), 3=9-11mm (CEJ between 8.5mm and 11.5mm ring), 4= 12mm or more (CEJ beyond 11.5mm ring)

Loss of attachment was measured by dividing mouth into six sextants, defined by tooth numbers: 17-14, 13-23, 24-27, 38-34, 33-43 and 43-47. It is recorded only for index teeth.

Index teeth for adults aged 20 years or more

17/16	11	26/27
47/46	31	36/37

Index teeth for patients below 19 years:

16	11	26
46	31	36

Two molar teeth in each posterior sextant were paired for recording and, if one was missing, there was no replacement. If no index tooth was present, all the teeth present in the sextant were examined and the highest score was recorded. Bleeding and pocket depths were measured in all teeth.

The data were analysed using SPSS 16 software. Chi Square test was used to assess associations between age group, bleeding score, pocket score, and loss of attachment among smokers. A p value of <0.05 was taken as significant.

## RESULTS

A total of 231 patients were enrolled in this study, with majority of males 200 (86.58%) and 31 (13.41%) females with mean age of 36.11 years. Out of 231 smokers, 63 were former smokers, 168 were current smokers out of which 88 were light smokers, 61 were moderate smokers and 19 were heavy smokers. Out of 231 smokers, 229 individuals (99.13%) had gingival bleeding, 209 individuals (90.47%) had periodontal pockets greater than 3mm and 198 individuals (85.71%) had loss of attachment more than 3mm.

Table 1 shows modified CPI scores among different age groups and gender. Gingival bleeding, periodontal pocket and loss of attachment were seen to be more prevalent among higher age groups individuals. 100% gingival bleeding was seen among smokers of 34-44 years. 99% of males had gingival bleeding, whereas 100% of females had gingival bleeding and periodontal pockets of more than 3mm.

Table 2 shows distribution of modified CPI among individuals of different smoking status allowing for comparison and analysis of impact of smoking on periodontitis. Smoking status demonstrated a statistically significant association with both the prevalence of gingival bleeding and the severity of periodontal pockets (p < 0.05). A positive gradient was observed between smoking intensity and disease severity, with the heavy smokers exhibiting the highest

proportion (73.68%) of periodontal pockets exceeding 6 mm in depth.

All the heavy smokers had loss of attachment of more than 3mm with one individual with loss of attachment of 12mm or more. Loss of attachment was significantly prevalent among smokers with Pearson’s chi square p-value of 0.000 and linear by linear association of 0.023.

**Table 1. Periodontal status of the smokers as per age groups and gender.**

Variable	Sample n (%)	Bleeding (%)		Periodontal pocket (%)			Loss of attachment (%)				
		0 (%)	1 (%)	0 (%)	1 (%)	2 (%)	0(%)	1 (%)	2 (%)	3(%)	4 (%)
Age											
15-29	52 (22.5%)	1 (1.92%)	51 (98.07%)	15 (28.84%)	29 (55.76%)	8 (15.38%)	22 (42.30%)	18 (34.61%)	8 (15.38%)	4 (7.69%)	0 (0%)
30-44	85 (36.79%)	0 (0%)	85 (100%)	5 (5.88%)	36 (42.35%)	44 (51.76%)	10 (11.76%)	29 (34.11%)	22 (25.88%)	20 (23.52%)	4 (4.7%)
45-59	92 (39.82%)	1 (1.08%)	91 (98.91%)	2 (2.17%)	25 (1.08%)	65 (70.65%)	1 (1.08%)	16 (17.39%)	35 (38.04%)	31 (33.69%)	9 (9.78%)
Gender											
Male	200 (86.58%)	2 (1%)	198 (99%)	22 (11%)	82 (41%)	96 (48%)	32 (16%)	55 (27.5%)	52 (26%)	49 (24.5%)	12 (6%)
Female	31 (13.41%)	0 (0%)	31 (100%)	0 (0%)	9 (29.03%)	22 (70.96)	1 (3.22%)	9 (29.03%)	14 (45.16%)	6 (19.35%)	1 (3.22%)

**Table 2. Periodontal status of patients as per smoking status.**

Variable	Former smoker	Current smokers (all)	Light smoker	Moderate smoker	Heavy smoker	p- value
<b>Bleeding</b>						
0	0 (0%)	2 (1.19)	2 (2.27%)	0 (0%)	0 (0%)	0.002*
1	63 (100%)	166 (98.80%)	86 (97.7%)	61 (100%)	19 (100%)	
Total	63	168	88	61	19	
<b>Periodontal pocket</b>						
0	4 (6.34%)	18 (10.71%)	13 (14.77%)	5 (8.19%)	0 (0%)	0.002*
1	22 (34.92%)	69 (41.07%)	46 (52.27%)	18 (29.50%)	5 (26.31%)	
2	37 (58.73%)	81 (48.21%)	29 (32.95%)	38 (62.29%)	14 (73.68%)	
Total	63	168	88	61	19	
<b>Loss of attachment</b>						
0	5 (7.93%)	28 (16.66%)	23 (26.13%)	5 (8.19%)	0 (0%)	0.000*
1	18 (28.57%)	46 (27.38%)	31 (35.22%)	11 (18.03)	4 (21.05%)	
2	20 (31.74%)	46 (27.38%)	23 (26.13%)	18 (29.50%)	5 (26.31%)	
3	15 (23.80%)	40 (23.80%)	9 (10.22%)	22 (36.06%)	9 (47.36%)	
4	5 (7.9%)	8 (4.76%)	2 (2.27%)	5 (8.19%)	1 (5.26%)	
Total	63	168	88	61	19	

\*P <0.05

## DISCUSSION

The findings of the present study demonstrate a significant positive association between smoking and periodontal disease. Our study shows that gingival bleeding, pocket depth and loss of attachment were significantly higher

among smokers. These periodontal parameters were also compared with smoking status, and it was found that higher pocket depth and loss of attachment were seen among heavy smokers. Similarly, heavy smokers showed no site without gingival bleeding.

Some potential mechanisms have been hypothesized by which tobacco smoking affects the incidence and the progression of periodontitis. They include the effect of smoking on the microbiota composition, on the immune response, and the healing capacity of the periodontium.<sup>10</sup> Smoking may lead to a shift in the composition of the subgingival biofilm with an increase in the prevalence of periodontal pathogens.<sup>11</sup> Smoking has been implicated in the delay of neutrophils recruitment and migration into periodontal tissues, thus compromising the acute immune response.<sup>12</sup> The interference with vascular and inflammatory phenomena may also be one potential mechanism for the destructive effects of smoking on periodontal tissues.<sup>13</sup>

Several studies have shown decreased signs of gingival inflammation and bleeding of probing associated with smoking.<sup>14, 15</sup> Several authors have claimed an altered gingival inflammatory response to supragingival plaque in smokers. Smoking considerably attenuates the site-specific association between plaque and BOP with a dose-dependent effect.<sup>16</sup> In our study, smokers showed marked bleeding and heavy smokers showed no site without gingival bleeding. Baab and Öberg observed an acute increase in blood flow to the gingiva during smoking, which might lead to an increased bleeding tendency after probing.<sup>17</sup> In contrast to our study, bleeding tendency was lower in smokers for plaque-free sites, as compared to non-smokers. Although Albeit bleeding tendency was slightly increased in plaque-covered sites in smokers, it was considerably lower as compared to plaque-covered sites in non-smokers.<sup>18</sup>

Gingival bleeding, periodontal pocket and loss of attachment were seen to be more prevalent among 30-44 and 45-59 year groups compared to the 15-29 year group individual. In our study, CPI scores were also more prevalent among higher age group individuals which was similar to the findings by Gupta et. Al,<sup>19</sup> which showed higher age predicted greater incidence of periodontal disease. Age has been described as a non-modifiable predisposing factor<sup>20</sup> which strengthens the evidence even more that the periodontitis tends to cumulate for life. Individuals that had smoked the most during the life course had worse periodontal status (greater incidence and faster progression of periodontitis) than their non-smoking counterparts.<sup>21</sup>

The majority of the smokers who participated in our study were males (86.58%). However, this may not be the true picture of smoking status as many female smokers might not have accurately responded due to social and cultural aspects. Smoking was found to be significantly related to male gender<sup>22</sup> while study by Gupta et al,<sup>19</sup> showed no difference in the proportion of periodontal disease in male and female.

Smokers have a significantly higher risk for periodontal disease compared to non-smokers and the risk is increased with duration and rate of smoking.<sup>23</sup> Smokers exhibit a clinically distinct predisposition to periodontal disease, with deeper pockets, more extensive and severe attachment loss, greater levels of bone destruction, and a higher rate of tooth loss.<sup>24</sup> Khan et al,<sup>22</sup> depicted 3.5 times higher risk of chronic periodontitis among smokers. In our study, periodontal parameters were also compared with smoking status and it showed similar findings that higher pocket depth and loss of attachment were seen among heavy smokers.

In contrast to earlier research findings, which indicated a higher incidence of periodontal disease among current smokers compared to former smokers, our study revealed a different trend, with a somewhat higher prevalence observed among former smokers. Years of tobacco exposure, pack years, time since quitting would also affect these findings.

The common description used for periodontal diseases is the CPI modified, which was used in the present study and was the method recommended by WHO 2013<sup>9</sup> for population screening purposes. Due to several advantages, such as standardisation, validation and ease of measurement by clinician and universal use, which allows comparison among studies, these measures have been used in our study. Another strength of the study is the classification of individuals into current, former and non-smokers which minimised the bias and contributed to a better analysis of the evidence.

The weaknesses of the present study include the outcome assessment. Although probing depth and loss of clinical attachment are a common measure of periodontal disease assessment in the majority of studies,<sup>25, 26</sup> it is not a true outcome. The best measure for diagnosis of periodontitis would be tooth loss, a true outcome that represents unambiguous evidence of concrete consequences perceived by the patient, instead of probing depth or attachment level, which are physical signs that are used as an alternative to a clinically significant end point.

Another limitation of the study is enrolling only those patients coming to dental department, Bir Hospital in the study. A longer study period including larger population would have given better picture.

There could also be potential sample bias in the study. The study may have inadvertently included a disproportionate number of smokers with pre-existing periodontal issues, leading to an overestimation of the association between smoking and periodontal disease severity. A more representative sample of smokers and non-smokers could yield different results.

Although an association between smoking and periodontal pocket formation and attachment loss was observed, this relationship may be influenced by alternative explanations. Heavy smokers may have coexisting risk factors such as poor oral hygiene, genetic predisposition, systemic conditions, altered plaque characteristics, variations in oral microbiota or host response, as well as behavioural factors including infrequent dental visits, poor nutrition, high stress levels, and lower socioeconomic status with limited access to dental care. Despite statistical adjustments, unmeasured confounders such as alcohol consumption, medication use, or occupational exposures may also influence both smoking behaviour and periodontal health, potentially leading to spurious associations and highlighting the multifactorial nature of periodontal disease.

Future prospective longitudinal studies are needed that concentrate on quantifying the possible dose-dependent effect of tobacco smoking on periodontitis. Population based smoking cessation programs at primary health care should be implemented which help in preventing, initiating and progression of periodontal diseases and other systemic diseases. Primary prevention of periodontal diseases may lead to benefits beyond dental health and may have greater implications for general health.

## CONCLUSIONS

In this cross-sectional study, assessment of periodontal status among smokers using the CPI modified index revealed a substantial prevalence of gingival bleeding, periodontal pockets, and loss of attachment across different smoking categories. These findings provide a comprehensive overview of periodontal health in smokers and emphasize the importance of integrating periodontal health assessments with awareness and counseling efforts as part of preventive oral health

strategies.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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