Effect Of Cigarette Smoking On Periodontal Health Status In Nepalese Population: A Cross-Sectional Study
Dhami B¹, Shrestha P², Humagain M³

¹ Lecturer, Dept of Periodontics Kantipur Dental College, Kathmandu, Nepal  ² Lecturer, Dept of Periodontics KIST Medical College, Lalitpur, Nepal  ³ Assistant professor, Dept of Periodontics Kathmandu University School of Medical Sciences, Dhulikhel, Nepal

Abstract

Background:
Smoking is the major risk factor in the prevalence, extent and severity of periodontal diseases. Tobacco smoking, mostly in the form of cigarette smoking, is recognized as the most important environmental risk factor in periodontitis.

Aim and objectives:
The aims of the study were to evaluate the periodontal health status among cigarette smokers and non-cigarette smokers and to assess effect of smoking on periodontal health with the duration of smoking.

Materials and Methods:
The study included 300 subjects (101 cigarette smokers and 199 non-smokers) aged 15-74 years. The subjects were randomly selected. Community Periodontal Index (CPI) scores was recorded for each patient. Chi Square test was applied for comparing the associations. A p value of less than 0.05 was considered as significant.

Results:
Periodontal condition as assessed by CPI score showed that there was statistically significant difference in the findings between cigarette smokers and non-smokers. The results also showed that those who smoked for lesser duration of time had less periodontal destruction than the subjects who smoked for longer duration

Conclusions:
The current study shows that smoking is one of the major environmental factors associated with accelerated periodontal destruction. It was found that cigarette smoking was associated with lesser gingival bleeding and deeper pockets as compared to non-smokers.

Keywords:
Community periodontal index, periodontal disease, smoking

Correspondence:  Dr. Bhageshwar Dhami, Lecturer and In charge, Department of Periodontics, Kantipur Dental College, Kathmandu, Nepal, E-mail:dhamibhagesh@hotmail.com
INTRODUCTION

Periodontal disease is one of the most common chronic diseases in adults. Smoking is the major risk factor in the prevalence, extent and severity of periodontal diseases. Cross-sectional studies have shown that smokers are two to seven times more likely to present periodontitis, compared to nonsmokers, and smoking has been associated with tooth loss during periodontal maintenance as well. With respect to surgical or non-surgical periodontal therapy, several studies have shown that smokers have a worse response to treatment when compared to nonsmokers.

Tobacco smoking, mostly in the form of cigarette smoking, is recognized as the most important environmental risk factor in periodontitis. Smoking is thought to impair the immune response and compromises the periodontal tissues ability to heal following a period of disease activity. Gingival bleeding has been consistently reported to occur less in smokers due to nicotine induced vasoconstriction in smoker's gingiva as well as heavy gingival keratinization. Pocket depth measurements are found to be greater in smokers due to increased alveolar bone loss. Periodontal breakdown has been shown to be more severe among current smokers compared to former smokers. Those who have never smoked have been observed to have the lowest risk. Smoking has a strong negative impact on regenerative therapy, including osseous grafting, guided tissue regeneration, or a combination of this treatment.

The aims of this study were to evaluate the periodontal health status among cigarette smokers and non cigarette smokers and to assess effect of smoking on periodontal health with the duration of smoking.

MATERIALS AND METHODS

A cross sectional study design was used for the study. A total number of 300 subjects of 15-74 years were chosen by random sampling technique. They were examined in the Department of Periodontics, Kantipur Dental College, Kathmandu. A prior consent of all patients was obtained for the screening. A detailed questionnaire which included personal data regarding each subject was recorded on the proforma. The recording of data was based on the World Health Organization (WHO) Oral Health Assessment Survey Form [proforma]. Community Periodontal Index (CPI) was used depending on following criteria.

Exclusion Criteria:
1. For 15-19 years, only six index teeth are examined. Second molars are excluded.
2. Third molars are not included, except where they are functioning in place of second molars.
3. Systemic conditions that might affect periodontal disease activity or requiring premedication with antibiotics for periodontal probing; medications such as antibiotics, steroids, or non-steroidal, anti-inflammatory drugs within the past 6 months.

Inclusion Criteria:
1. Male and female subjects from 15 to 74 years.
2. Two or more teeth present in a sextant that are not indicated for extraction.

Subjects were divided into two groups:
- Cigarette smokers
- Non-smokers

The clinical examination included an assessment of periodontal condition using the CPI on the index teeth. Each subject was examined on dental chair using a mouth mirror and CPI Probe. The examination was performed in a systemic manner beginning from maxillary right sextant.

The following CPI codes and criteria were used to record periodontal status: Code 0: healthy periodontium, Code 1: bleeding observed, directly or by using a mouth mirror, after probing, Code 2: calculus detected during probing, but the entire black band on the probe visible, Code 3: Pocket 4-5mm (gingival margin within the black band on the probe), Code 4: Pocket 6mm or more (black band on the probe not visible), Code X: Excluded Segment(Less than two teeth present) and Code 9: Not recorded.

Chi Square test was applied for comparing the associations. A p value of less than 0.05 was considered as significant.
RESULTS

In the present study, a total number of 300 subjects were examined for assessing their periodontal status. The subjects were 15 to 74 years of age. Of the 300 subject, 151(50.3%) were males and 149(49.7%) were females (Figure 1) and 199(66.3%) were non smokers and 101(33.7%) were smokers (Figure 2). In this study, 39.6% smoked less than 10 years, 40.6% smoked for more than 10 years but less than 20 years and 19.8% smoked for more than 20 years (Figure 3).

Among non smokers, 1% had healthy periodontium, 44.2% had bleeding on probing, 49.2% had calculus, 5% had shallow pockets and 0.5% had deep pockets. Among smokers, 11.9% had bleeding on probing, 32.6% had calculus, 30.7% had shallow pockets and 24.8% had deep pockets (Table 1). It can thus be concluded that the non smokers had better periodontal status than smokers. This association was highly significant. (p<0.001)

Among the subjects who smoked for less than 10 years, 12.5% had bleeding on probing, 42.5% had calculus, 15% had shallow pockets and 30% had deep pockets, who smoked for 10-20 years, 12.2% had bleeding on probing, 31.7% had calculus, 41.5% had shallow pockets and 14.6% had deep pockets and who smoked more than 20 years, 10% had bleeding on probing, 15% had calculus, 40% had shallow pockets and 35% had deep pockets (Table 2). The results thus suggest that those who smoked for lesser duration of time had less periodontal destruction than the subjects who smoked for longer duration. However this association was not significant.

Figure 1: Gender Distribution: Male to Female Patient Ratio

Figure 2: Distribution of the study population according to smoking habit

Figure 3: Distribution of the subjects according to duration of smoking

Table 1: Comparison of CPITN Score between smokers and non smokers

<table>
<thead>
<tr>
<th>Highest CPI score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Total</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non smoker</td>
<td>2</td>
<td>1%</td>
<td>88</td>
<td>44.2%</td>
<td>98</td>
<td>10%</td>
<td>1%</td>
</tr>
<tr>
<td>Smokers</td>
<td>0</td>
<td>0%</td>
<td>12</td>
<td>11.9%</td>
<td>33</td>
<td>31%</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>0.7%</td>
<td>100</td>
<td>33.3%</td>
<td>131</td>
<td>41</td>
<td>26</td>
</tr>
</tbody>
</table>

*highly significant
DISCUSSION

The detrimental effect of cigarette smoking on the periodontal health was demonstrated in this study. The estimated risk of periodontal destruction for smokers increased compared to the non-smokers. The result of the present study is similar to the study conducted by Goultschin. Various factors such as altered host response and changes in oral microflora may probably contribute to more severe forms of periodontal disease in smokers.

The findings in the present study are consistent with the study of Feldman et al. which showed that smokers with periodontal disease had less clinical inflammation and gingival bleeding when compared with non-smokers. This may be explained by the fact that one of numerous tobacco smoke by-products, nicotine, exerts local vasoconstriction thus reducing blood flow and edema. The results of the current study are similar to those reported by Linden and Mullally, Harber et al., Schenkein et al., and Haffajee. All of these studies have shown that young adult smokers have a higher prevalence and severity of periodontitis compared to non-smokers.

There was a direct correlation observed in this study between duration of smoking and periodontal destruction. This finding is in accordance with other studies. Tobacco smoke contains many cytotoxic substances such as nicotine, which can penetrate the soft tissue of oral cavity, adhere to the tooth surface or enter to the blood stream. Potential molecular and cellular mechanisms in the pathogenesis of smoking associated periodontal diseases has been reported and these include, immuno-suppression, exaggerated inflammatory cell responses, and impaired stromal cell functions of oral tissues. The association between cigarette smoking and periodontal diseases represent a significant oral health problem.

Some in vitro studies provided other possible intimate mechanisms by which smoking may affect bone metabolism. Rosa et al. reported that nicotine increased the secretion of interlukin-6 and tumor necrosis factor alpha in osteoblasts and production of tissue-type plasminogen activator, prostaglandin E2, and matrix metalloproteinases, thereby tipping the balance between bone matrix formation and resorption toward the latter process, as reported by Katano et al. Although bacteria are the primary etiologic factors in periodontal disease, the patient's host response is a determinant of disease susceptibility. Smokers appear to have depressed numbers of helper lymphocytes, which are important to B-cell function and antibody production. The combined effect of bacterial colonization and the local and systemic effect of smoking are responsible for the greater severity of periodontal destruction in smokers of the current study.

Table 2: Comparison of CPI TN Score with duration of smoking

<table>
<thead>
<tr>
<th>Highest CPI score</th>
<th>1%</th>
<th>2%</th>
<th>3%</th>
<th>4%</th>
<th>Total %</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤10 years</td>
<td>5</td>
<td>17</td>
<td>6</td>
<td>12</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>≤10 years &amp; ≤20 years</td>
<td>5</td>
<td>13</td>
<td>17</td>
<td>6</td>
<td>41</td>
<td>100%</td>
</tr>
<tr>
<td>≥20 years</td>
<td>2</td>
<td>3</td>
<td>8</td>
<td>7</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>33</td>
<td>31</td>
<td>25</td>
<td>101</td>
<td>100%</td>
</tr>
</tbody>
</table>

*not significant
CPI as recommended by the World Health Organization has been used in the present study. It was originally proposed as an appropriate estimation of disease in large epidemiological surveys and has contributed to an understanding of the epidemiology of periodontal disease on a global level. However, CPI measurements are done only in the index teeth so it can overestimate or underestimate the prevalence of periodontal disease. Exact age and gender matching of smokers and non-smokers was not possible. Finally we could not obtain clinical attachment level data, mainly because of the limited time allowed for the single-appointment clinical examination. However our results may offer an estimation of the prevalence of moderate to deep periodontal pockets and thus confirms a consistent association between smoking and periodontal status in Nepal. Further studies utilizing more comprehensive clinical assessment and quantitative microbiological method such as real-time PCR are necessary.

CONCLUSION

The current study shows that smoking is one of the major environmental factors associated with increased periodontal destruction. Within the limit of this study, positive association was observed between periodontal disease and cigarette smoking. It was found that cigarette smoking was associated with lesser gingival bleeding and deeper pockets as compared to non-smokers. Smoking cessation should be considered in the treatment of periodontitis and be a part of health prevention in dentistry. In addition, smoking cessation is the main option to revert the harmful effects of smoking on periodontal risk and therapy.

REFERENCES